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CONTAGIOUS CAPRINE PLEUROPNEUMONIA (CCPP) IN SMALL RUMINANTS: UPDATED REVIEW

[a,b]Ghulam Mustafa Solangi, [a]Zaheer Ahmed Nizamani, [c]Zainab Lanjar, [a]Barirah Rahman Talpur, [a]Naeem Aziz Soomro, [a,b]Muhammad Ali Chandio, [a]Ayaz Ali Panhwar, [d]Reema Bughio

[a] *Department of Veterinary Pathology, Sindh Agriculture University Tandojam, Pakistan*

[b] *Department of Veterinary Pathology, Shaheed Benazir Bhutto University of Veterinary & Animal Sciences, Sakrand, Pakistan*

[c] *Department of Veterinary Microbiology, Shaheed Benazir Bhutto University of Veterinary & Animal Sciences, Sakrand, Pakistan*

[d] *Department Animal Breeding and Genetics, Shaheed Benazir Bhutto University of Veterinary & Animal Sciences, Sakrand, Pakistan.*

A B S T R A C T

Contagious Caprine Pleuropneumonia (CCPP), a highly contagious Mycoplasmal respiratory disease affecting goats, poses a significant global threat. Sheep and goats are vulnerable to CCPP in various countries in Asia and Africa. The first occurrence of this sickness was recorded in Algeria in 1873. The cause of CCPP is *Mccp*. These bacteria are Mollicutes, a kind of organism with tiny genomes and galactan as food but no cell walls (0.58–1.35 Mb). They have a limited capacity for biosynthesis and can infect animals with a variety of diseases. This abstract outline the economic impact, historical context, epidemiology, pathogenesis, clinical manifestations, and diagnostic approaches associated with CCPP. One of the most serious and contagious diseases affecting goats is known as CCPP, in countries that use goat husbandry, especially in Africa, Asia, and the Middle East. It has caused significant losses in terms of economics, and there have been substantial morbidity and mortality rates, particularly for exotic breeds; a 100% morbidity rate and an 80% mortality rate have been observed in native and naive herds. The disease has the potential to spread to countries with no prior exposure through trade connections. CCPP is becoming a major concern to other countries that have either never experienced this highly infectious illness or are at risk of catching it due to frequent commerce with the afflicted countries or the geographically nearby neighborhood. Molecular assays are emerging as crucial diagnostic tools due to their high sensitivity and specificity. Understanding CCPP's history, impact, and diagnostic methods is essential for effective prevention, control, and management of this infectious disease.

Keywords: CCPP, Control and management, Clinical findings, Prevalence, Prevention, Small ruminants.

Corresponding Author: Ghulam Mustafa Solangi
Email: drsolangipathologist@gmail.com

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INTRODUCTION

Contagious caprine pleuropneumonia (CCPP) is a Mycoplasmal respiratory disease that quickly spreads among goats and affects a large number. Because CCPP is so important, steps are being taken all over the world to learn more about how to prevent, control, and better diagnose it. Agencies with international and national responsibilities, as well as reputable researchers, look at how diseases change over time on a regular basis. This is done to keep an eye out for emerging transboundary threats that, if they spread to disease-free countries, could harm uninfected goat populations in neighboring countries that work together (Nicholas & Churchward, 2012, Samiullah, 2013). This field involves a comprehensive study that encompasses progress in the fields of pathophysiology, epidemiology, prophylactics, and diagnostics, with a particular emphasis on future challenges, prophylactics, and diagnostics (OIE, 2017).

Economic Losses

One of the most serious and contagious diseases affecting goats is known as CCPP, in countries that use goat husbandry, especially in Africa, Asia, and the Middle East (OIE, 2014). It has caused significant losses in terms of economics, and there have been substantial morbidity and mortality rates, particularly for exotic breeds; a 100% morbidity rate and an 80% mortality rate have been observed in native and naive herds. (Thiaucourt & Bölske, 1996). According to estimates, the annual cost of CCPP in endemic regions is around US$507 million, resulting in significant economic losses. In addition to the expenditures associated with prevention, control, and treatment, economic losses are caused by sickness, death, and a deterioration in or loss of productive performance (Yatoo et al., 2018). Morbidity is a major obstacle to livestock management, which also impact commerce and transportation due to the high treatment costs. As a result of mortality, valuable animals are lost directly. In developed countries, ill animals are frequently euthanized, but this practice is not feasible in developing and underdeveloped countries. (Parray et al., 2019).

Countries Threat

CCPP is becoming a major concern to other countries that have either never experienced this highly infectious illness or are at risk of catching it due to frequent commerce with the afflicted countries or the geographically nearby neighborhood (Manso-Silvan et al., 2011; OIE, 2017). CCPP spreading in Russia and Europe is a persistent danger after Turkey, which borders Bulgaria and Greece as well as Georgia and Armenia on the Russian side (Dudek et al., 2022). There have been reports of outbreaks of *Mycoplasma* in Greece, but the cases have not been proven (OIE, 2008). The CCPP is endemic in domestic and wild animals in many parts of Africa, the Middle East, and Asia (Awan et al., 2010; Nicholas & Churchward, 2012). China and Tajikistan have reported CCPP (Wang et al. 2014). This illness has not been documented or is not known to exist in Oceania, North America, Central and South Europe (Nicholas et al., 2008; OIE, 2009; Dudek et al., 2022). Therefore, CCPP has lately been recognized as an emerging infectious illness and offers a hazard of a transboundary pandemic (Nicholas & Churchward, 2012). Preventive measures must be undertaken to control and prevent this endemic threat (Prats-van der Ham et al., 2015).

CCPP Outbreaks and Historical Perspectives

Sheep and goats are vulnerable to CCPP in various countries in Asia and Africa. The first occurrence of this sickness was recorded in Algeria in 1873. Prior to the discovery of *Mycoplasma* F38 in 1976, the only known cause of CCPP was *Mycoplasma mycoides capri* (Wesonga et al., 1993). Before MacOwan's research in 1976, *Mycoplasma* F38 was commonly believed to be the root cause of CCPP. According to MacOwan, Koch's postulate for CCPP can only be fulfilled by *Mccp*, previously known as the biotype F38, is the cause of the typical acute form of CCPP, according to Rurangirwa et al. (1987). In Kenya, this bacterium was first identified as a cause of CCPP. Later, it vanished from various African and Asian countries (Srivastava et al., 2010). When Thomas first described CCPP, also known as "bou frida" in Algeria, he implied that just one lung was impacted by the condition (Pettersson et al., 1998). Despite being ubiquitous, the illness was originally believed to be a meteorological phenomenon rather than a contagious disease. It was reported earlier that Contagious Caprine pleuropneumonia infected goats spread the disease that were imported from Turkey (MacOwan & Minette, 1976). It remained a mystery as to what caused CCPP for a long time. When scientists first started looking into the aetiology of CCPP since 1976 (McMartin et al., 1980), they found *Mycoplasma F38*. It was officially identified as *Mccp* in 1993 (Leach et al., 1993). According to several research teams, an experimental *Mccp* caused a disease with epidemic-like symptoms (Ostrowski et al., 2011). Thiaucourt & Bölske, (1996) study in studies conducted in France and by Hauksdottir et al. (1997) in India, *Mmc* was identified as a significant source of disease in goat herds. Since then, *Mmc* has been successfully isolated and cultivated in numerous countries worldwide (Manimaran et al., 2020). Between investigating the extensive destruction of goat husbandry caused by the CCPP in several countries. Between 1920 and 1930, Greece had a 98% morbidity CCPP outbreak (Stylianopoulos, 1933), however, it was later confined and eradicated (Nicholas & Churchward, 2012).

Epidemiology

Etiology

The cause of CCPP is *Mccp* (Leach et al., 1993). These bacteria are Mollicutes, a kind of organism with tiny genomes and galactan as food but no cell walls (0.58–1.35 Mb). They have a limited capacity for biosynthesis and can infect animals with a variety of diseases. (Razin et al., 1998). Different geographic regions are associated with each of the four *Mycoplasma* lineages. This genus encompasses a variety of species and subspecies, such as clusters of *Mycoplasma mycoides*, *Mycoplasma capricolum* subspecies *capricolum*, *Mycoplasma capricolum* subspecies *capripneumoniae*, *Mycoplasma mycoides* subspecies *capri*,

Mycoplasma mycoides subspecies *mycoides* in both large and small colonies, and *Mycoplasma* bovine group seven (Manso-Silvan et al., 2009). *Mycoplasmas* cause illnesses like these to sheep and goats and other pulmonary effects in some other animals. Contrary to earlier misconceptions, CCPP is not the same illness produced by *M. mycoides subsp. capri.* (Leach et al., 1989).

Taxonomic relationships among F38 group viz., *M. capricolum* and *M. capripneumonia* have been looked at by numerous studies (Cottew et al., 1987). There have been reports of the mycoides cluster, particularly *Mccp*, having a tight phylogenic connection (Ostrowski et al., 2011). Traditional biochemical, serological, and notably molecular tests, as well as culture and colony traits, may still be used to differentiate *Mccp* from other members (Maigre et al., 2008). *Mccp* strains F38, M1601, 9231-Abomsa, and ILRI181, as well as biochemical groups such as the organic acid-oxidizing group, have all been found (Soayfane et al., 2018).

Host

Domestic goats are particularly naturally sensitive animals to CCPP, and sheep may be impacted as well (Bolske et al., 1995; Yu et al., 2013). The innate sensitivity of numerous wild animal species to CCPP has also been documented (Dudek et al., 2022). CCPP has been documented in various wild species, including desert gazelles, Tibetan antelope, Arabian oryx, wild sheep, wild goats, and gazelles. Ruminants may be among the species found in natural reservoirs of CCPP (Lignereux et al., 2018).

Transmission

The primary mode of transmission is by inhalation of contaminated aerosols. Direct contact with afflicted animals is the primary cause of infection (OIE, 2017). According to Lignereux et al. (2018), airborne transmission has been reported to spread as far as 50 meters. The transmission of infected items, vectors, fomites, and animal products is unknown (Dudek et al., 2022). Pathogens may survive longer in damp, chilly, and crowded environments and may cause serious outbreaks. *Mccp* transmission is hampered by a shorter survival period (3–14 days) in the environment (OIE, 2008). *Mccp* can be found in frozen infected pleural fluid for up to ten years. (OIE, 2008). According to Justice-Allen (2010), moisture and humidity impact *Mycoplasma* survival and, therefore, transmission.

Risk Factors

Habitat suitability, pathogen concentration, breed, herd density, and animal immunity are all factors that may affect an organism's ability to persist in its environment or population of animals. Though the latest research indicates greater persistence in infected animals (weeks to months) and shorter persistence in the environment (hours to days), infection duration is highly variable (Wesonga et al., 2004). Due to its greater fragility, *Mccp* does not last as long outside of the animal or the environment. As a result, an organism will survive in its environment is less likely (Radostitis et al., 2009). Lack of a cell wall, a pathogen-related feature that may rapidly inactivate *Mccp*, and external causes such as UV radiation can also be the reason for the pathogen's worse environmental survival due to its greater fragility (OIE, 2008).

Mccp was isolated in experimentally infected goats on days 9 and 16 but not on any subsequent occasions (Lignereux et al., 2018). Animals that have been harmed but have survived are contagious until they fully recover (Moti & Bulto, 2024). The Figure 1 shows the modes of the *Mccp* transmission process (Yatoo et al. 2018).

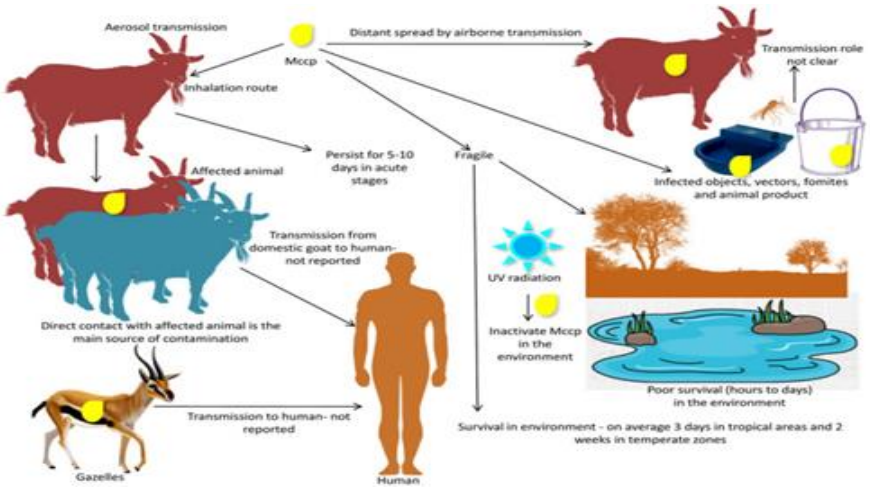

Figure 1. Transmission of *Mycoplasma capricolum* subsp. *capripneumoniae*.

Variability in Prevalence Studies

Mekuria et al. (2008) used serological testing to determine that the overall seroprevalence of CCPP was 16%. Asmare et al. (2016) meta-analysis research in Ethiopia found that the combined prevalence in field samples and slaughterhouse samples was 22%, for a net combined prevalence of 39%. According to Mbyuzi et al. (2014), seroprevalence ranged from 35 to 52 percent in goats and 23 to 36 percent in sheep in Southern Tanzania. *Mccp* has been isolated in 38% of herds in eastern Turkey, despite goats exhibiting varying incidence in different geographic locations. (Cetinkaya et al., 2009). In India, that varies from 5% to 64% (Ramdeva et al., 2008). There have been reports of prevalence ranging from 5% to 8% for non-descriptive mycoplasmosis (Udit Jain et al., 2015). In Pashmina goats from the Indian state of Jammu and Kashmir have a seroprevalence of roughly 10% (Parray et al., 2019). These studies provide evidence that prevalence varies by geographic location, screening technique, and animal type. However, even within the same region, variations in occurrence are seen, showing a range in the conditions of CCPP-affected goats throughout the country. Solangi et al. (2023) concluded that CCPP is well prevalent in Sindh province of Pakistan. The seroprevalence was found higher in females and adult goats and most of them were from Tapri breed. Among the four selected districts of Sindh province the seroprevalence of CCPP was the highest in Tharparkar district.

Risk factors may also impact prevalence (Kipronoh et al., 2016). According to PCR results from goats in Balochistan, Pakistan, Awan et al. (2010) reported 18% for *Mccp.* El-Deeb et al. (2017) also used targeted PCR and found 29% for *Mccp* in Saudi Arabia. Swai et al. (2013) observed herd-level seroprevalence of 3% and village level 32% in Tanzania using monoclonal antibody-based cELISA. In the Narok region of Ethiopia, the prevalence of CCPP varied from 6% to 90%, whereas it was only 15% in the Afar region. In the Borana pastoral regions of Ethiopia, severe CCPP lesions were observed in 10% of goats, with an overall prevalence rate of 13% (Ter Laak, 1992). In a global study, Peyraud et al. (2003) utilized a competitive ELISA method to estimate the CCPP seroprevalence. They found a seroprevalence rate of 15% both in Ethiopia and Mauritius. Solangi et al. (2023) revealed that CCPP is an endemic disease of goats in the Sindh Province. The disease in the studied population is caused by two species namely M. mycoides subsp. capri and M. capricolum subsp. capripneumoniae. The results indicate that there are multiple genotypes and species associated with this disease in the study areas as well as in the country which may vary depending on region as well as other risk factors such as husbandry practices or environmental conditions which can influence its spread or severity within an area or population group.

Morbidity Rates

Morbidity, or the proportion of sick animals, can reach 100%, exhibiting the disease's severe contagiousness (MacOwan & Minette, 1976). According to Rurangirwa & McGuire (1996), in field cases, a morbidity of 80–100% has been observed, although in experimental infections, all the goats typically get the illness, which is comparable to a morbidity of around 100% (Wesonga et al., 2004). According to Stylianopoulos (1933), the disease's morbidity had reached 98% in Greece and 90% in Eritrea by the year 2000. This shows that morbidity varies based on breed, prior exposure, or CCPP endemicity. Location and weather-related variations might also exist. The morbidity rates ranged from 5 to 30% at lower altitudes and from 56 to 68% at higher altitudes. This could be a result of the risk factor of the colder climate at higher elevations (Ali et al., 2023). Nearly 41% of the 24 herds in different regions of eastern Turkey (Cetinkaya et al., 2009). Morbidity in wild animals varies similarly, ranging from 83% to 100% in ibex and wild goats (OIE, 2017).

Mortality Rates

According to MacOwan & Minette (1976), the death rate may reach 90%, and the case fatality rate may vary from 60% to 100%. However, mortality is often around 80% (OIE, 2009). Though, it may range from 9% to 100% (Yatoo et al., 2018; Dudek et al., 2022). This variation is also caused by the study's sample size, the number of infected animals affected by the disease outbreak, the observation time, the observation region, and the observer are other factors that contribute to this difference. Animal mortality may also vary across domestic and wild animals, with first-exposure species frequently showing higher mortality (OIE, 2017). Lignereux et al. (2018) recently found an increase in mortality of 70% in affected sand gazelles. Low mortality may occur in certain epidemics or experimental investigations. In Pakistan, a 9% mortality rate was recorded in a Beetal goat pandemic region (Yatoo et al., 2018).

Public Health

No evidence exists that *Mccp* has affected humans (Spickler, 2015; OIE, 2017; Dudek et al., 2022).

Clinical Signs

The signs and symptoms of severe Contagious caprine pleuropneumonia include loss of body condition, thorax

pain, lying down, lethargy, nasal discharge, coughing, high fever (41–44 ^{0}C), dyspnea, depression, and anorexia. Morbidity and mortality are also 100% and 80–100%, respectively (OIE, 2014). Typically, respiratory symptoms develop 2-3 days following pyrexia (40.3-41.1 ^{0}C) (Radostitis et al., 2009). In clinical situations, other symptoms include productive coughing, pleurodynia, and wheezing or snoring. In the last stage, immobility, usually in a reclined position and, if upright, with a broad base and stretched neck (Tharwat & Al-Sobayil, 2017).

In addition, inspiratory dyspnea caused by inflammatory lesions or exudates may also be accompanied by grunting and snoring. Coughs are often uncomfortable, wet, and productive, worsening over time. A continuous nasal discharge that is first thick mucoid or purulent and rust-colored exudate followed by serofibrinous straw-colored exudate may be seen (Zinka et al. 2013). In pregnant animals, abortion occurs sporadically, and fever-induced anorexia is common (Wang et al., 2014). Clinical signs of CCPP are common in sensitive herds and are unaffected by age or gender. These indications are frequently associated with respiratory illness. During an outbreak, however, these clinical signs can vary from animal to animal depending on the species, the severity of the disease's per-acute, acute, or chronic phases, and other factors. It is difficult to make an accurate provisional diagnosis of the illness because of the diversity and uncertainty of the clinical signs (Shah et al., 2017). Solangi et al. (2023) observed moderate gross pathological changes in the trachea and lungs of experimentally infected goats with CCPP were congestion, thin fibrinous mucous plugs in the tracheal lumens, hemorrhages, purulent exudate, and enlarged mesenteric lymph nodes in the trachea. The lungs exhibited consolidations, enlarged bronchial and mediastinal lymph nodes, frothy and fibrinous exudates, straw-colored fluid filling the thoracic cavities, multifocal necrotic lesions and abscesses on the lung surface, and varying hepatization colors ranging from deep blue to reddish. Histopathological examination of trachea sections revealed severe CCPP infection. The respiratory ciliated epithelial layer erupted and erosion of lamina propria in the later days of infection. Histopathological examination of lungs sections revealed that in group A, interalveolar capillaries were severely congested, the bronchioles were flooded with fibrinous or serofibrinous exudate, the bronchioles were narrowed by exudate, and the alveolar gaps were filled with fluid.

Pathogenesis and Pathology

The most common pathological finding in CCPP is extensive lung consolidation, which is typically unilateral (Yatoo et al., 2018). Young, immune weak animals frequently exhibit acute pathologic lesions, whereas resistant and healthy animals typically experience chronic lesions (OIE, 2017).

The extracellular pathogens, known as *Mycoplasma*, are believed to be on mucous membranes and can adhere to epithelial cells (Nicolet, 1996). Pathogen adhesion to host cells promotes colonization and infection setup. *Mycoplasma's* metabolic processes produce free radicals, which can damage cell membranes and cilia. Similar to other *Mycoplasma* species, these occurrences might be related (Tanaka et al., 2014). This characteristic may be significantly influenced by a type of capsule present in various *Mycoplasma*s, notably the *Mccp* galactan (Nicolet, 1996). Immunity may be inhibited or suppressed by the antigens of *Mccp* stimulating immune cells. After stimulation, immune cells (macrophages and monocytes) produce pro-inflammatory cytokines like tumor necrosis factor alpha (TNF-a), interleukins, and interferon C are generated, but the exact mechanism for *Mccp* is still unknown (Totte et al., 2015). Along with this interaction and the production of cytokines are primarily mediated by Mycoplasmal structures. It is thought that *Mycoplasma*'s biological membranes, particularly the plasma membrane, and their constituent lipids and lipoproteins cause the release of cytokines (Nicolet, 1996).

A deeper investigation of the molecular basis of *Mccp*-specific pathophysiology is required in light of the afore-mentioned results. Figure 2 describes the pathophysiology of *Mccp* in detail (Yatoo et al. 2018).

Diagnosis

One of the most crucial and difficult parts of the illness is the diagnosis, which affects the preventative and therapeutic regimens and the control measures for halting worldwide disease expansion. After a preliminary clinical diagnosis, additional testing such as serological, biochemical, microbiological, PCR are necessary. Additionally, *Mccp* organisms need meticulousness, and special requirements make diagnostics issues. As a result, alternative diagnostic techniques should be used (Thiaucourt et al., 1996).

Sampling

An essential component of a precise illness diagnosis is appropriate sampling. Size, kind, and processing of the sample all rely on local feasibility, facilities, and availability. In an aseptic atmosphere, as many samples as possible should be collected using standardized techniques (OIE, 2014). Goats with classic CCPP symptoms had their nasal swabs, pleural fluid samples, and lung samples (from animals that had been necropsied) taken. After properly

cleansing the external nares, nasal swabs are collected and placed in a transport medium. After a thorough cleaning, hair is often cut, and skin is given a topical anesthetic. The required amount of pleural fluid is 10 ml. Hepatized pulmonary tissue samples are also acceptable. The damaged lung tissue must be separated into three-square-centimeter portions close to the surrounding healthy tissue (Thiaucourt et al., 1996). For serology, exudates, tissues, blood specimens are required. Blood samples can be obtained via the jugular vein, and serum can be extracted and kept at 4 C (El-Deeb et al., 2017). Tissue samples must be appropriately managed before further investigation for precise histopathology and organism isolation; lung sample processing is crucial (OIE, 2014).

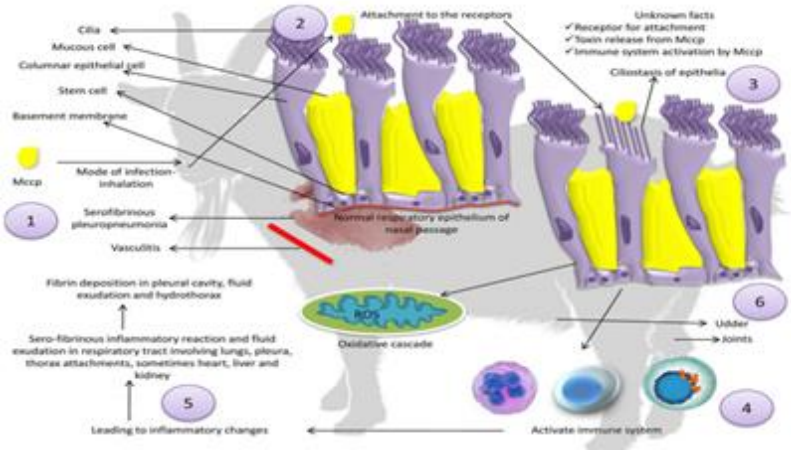

Figure 2: Pathogenesis of *Mycoplasma capricolum* subsp. *capripneumoniae.*

Haemato-Biochemical Parameters

A CCPP infection in goats was investigated for hematological changes by Kumar et al. (1994). Hematological signs are often not very helpful in diagnosing disease in field cases; however, anaemia and leukocytosis with subsequent leucopenia have been seen in animals with mycoplasmosis infection (Mondal et al. 2004). Compared to control goats, *Mycoplasma*-infected goats have been reported to have lower albumin and protein and higher levels of globulin, glucose, Ca, ALT, and AST (Mondal et al. 2004).

Sero-Molecular Tests

Molecular assays have become potential diagnostic methods for CCPP because of their high specificity and sensitivity and the challenges associated with cultivating *Mccp*. Globally, different molecular assays are used to diagnose CCPP. Their ability to diagnose mostly depends on antigens, antibodies, and genes or DNA that are either present in or against *Mccp*. They are primarily serological and nucleic acid amplification-based diagnostic techniques.

Molecular Tests (Gene/DNA based)

Mccp could be separated from the other *M. mycoides* cluster member using a DNA probe developed by Taylor et al. (1992). For the specific identification of *Mccp*, PCR-based diagnostic techniques have emerged as potential tools for the early and quick identification and correct distinction of members of the Mm cluster (Bashiruddin et al. 1994; Le Grand et al. 2004). Members of the Mm cluster have a genome with several distinctive characteristics, such as two rRNA operons (Sawada et al., 1984). They may be distinguished from other *Mycoplasma*s by their rRNA operons. However, even among the cluster members, distinct genes have been found in a few, such as the 16S rRNA genes of the two operons in *Mccp*, which aid in identifying these individuals.

For the phylogeny of the *Mycoplasma mycoides* cluster, Thiaucourt et al. (2000) sequenced a putative membrane protein gene. They noticed that *Mccp* may be quickly recognized by three nucleotide locations or by sequencing the 1298 bp long fragment. As a result of PCR invention for conventional and easy detection of particular DNA segments, new PCR procedures for *Mccp* have emerged. For *Mccp*, Woubit et al. (2004) developed a different PCR technique. The arcD gene was identified by this technique as a distinguishing DNA segment for *Mccp*. Prior *Mccp* detection methods relied on qualitative PCR-based testing. Q-PCR, however, was created by Lorenzon et al. (2008). In addition to being a quick and accurate test, it was a specialized RT-PCR assay for detecting and quantifying *Mccp*. Similarly, Righter et al. (2011) standardized RT-PCR

assays to identify *Mccp* and other members of the *Mycoplasma mycoides* cluster. Beyond their use on tissues, sensitivity and specificity prompted a few questions concerning PCR methods. Borang et al. (2024) employed loop-mediated isothermal amplification (LAMP) to explore the H2 gene sequences of *Mccp* in order to sensitively and quickly identify *Mccp*. It can detect *Mccp* in tissue and has a high level of sensitivity and specificity for *Mccp*.

CONFLICT OF INTEREST

The authors declare that there is no conflict in the publication of this article.

AUTHOR'S CONTRIBUTION

All the authors contributed equally.

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