REVIEW ARTICLE
Mycoplasma bovis: Taxonomy, Characteristics, Pathogenesis and Antimicrobial Resistance

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Abstract
Mycoplasma bovis (M. bovis) is one of the most significant bacteria, which leads to multiple bovine diseases such as keratoconjunctivitis, otitis media, arthritis, genital disorders, mastitis, and pneumonia in cattle. M. bovis is considered the second most pathogenic mycoplasmas after Mycoplasma mycoides sub sp. mycoides (Mmm) that causes contagious bovine pleuropneumonia. M. bovis is a wall-less host-specific bacterium. Currently, it is responsible for important economic problems worldwide such as reducing production, premature culling, and increasing the mortality of the affected animals. The infection caused by M. bovis is hampered due to the lack of effective vaccines and treatment. Besides, antibiotic resistance to macrolides and fluoroquinolones, the drug of choice for M. bovis treatment, has formidable economic losses due to treatment limitations by these antimicrobials. Our review highlights and discusses the taxonomy, general characteristics, economic importance, isolation, identification, and pathogenesis of M. bovis. Finally, it focused on the antimicrobial resistance of this particular bacterium.

Keywords: Mycoplasma bovis, mycoplasmosis, pathogenesis, antimicrobial resistance, cattle.

Introduction
Mycoplasma bovis (M. bovis) is a significant microorganism for the cattle industry, which leads to economic important losses worldwide [1]. The genus Mycoplasma is characterized by a small genome size, the lack of its cell wall and low G+C content (23–40 %) [2]. Currently, genus Mycoplasma contains at least 130 species (spp.) and M. bovis is considered one of the important causes of bovine mycoplasmosis [3]. Additionally, M. bovis is considered the second most pathogenic mycoplasmas after Mmm that causes contagious bovine pleuropneumonia [4]. In 1961, M. bovis is observed in a case of mastitis in the USA [5] and since then it has been linked with a wide range of clinical infections such as genital disorders, arthritis, bovine respiratory diseases, and otitis media [6]. M. bovis can persist for very long periods in a herd with the possibility of shedding the microorganism for a few weeks to several months by the infected animals [7, 8].

M. bovis has significant virulence properties, which help in evading the host immune system such as adhesion, host cells invasion, host immune system modulation, production of secondary metabolites, biofilm formation, and synergistic infections with other viral, and/or bacterial microorganisms [9]. Additionally, M. bovis became urgently resistant to several antimicrobial classes such as fluoroquinolones, macrolides, tetracyclines, and β-lactams because of the uncontrolled usage of antimicrobial agents in the animal industry. Hug economic losses have been occurring due to treatment...
limitations by these antimicrobials [10]. Therefore, this review spot the light on all fundamental issues related to *M. bovis* including (i) the history, taxonomy and, general characteristics, (ii) clinical and economic importance, (iii) isolation and identification, and (iv) pathogenesis and antimicrobial resistance.

**The historical standpoint of Mycoplasma species**

Mycoplasma name is derived from the Greek words mykes (fungus) and plasma (formed). Before the 1930's, *Mycoplasma* spp. They were thought to be viruses due to their small size genome; they could pass through filter paper, which prevents the passage of ordinary bacteria and its ability to make a cytopathic effect when it cultured on the embryonated chicken egg. Latterly, they were considered commensal growing bacteria with *Streptobacillus* spp. Thereafter, they were considered to be bacteria, which had lost their cell wall (L-form bacteria) and characterized by unusual colony shape "fried-egg" [11]. When DNA hybridization gave the first genomic data analysis, it excluded any association between *Mycoplasma* spp. and stable L-forms of bacteria [12]. In 1950, *Mycoplasma* name is used as an alternative to the term pleuropneumonia-like organisms (PPLO) [13]. In 1961, *M. bovis* was firstly observed in the USA in a case of mastitis [5]. Subsequently, in 1976, it was described as the cause of pneumonia and arthritis in calves [14] and since then *M. bovis* has been detected in most countries worldwide. Initially, *M. bovis* was named *M. agalactiae subspecies bovis* due to both biochemical and clinical similarities in many aspects with the small ruminant pathogen "*M. agalactiae*" [14-16]. In 1985 [17] and 1986 [18], *M. bovis* was firstly detected from bovine mastitis outbreaks in Egypt and then has persisted in Egyptian cattle herds [19]. In 1990, *M. bovis* was isolated from cases of arthritis and pneumonia in calves in Europe due to increased calf importation. In 1994, *M. bovis* was firstly reported in the Republic of Ireland, a group of cattle showing severe acute respiratory infections was imported from France [20].

**Taxonomy and classification of Mycoplasma species**

*Mycoplasma* belongs to the class *Mollicutes*, which means in Latin soft skin, and family *Mycoplasmataceae*, which consists of eight genera: *Mycoplasma, Anaeroplasma, Spiroplasma, Ureaplasma, Acheloplasma, Asteroplasma, Entomoplasma,* and *Mesoplasma* [19, 21, 22]. Currently, the genus *Mycoplasma* contains at least 130 spp., which contains clusters of subspecies [3]. Several *Mycoplasma* spp. (bovine mycoplasmas) are important in cattle with variable degrees of clinical importance; these spp. include *M. bovis, M. californicum, M. bovigenitalium, M. bovirhinis, M. bovoculi, M. leachii* (previously known as *Mycoplasma* spp. bovine group 7), *M. mycoides sub sp. mycoides* [23], *M. wenyonii, M. arginini, M. alkalescens, M. canadense, M. canis,* and *M. dispar* [2, 24].

The classification scheme for the current taxonomic standards of bovine mycoplasmas was based upon 16S rRNA gene sequencing for differentiation between closely related spp. (Figure 1) [25].
Figure (1): Phylogenetic tree of the family Mycoplasmataceae based upon 16S rRNA gene sequence similarity [25].
General characteristics and habitat of *Mycoplasma* species

*Mycoplasmas* are one of the simplest and smallest prokaryotes, which have only the minimal cellular machinery needed for survival and replication. They evolved from Gram-positive bacteria by degenerative evolution through the reduction of genome and cell wall loss [25, 26]. They are characterized by their small genome size, low G+C content (23–40 %), ß-lactam resistance, and lack of cell wall connected with a highly limited metabolic capacity [2, 27]. The lack of a cell wall explains several unique characters of *Mycoplasma* spp. such as formation of the characteristic colonies of peculiar fried egg-shaped, penicillin resistance, and sensitivity to detergents and osmotic shock. Thin sections of *Mycoplasma* spp. showed that the cells are built mainly of three organelles including circular densely packed and double-stranded DNA molecules, ribosomes, and the cell membrane [12, 28]. As *Mycoplasma* spp. are the smallest known free-living pathogens, they can adapt to a special lifestyle as opportunistic organisms or commensal pathogens. They have an inadequate biosynthetic capacity that means they don't have several biochemical pathways present in the Eubacteria. They are greatly adapted to the hosts that provide them with most of their growth nutritional requirements [29].

*M. bovis* can be transmitted directly through nose-to-nose contact or aerosols or indirectly through contaminated utensils and feed by respiratory secretions of the infected animals, [30]. Mycoplasmas are susceptible to sunlight and dehydration, but *M. bovis* can survive for long periods in protected environments with the greatest survival in humid and cool conditions. *M. bovis* has been observed to persist in recycled sand bedding for months [31]. In addition, it can survive in milk and sponges for nearly two months and in water for over two weeks at 4 °C, but survival rates drop considerably at higher temperatures [32].

The incubation period for *M. bovis* infection is difficult to be defined because it depends on many factors including the stress state of the animals, especially after translocation, herd management, the presence of co-infections, the infectious dose, the pathological and clinical effects of the infection, the age of the infected animal, and the virulence of field isolates. In experimental infections, the incubation period for mastitis is shorter than pneumonia, which reaches seven days. *M. bovis* shedding is intermittent; thus, diagnosis by detection of the organism in individual animals may be variable. Therefore, herd diagnosis especially by cheaper serological techniques may be more reliable for persistent infections. The ability to detect *M. bovis* is also affected by collection techniques, transportation, and storage the specimens after reaching the laboratory and how they are stored. The infected animals can shed *M. bovis* for months and may be years, and thus act as reinfection source and future outbreaks of clinical infection [3].

Clinical importance of *Mycoplasma bovis* in the veterinary field

*M. bovis* is an opportunistic bacterium, which normally colonizes the bovine upper respiratory tract; but under stressful conditions, *M. bovis* becomes pathogenic and starts replication and distribution to other sites such as the lower respiratory tract, middle ear, joints and mammary gland [33, 34].

*M. bovis* may act synergistically with other pathogens including *Actinomyces pyogenes*, *Haemophilus somnus*, and *Pasteurella* spp. causing bovine pneumatic pasteurellosis, that also known as respiratory disease complex of cattle, bovine enzootic bronchopneumonia, or bovine respiratory disease (BRD) [2]. Clinical signs of pneumonia caused by *M. bovis* infection are
not characteristic; they are in the form of mild to continuous cough, hyperpnoea, dyspnea, nasal discharge, loss of appetite, runny eyes, mild depression, and low-grade fever [35]. Pneumonia can occur as a single manifestation of *M. bovis* infection or in combination with other clinical manifestations such as otitis media in young calves, polyarthritis in adult animals, and mastitis in dairy cows [36, 37].

Additionally, *M. bovis* induced arthritis can occur in cattle at any age, but it usually occurs in pre-weaned calves and is associated with respiratory infection. Moreover, it is characterized by fever, lameness, joint swelling, and pain in the acute phase. Big rotator joints (carpal, elbow, shoulder, hock, stifle, and hip) are frequently affected. Poor response to antibiotic therapy is also a common characteristic of the disease. Lesions in the joints are characterized by necrotizing fibrinosuppurative arthritis and tenosynovitis. In chronic cases, the affected joints contain yellowish-white fibrinous or caseous material in the thickened joint capsule. Involvement of the adjacent ligaments and tendons is common [35, 38]. Furthermore, *M. bovis* infection results in decreasing milk production in dairy cows, mortality, and weight loss in surviving calves [39].

*M. bovis* either alone or in association with other pathogens is believed to be one of the main causative agents of otitis media interna in calves with various degree from pyrexia to neurologic manifestations. These symptoms are secondary to dysfunctions of the facial (cranial nerve VII) and vestibuleocochlear (cranial nerve VIII) nerves. As a consequence of otitis media, poor appetite, obtundation, pain and conjunctival discharge, and meningitis can be identified [40, 41]. In addition, *M. bovis* was isolated in case of cattle with corneal opacity, corneal ulceration, and marked swelling of eyelids during the outbreak of severe conjunctivitis [42, 43].

**Economic importance of *Mycoplasma bovis***

Globally, *M. bovis* has a fundamental economic effect on the cattle industry in the form of reduced production, increased mortality rates, premature culling of infected animals, treatment, labor expenses, besides, the application of diagnostic methods and different control and preventive measures [1, 35]. The economic effect of *M. bovis* infections is difficult to be measured because the clinical and pathological signs are unspecific, further, the diseases attributed to this agent commonly include interactions of more than one pathogen [44]. *M. bovis* is responsible for one third of the economic losses in the cattle industry in Europe [15]. The prolonged antibiotic treatment of chronic *M. bovis* associated diseases contributes to the development and spread of antimicrobial resistance [44].

Several authors reported high prevalence rates of *M. bovis* from different sources worldwide such as lung tissues in; Canada (98%) [45], the United Kingdom (86.4%) [46], Argentina (70%) [47], nasal swabs, lung tissues, and milk in France (55%) [48], nasal swabs in Poland (47.8%) [49], lung tissues in the United States of America (USA) (41.1%) [50], nasal swabs in the United Kingdom (40.2%) [51], milk in; the United Kingdom (38%) [46] and Poland (29.6%) [49]. On the other hand, *M. bovis* was isolated with low prevalence rates from milk in; the Czech Republic (8%) [52], Northern Greek (8.2%) [53] and Spain (16.36%) [1].

In Egypt, high prevalence rates of *M. bovis* were recorded from clinical mastitic milk in Alexandria (70.83%) [54], nasal swabs in Cairo (61.5%) [55], lung tissues and joints in Kafr El-Sheikh (40.9%) [1], nasal swabs in El-Menofia (40%) [56], lung tissues in Cairo (36.8%) [55], milk in; El-Fayoum (31.6%) [57], Ismailia (28.7%) [19], oral swabs in El-Menofia (25%) [56], milk in El-Dakhlia (22%) [57] and conjunctival swabs in El-Menofia (20%) [56].
Isolation and identification of *Mycoplasma bovis*

Traditionally, the detection and identification of bovine *Mycoplasma* spp. have been done through microbial culture. *M. bovis* is less fastidious to culture than other pathogenic mycoplasmas; therefore, the isolation techniques of *M. bovis* need complex media, specialized equipment types and technical skills [13, 58]. In pneumonic cattle, bronchoalveolar lavage fluid or the affected lung tissues are the most suitable samples used for the isolation of *M. bovis* compared with nasal swabs [59]. *M. bovis* can affect wide several organs and tissues and can also be recovered from apparently healthy cattle [9]. It can grow in various media, but all of them should contain yeast extract, tryptone (amino acid source), serum (sterol source), glucose, and/or pyruvate (energy source), in addition to, penicillin or other β-lactam antibiotics (selective agent), phenol red or other pH indicators (for detection of the bacterial growth) [13, 44].

Various kinds of media are widely utilized in confirmation of *M. bovis* infection such as Eaton’s, modified PPLO, and Hayflick’s media [46]. Broth cultures are incubated at 37 °C under aerobic conditions and growth of the bacteria is frequently apparent after 48 h, but incubation up to 10 days is recommended before the sample is considered negative. Agar plates are incubated under 5 - 10 % CO₂ at 37 °C atmospheric condition until visible colonies appear (2-4 days). Colonies of *M. bovis* are examined by stereomicroscope, which shows 0.1 to 0.5 mm diameter with a typical fried egg appearance [35]. Recently, a selective diagnostic medium; modified PPLO is available for the detection of *M. bovis* with red colonies appearing in several days using stereomicroscope [60].

Additionally, the digitonin test is important in the identification of the class *Mollicutes* depending on the sterol requirement. This test can also differentiate the sterol-requiring *Mollicutes* (Genus *Spiroplasma, Entomoplasmata, Ureaplasma, Mycoplasma*, and *Anaeroplasma*) from the non-sterol requiring *Mollicutes* (Genus *Acholeplasma, Asteroplasma*, and *Mesoplasma*) [61]. Usually, the diagnosis and identification of *M. bovis* have been done through microbial culture, biochemical characteristics, and serological tests. There are only few biochemical properties that can be helpful in its diagnosis (Table 1). Therefore, identification of *Mycoplasma* spp. is greatly dependent on serological tests, which are based on the detection of structural membrane proteins using specific antisera such as indirect ELISA for the recognition of anti-mycoplasma antibodies in milk and sera [2, 15, 29].

| Table (1): Comparative biochemical characteristics of *Mycoplasma* species affecting cattle [15] |
Recently, molecular methods such as polymerase chain reaction (PCR) are widely used to identify *Mycoplasma* spp. from various bovine samples. This technique is based on the amplification of conserved bacterial 16S rRNA gene [62]. Interestingly, only eight nucleotides differ between the sequences of the 16S rRNA of *M. agalactiae* and *M. bovis* [63]. Therefore, PCR techniques targeting the 16S rRNA gene do not reliably distinguish between them unless additional methods are used such as melting temperature analysis of the PCR products, denaturing gradient gel electrophoresis (DGGE) of the amplicons [64], the use of different spp. primers [65], DNA sequencing [25]. Recently, DNA sequencing based on 16S rRNA sequence analysis can distinguish between 130 different *Mycoplasma* spp. such as *M. agalactiae* and *M. bovis* [13, 25].

**Pathogenesis of Mycoplasma bovis**

*M. bovis* is known to be responsible for serious health complications and economic losses in cattle due to the possession of several virulence properties such as variable surface lipoproteins (Vsps), adhesion, host cell invasion, the host immune system modulation, the production of secondary metabolites, and biofilm formation [9]. One of the first steps during *M. bovis* infection is the adherence to bovine tracheobronchial epithelial cells, which facilitates its colonization in the lung. It is mediated by membrane proteins including Vsps, a family of immunodominant lipoproteins on the bacterial surface and unrelated proteins such as P26 and pMB67 [66, 67]. Cytoadherence is differing among the strains and correlates with the pathogenicity and the number of in vitro passages [67]. After colonization, *M. bovis* can invade the immune cells contributing to its distribution to different infection sites of the host such as the respiratory tract, middle ear, joints, lymph nodes, and mammary glands [68]. Variable surface lipoproteins are responsible for the highly variable antigenic profiles of *M. bovis* [9, 69]. Moreover, *M. bovis* field isolates have modified versions of the *vsp* gene complex, where extensive variations in the gene sequence primarily occur in the repeated coding sequences of the *vsp* structural genes. These results demonstrated that there are antigenic variations within *M. bovis* populations [70]. Additionally, the ability to
undergo antigenic variations by phenotypic modulations of immunodominant surface lipoproteins and the resulting alteration of the host immune response can lead to *M. bovis* persistence of and the development of chronic infection [45].

*M. bovis* also generates secondary metabolites such as hydrogen peroxide to damage the host cells. The amount of peroxide produced varies among different isolates and *in vitro* passages of strains led to a reduction of hydrogen peroxide levels [71]. Finally, biofilm formation of *M. bovis* may contribute to the persistence of this pathogen as well as increase its resistance to desiccation and heat stress [72].

**Resistance of Mycoplasma bovis to antimicrobial agents**

Antimicrobials are one of the most fundamental therapeutic methods in human and veterinary medicine. Additionally, they are used as growth promoters in the animal industry, but their use is limited due to the emergence of antimicrobial resistance among pathogenic bacterial strains. The main factors that affect bacterial resistance are the random utilization of antimicrobials to treat human infections besides their excessive use in veterinary medicine [73-75].

Interestingly, antimicrobial resistance is fast developing in *M. bovis* strains worldwide due to the uncontrolled extensive usage of antimicrobials in treatment of bovine pneumonia [76]. *In vivo* potential effectiveness of antimicrobials can be assessed by *in vitro* susceptibility testing using broth microdilution, agar dilution, and the E-tests to determine the minimum inhibition concentration (MIC). It refers to the lowest concentration of antimicrobials that inhibit the growth of microorganisms, which can provide the basis for the best choice of antimicrobials in the treatment [10, 35]. *M. bovis* become resistant to several antimicrobial classes including fluoroquinolones, macrolides, and tetracyclines. *M. bovis* resistant isolates have been increasingly observed around the world as a result of the overutilization of antimicrobials to treat bovine pneumonia. The frequency of fluoroquinolones resistant isolates differs considerably from one country to another [77].

**Fluoroquinolones**

Fluoroquinolones are the most effective antimicrobials in inhibiting *M. bovis* infections [78]. They act by inhibiting the DNA gyrase and topoisomerases II and IV that are important for DNA replication [79]. Resistance to fluoroquinolones in many *M. bovis* isolates is because mutations in the quinolone resistance-determining regions (QRDRs) of the *gyrA* and *gyrB* genes encoding DNA-gyrase as well as, the *parC* and *parE* genes encoding topoisomerase IV [80]. All *M. bovis* strains with high fluoroquinolone resistance (MICs ≥10 μg/mL) have at least one substitution in both *gyrA* and *parC* genes. Earlier studies reported mutation hotspots in *gyrA* and *parC* genes of naturally and artificially selected resistant *M. bovis* strains have been observed among amino acid positions 81 to 87 and positions 78 to 84, respectively [77].

**Tetracycline**

Tetracyclines are broad spectrum antimicrobials, which have been greatly utilized in veterinary and human medicine. They prevent protein synthesis by binding to the 30S ribosomal subunit and preventing the attachment of aminoacyl-tRNA to the A-site [81, 82]. Decreased susceptibility to tetracycline in *M. bovis* strains (MICs ≥ 2 μg/mL) was linked with mutations at two or three positions of the 16S rRNA-encoding genes, which affect the tetracyclines binding sites. Cross-resistance between spectinomycin and tetracycline was observed in tetracycline-resistant *M. bovis* mutants induced *in vitro* [82, 83].

**Macrolides and lincosamide**

Macrolide and lincosamide antimicrobials are chemically different, but they have a similar mode of action. *M. bovis* become resistant to macrolide and lincosamide antimicrobials by drug inactivation and target-site modifications through mutation or methylation, which prevent the binding of the antibiotics to their
ribosomal targets. Modifications of the ribosomal target lead to broad-spectrum resistances to lincosamides and macrolides, while drug inactivation affects only some of these molecules. Resistance to macrolides can also occur due to methylation of key nucleotides in domains II (G748 nucleotide of 23S rRNA gene) and/or in domain V (A2058 nucleotide of 23S rRNA gene) in *M. bovis* [84, 85].

**Aminoglycosides**

The aminoglycosides are amino sugars that are present in two wide groups due to their chemical structures, streptomycin and its derivatives and deoxystreptamines. The ribosome is the primary target of their action, but other actions on membranes and RNA synthesis modifications have been also reported. These antibiotics are thought to be mycoplasmacidal, depending on the concentration. No enzymes responsible for aminoglycosides chemical modification have been observed in the *M. bovis* genome but decreased susceptibility in *M. bovis* has been linked with a mutation in the 16S rRNA genes [10].

**Molecular techniques for the detection of antimicrobials resistance genes**

The resistant molecular mechanisms to lincosamides, macrolides, tetracyclines, and fluoroquinolones were reported in *M. bovis* for the identification of mutations responsible for the high MICs of these antimicrobials [83]. The development of rapid genetic-based diagnostic techniques for the detection of *M. bovis* resistance is important in understanding the antibiotic resistance mechanisms, which helps in controlling the resistant *M. bovis* strains. Several studies have reported the utilization of various genetic techniques for the detection of *M. bovis* resistant strains such as PCR-oligonucleotide ligation [86, 87], PCR-restriction fragment length polymorphism (PCR-RFLP) analysis [88], a real-time PCR assay [89, 90], and DNA sequencing of target genes [83].

The PCR assay is a rapid and simple technique utilized for the detection of fluoroquinolone resistance in *M. bovis* via using specific primers targeting the gyrA gene and/or *parC* genes. Additionally, it has a higher sensitivity, specificity, and efficiency for diagnosis of *M. bovis*, when compared with conventional culture-based techniques [91]. Real-time polymerase chain reaction provides a more sensitive and quicker diagnosis, but it can detect only one *Mycoplasma* spp. at a time. Combining PCR with denaturing gradient gel electrophoresis (DGGE) can lead to the simultaneous detection of mixed mycoplasma cultures as well as new and uncultivable spp. [16, 64]. Real-time PCR technique, which is based on using hybridization probes has been used to detect the presence of mycoplasmas in mastitis milk samples and animal tissues within a few hours. Also it can distinguish between the closely related *Mycoplasma* spp., and detect *M. bovis* in the milk and lung tissue of cattle [62, 92]. Additionally, DNA sequencing is a direct and accurate technique used for detecting the mutations in QRDRs of specific genes such as gyrA, gyrB, *parC*, and *parE* for controlling the infections caused by quinolone-resistant *M. bovis* strains [93]. Recently, whole-genome sequencing is used to identify the potential new antimicrobial resistance mechanisms in *M. bovis*, but it is more expensive and takes a longer time [94].

**Conclusion**

*M. bovis* is the primary causative agent of bovine mycoplasmal diseases leading to important economic losses in the cattle industry due to its strong infectivity and severe disease associated signs such as pneumonia, mastitis, arthritis, otitis media, and keratoconjunctivitis. *M. bovis* become resistant to several antimicrobial classes such as macrolides and fluoroquinolones, which are the drugs of choice, utilized for the treatment of *M. bovis* infections. Therefore, detecting the mutations involved in decreased susceptibility of *M. bovis* to antimicrobial agents is important to easily select the appropriate antimicrobials for treatment. Moreover, this will help to
understand the antimicrobial-resistant mechanisms, which will enhance the treatment rates and stop the overuse of ineffective antimicrobial agents, and thereby controlling the *M. bovis* resistant strains.

**Conflict of interest**
None of the authors have any conflict of interest to declare.

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تعتبر الميكوبلازما بوفيس واحدة من أهم مسببات الأمراض التي تسبب العديد من أمراض الماشية الهامة مثل الالتهاب الرئوي، والتهاب الضرع والتهاب المفاصل. بالإضافة إلى ذلك، تعتبر الميكوبلازما بوفيس ثاني أكثر أنواع الميكوبلازما ضرادة بعد الميكوبلازما ميكويدس المسببة للالتهاب الرئوي المعدي في الأبقار.

الميكوبلازما بوفيس عبارة عن بكتيريا موجبة الجرام ليس لها جدار خلوي ولها إنتشار واسع في البيئة والعائل وتعتبر مسؤولة عن مشاكل اقتصادية مهمة في جميع أنحاء العالم مثل تقليل الإنتاج والاستعداد المبكر وزيادة نفوق الحيوانات المصابة. يعوق السيطرة على إنتشار الميكوبلازما بوفيس نقص اللقاحات والعلاجات الفعالة بسبب الإتجاهات المتزايدة في مقاومة مضادات الميكروبات وخاصة الفلوروكينولونات والماكروليدات وهي الأدوية المختصة في علاج عدوى الميكوبلازما بوفيس. وعلى النقيض من أحدث المقالات المشتركة التي تعتدل جاتان مهداً من الميكوبلازما التي تسبب الأبقار، فإن هذا المقال يوضح ويناقش شبه الخصائص العامة، تصنيف وضرورة الميكوبلازما بوفيس. وفي النهاية، يركز هذا المقال على مقاومة مضادات الحيوية لهذه الأمراض الاقتصادية.