

Original Article

Pathogenicity of local isolates of *Mycoplasma ovipneumoniae* and *Mycoplasma arginini* in experimental West African Dwarf goats

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ABSTRACT

Objective: This study was carried out to assess the pathogenicity of local isolates of *Mycoplasma ovipneumoniae* and *M. arginini* in West African dwarf goats (kids) in Nigeria.

Materials and methods: A total of 22 goats aged less than 1-year were purchased from markets. The goats were divided into six groups comprising of four experimental groups (EG; 4 in each) and two control groups (CG; 3 in each). The goats were fed *ad libitum* with standard diets and safe water. Groups EG1 and EG2 were infected with *M. ovipneumoniae* through trans-tracheal (TT) and intravenous (IV) routes, respectively, while those in groups EG3 and EG4 were infected with *M. arginini* through the same routes. Goats in groups CG1 and CG2 were inoculated with sterile *Mycoplasma* broth through TT and IV routes, respectively. In all cases, the amount of bacteria inoculated was 1.5×10^8 cells/mL. After the onset of the disease in goats, re-isolation of *Mycoplasma* was performed by culturing on mycoplasma agar supplemented with mycoplasma supplement G. The goats were monitored for 14 days post-infection (PI) to observe respiratory signs and mortality. Post-mortem (PM) examination was performed on each animal that died, while one surviving goat from each of the groups was sacrificed at 14 days PI for PM. After PM, histopathology was performed to observe the changes in tissues.

Results: Cough and nasal discharges were observed in all the experimentally infected goats seven days PI. Mortalities were recorded in goats in EG1 (two goats), EG2 (one goats), EG3 (two goats) and EG4 (one goat). At PM, pneumonic lesions were observed in the lungs of all the experimentally infected goats.

Conclusion: This study provides evidence that the local isolates of *M. ovipneumoniae* and *M. arginini* strains are pathogenic for goats in Nigeria.

KEYWORDS

Bronchopneumonia, Emphysema, Goat, *Mycoplasma*, Pathogenicity, Pneumonia

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INTRODUCTION

Many species of *Mycoplasma* are involved in the pathogenesis of pneumonic syndromes in small ruminants ([Bölske et al., 1994](#); [March et al., 2000](#); [Madanat et al., 2001](#); [Thomas et al., 2002](#); [Wesonga et al., 2004](#); [OIE, 2008](#); [Awan et al., 2009](#)). *Mycoplasma mycoides* subspecies *mycoides* (large colony type), *M. mycoides* subspecies *capri*, *M. arginini*, *M. ovipneumoniae*, *M. agalactiae* and *M. capricolum* are the commonly involved species for the mycoplasmal infections in small ruminants.

Mycoplasma species also cause other disease syndromes such as polyarthritis and mastitis ([Kinde et al., 1994](#); [Kusiluka and Kamarage, 1996](#); [Quinn and Markey, 2003](#); [Nicholas, 2005](#)) and contagious agalactia, which is characterised by arthritis, keratoconjunctivitis, pneumonia, and in females, mastitis and occasional abortion ([Madanat et al., 2001](#); [Peyraud et al., 2003](#); [Verbisck-Bucker et al., 2008](#)). However, *M. agalactiae* is the classical aetiological agent of the disease ([Nicholas, 2005](#)).

Despite reports incriminating *M. ovipneumoniae* ([Goltz et al., 1986](#); [Black et al., 1988](#); [McAuliffe et al., 2005](#); [Sharif and Muhammad, 2009](#); [Azizi et al., 2011](#)) and *M. arginini* ([DaMassa et al., 1992](#)) in diseases of small ruminants, many researchers ([Thiaucourt and Bolske, 1996](#); [Martrenchar et al., 1995](#)) believe that these mycoplasmas are saprophytes (non-pathogenic). These mycoplasmas are reported in the pathogenesis of pneumonic syndromes in small ruminants in different parts of the world ([OIE, 2008](#)). *M. ovipneumoniae*, as reported by [Nicholas et al. \(2008\)](#), can predispose animals to pasteurellosis and viral infections and it has been reported by [Azizi et al. \(2011\)](#) as an important agent of respiratory disease in sheep. According to the reports of [Ayling and Nicholas \(2007\)](#), *M. ovipneumoniae* causes atypical pneumonia and is frequently isolated from the lung, trachea and nose and occasionally from the eyes of sheep with pneumonia but can also be found in the respiratory tract of healthy sheep ([DaMassa et al., 1992](#)). Reports show growing evidence incriminating these agents in goat disease. [Egwu et al. \(2012\)](#) isolated *M. ovipneumoniae* from normal and pneumonic lungs of small ruminants in Maiduguri municipal abattoir, Borno State, Northeast Nigeria. [Akwuobu et al. \(2014\)](#) also isolated *M. ovipneumoniae* and *M. arginini* from the nasal cavity of small ruminants with and without nasal discharges in Benue State, North-central Nigeria. [DaMassa et al. \(1992\)](#) reported isolation of *M. arginini* from cases of ovine keratoconjunctivitis, pneumonic sheep lungs, mouth, and oesophagus by other researchers. In contrast, in an experimental trial by [Jones \(1985\)](#), *M. arginini* was not mastitogenic when injected into the lactating mammary gland of goats, but it

persisted in sheep udders at high titer for at least 9 days, causing lacteal neutrophilia without alteration in milk consistency and/or appearance ([DaMassa et al., 1992](#)). [Goltz et al. \(1986\)](#) also observed pathological lesions in the respiratory tract of goats inoculated with *M. arginini* in their study but did not attribute the lesions to *M. arginini*.

In spite of these reports of clinical cases involving *M. ovipneumoniae* and *M. arginini* in small ruminants, many researchers still consider these mycoplasmas as non-pathogenic organisms. Despite reports on the prevalence of these mycoplasmas, *M. ovipneumoniae* in particular, in northern parts of Nigeria, there is no information on the pathogenicity of the strains of these organisms in small ruminant in Nigeria. This investigation was carried out to determine, for the first time, the experimental pathogenicity of the strains of *M. ovipneumoniae* and *M. arginini* for West African Dwarf goats (kids) in Benue State, North-central Nigeria.

MATERIAL AND METHODS

Ethical approval: The experiment was performed with approval of the Animal Ethics Committee of the College of Veterinary Medicine, Federal University of Agriculture, Makurdi.

Experimental animals: Twenty-two (22) apparently healthy West African Dwarf goats (less than 1-year), obtained from goat markets within Makurdi, were used for the experiment. On arrival, the goats were housed in the large animal unit of the Veterinary farm, Federal University of Agriculture, Makurdi. The large animal unit was cleaned and disinfected before the arrival of the goats. The goats were vaccinated against peste des petits ruminants (PPR) 2 days after arrival. They were then assigned into six groups of four goats per group namely: groups EG1, EG2, EG3 and EG4, and three goats in groups CG1 and CG2. The groups were kept in separate compartments and were fed daily with cut grass. Drinking water was provided *ad libitum*. The goats were healthy and *Mycoplasma* organisms were not isolated from their nasal cavities for a period of 10 days prior to the experimental infection.

Inocula: Local isolates of *Mycoplasma* species from small ruminants (sheep and goats) were obtained from the Department of Veterinary Pathology and Microbiology, Federal University of Agriculture, Makurdi, Nigeria. These isolates were previously identified in the Veterinary Laboratory Agency (VLA), Weybridge, Woodham Lane, New Ham, Addlestone, Surrey, KT15 3NB using molecular methods according to Muzer et al. (1993) with minor modifications described by [McAuliffe et al. \(2005\)](#).

Extraction of DNA, 16S rDNA PCR and denaturing gradient gel electrophoresis (DGGE) were employed in the confirmation and speciation of the *Mycoplasma* isolates. Strains of *M. ovipneumoniae* and *M. arginini* were used as challenge organisms for the pathogenicity assay. The selected strains were isolated from small ruminants with nasal discharges. These strains were cultured five times in mycoplasma broth before used for the study. The preparation of the inoculum for each goat was done by obtaining a standardized suspension of whole cell broth culture (1.5×10^8 cells/mL) of each test *Mycoplasma* strain using spectrophotometer at OD₆₀₀. Aliquots of the broth cultures were streaked on mycoplasma agar to ascertain that the challenge suspensions were not contaminated.

Infection of experimental animals: The experimental infection procedure was performed according to [Wesonga et al. \(2004\)](#) with minor modifications. All goats in groups EG1 and EG2 were inoculated with the prepared suspension (1.5×10^8 cells/mL) of *M. ovipneumoniae* while goats in groups EG3 and EG4 were inoculated with *M. arginini*. Those in control groups CG1 and CG2 were inoculated with sterile mycoplasma broths. Following proper restraining, goats in groups EG1 and EG3 were respectively inoculated trans-tracheally with 1 mL suspensions of *M. ovipneumoniae* and *M. arginini*. One mL Tuberculin syringe (Jiangsu Jichun Medical Devices Co., Ltd, Zhenglu Industrial Park, Wujin, Changzhou, China) was used to deposit the inocula into the trachea after shaving and disinfection (with 75% ethanol) of a small area in the ventral region of the neck of the experimental goats. Goats in groups EG2 and EG4 were inoculated intravenously via the right jugular vein with 2 mL suspensions of *M. ovipneumoniae* and *M. arginini* respectively. Goats in CG1 received 1 mL of sterile mycoplasma broth (Oxoid®, CM0403B; Wade Road Basingstoke, Hampshire, RG24 8PW, UK) trans-tracheally while goats in CG2 were injected with 2 mL of sterile mycoplasma broth intravenously.

Sample collection: The body temperature and clinical signs attributable to pneumonia were recorded. Nasal swabs were collected as soon as respiratory signs were observed in the goats, for mycoplasmal isolation. From each group, one surviving kid was humanely sacrificed two weeks post-infection (PI) for post-mortem examination with a detailed examination on the respiratory tract. Kids that died earlier also underwent post-mortem examination. Samples of the trachea and the lungs were also collected for mycoplasmal isolation and for histological examination.

Isolation of *Mycoplasma*: Nasal swabs and lung tissues collected from the experimentally infected animals were cultured for *Mycoplasma* organisms by inoculation onto mycoplasma agar base (Oxoid®, CM0401B; Wade Road Basingstoke, Hampshire, RG24 8PW, UK) supplemented with mycoplasma supplement G (Oxoid®, SR0059C; Wade Road Basingstoke, Hampshire, RG24 8PW, UK).

Histopathology: Trachea and lung tissues for histopathological examination were processed using standard operating procedure for manual tissue processing as described by Drury and Wallington (1980). The slides were viewed under light microscopy and their photographs were taken.

RESULTS

All the goats were healthy and were eating well at the time of inoculation and those that were given trans-tracheal inoculation coughed for a few seconds after administration of the inocula. Mild fever was observed in goats in group EG1 four days PI. The fever persisted for 7 days. None of the goats in the other groups developed high body temperature throughout the experimental period. Rectal temperature variations of less than 1°C were recorded in all the goats in all groups except one in group EG3 with rectal temperature variation of 2.7°C below the initial rectal temperature (**Table 1**). Mean rectal temperature values of the goats within and between the groups were not statistically different ($P > 0.05$) throughout the experimental period (**Table 1**).

After day 3 post-inoculation, all the goats in group EG1 were found to have mucoid nasal discharges. Goats in this group were depressed and inactive on day 7 post-inoculation (**Figure 1, Panel A**). Between days 5 and 7 post-inoculation, the goats in groups EG2, EG3 and EG4 were seen to have nasal discharges. Also the goats in groups EG1, EG3 and EG4 were found to have ocular discharges (slight conjunctivitis) 7 days PI (**Figure 1, Panel B**).

Mortality was recorded in all the experimentally infected groups (**Table 2**). Six goats were died within 10 days of the experimental infection. By day 10 PI, two of the goats infected trans-tracheally and one of the goats infected intravenously with *M. ovipneumoniae* died. Similarly, two of the goats infected trans-tracheally and one of the goats infected intravenously with *M. arginini* died by day 10 PI. No mortality was recorded in the control groups.

No apparent gross lesions were observed in the lungs of goats in the control groups. Post-mortem examination

Table 1: Mean rectal temperature (°C) West African Dwarf goats (kids) inoculated with local *Mycoplasma* isolates

Experimental groups/ Inoculum	Animal no.	Rectal temperature (°C)				
		PrI	4 days PoI	7 days PoI	10 days PoI	IMRT
EG1 (IT): <i>M. ovipneumoniae</i>	2	37.5	37.7	38.2	37.4	37.7
	7	39.3	40.0	40.2	37.8	39.3
	3	38.4	38.0	39.2	-	38.5
	10	37.2	37.9	-	-	37.6
	GMRT±SD	38.1±0.9	38.4±1.1	39.2±1.0	37.6±0.3	
EG2 (IV): <i>M. ovipneumoniae</i>	15	37.5	37.2	37.9	36.7	37.3
	14	39.3	38.2	38.5	-	38.7
	17	38.2	37.8	38.5	37.6	38.0
	6	38.6	38.2	39.6	38.5	38.7
	GMRT±SD	38.4±0.8	37.9±0.5	38.6±0.7	37.6±0.9	
EG3 (IT): <i>M. arginini</i>	18	39.0	39.0	38.8	39.0	39.0
	4	38.1	37.2	37.3	37.1	37.4
	20	38.6	35.9	-	-	37.3
	6	38.4	-	-	-	30.4
	GMRT±SD	38.5±0.4	37.4±1.6	38.1±1.1	38.1±1.3	
EG4 (IV): <i>M. arginini</i>	24	38.2	38.8	38.8	38.3	38.5
	22	37.7	38.2	-	-	38.0
	25	38.1	38.0	38.5	38.8	38.4
	21	39.2	39.5	39.9	39.9	39.6
	GMRT±SD	38.3±0.6	38.6±0.8	39.1±0.7	39.0±0.8	
CG1 (IT): Sterile broth	5	38.1	38.2	38.1	38.0	38.1
	19	37.5	38.3	38.6	38.5	38.2
	13	38.5	38.6	38.5	38.7	38.6
	GMRT±SD	38.0±0.5	38.4±0.2	38.4±0.3	38.4±0.1	
CG2 (IV): Sterile broth	23	38.2	38.4	38.8	38.3	38.4
	12	40.0	39.4	40.2	38.8	39.6
	26	39.3	38.3	39.4	37.7	38.7
	GMRT±SD	39.2±0.8	38.7±0.9	39.5±0.6	38.3±0.8	

PrI = Pre-Infection; PoI = Post-Infection; GMRT = group mean rectal temperature; IMRT = individual mean rectal temperature.

Table 2: Pathogenicity of *Mycoplasma* species in West African Dwarf goats (kids)

Inoculum	Groups	Mortality of kids at days post-infection												Total
		1	2	3	4	5	6	7	8	9	10	11	12	
<i>M. ovipneumoniae</i>	EG1 (IT)	0/4	0/4	0/4	0/4	1/4	0/4	0/4	0/4	0/4	1/4	0/4	0/4	2/4
	EG2 (IV)	0/4	0/4	0/4	0/4	0/4	0/4	0/4	1/4	0/4	0/4	0/4	0/4	1/4
<i>M. arginini</i>	EG3 (IT)	0/4	0/4	0/4	1/4	0/4	1/4	0/4	0/4	0/4	0/4	0/4	0/4	2/4
	EG4 (IV)	0/4	0/4	0/4	0/4	1/4	0/4	0/4	0/4	0/4	0/4	0/4	0/4	1/4
Sterile broth	CG1 (IT)	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3
	CG2 (IV)	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3	0/3

* = Number dead/Number infected

revealed pneumonic lesions in the lungs of all the experimentally infected goats (**Figure 2, Panels A-D**). The lesions ranged from mild to severe congestion and edema. The two goats necropsied in group EG1 showed severe gross lung lesions. The entire lungs of goat no. 3 that died 10 days PI in this group were severely congested with mild edema (**Figure 2, Panel A**). Slight adhesion of the pleural surface to the wall of the thoracic cavity was observed in goat no. 7 that was sacrificed in the same group (**Figure 2, Panel B**). The lungs revealed mild edema and severe congestion of the cranial and medial

lobes of the lungs and diffuse nodules in the caudal lobes of the lungs. In group EG2, the entire lungs of the goat no.14 that died 8 days PI were severely congested while the lungs of the sacrificed goats (n=15) were edematous, congested and showed diffuse marbling (**Figure 2, Panel C**). The lungs of goats (n=20) that died 6 days PI and that of the sacrificed goats (n=4) (**Figure 2, Panel D**) in group EG3 showed mild edema and congestion. Lungs of goat no. 22 that died 5 days PI were edematous and congested while the sacrificed goat (n=21) in group EG4 had mildly congested lungs.



Figure 1: Kids experimentally infected with local isolates of *Mycoplasma* species (Panels A and B). A) Infected with *M. ovipneumoniae*, inactive and depressed 7 days post-infection. B) Ocular discharges (arrow) in a kid experimentally infected with *M. arginini* 7 days post-infection.

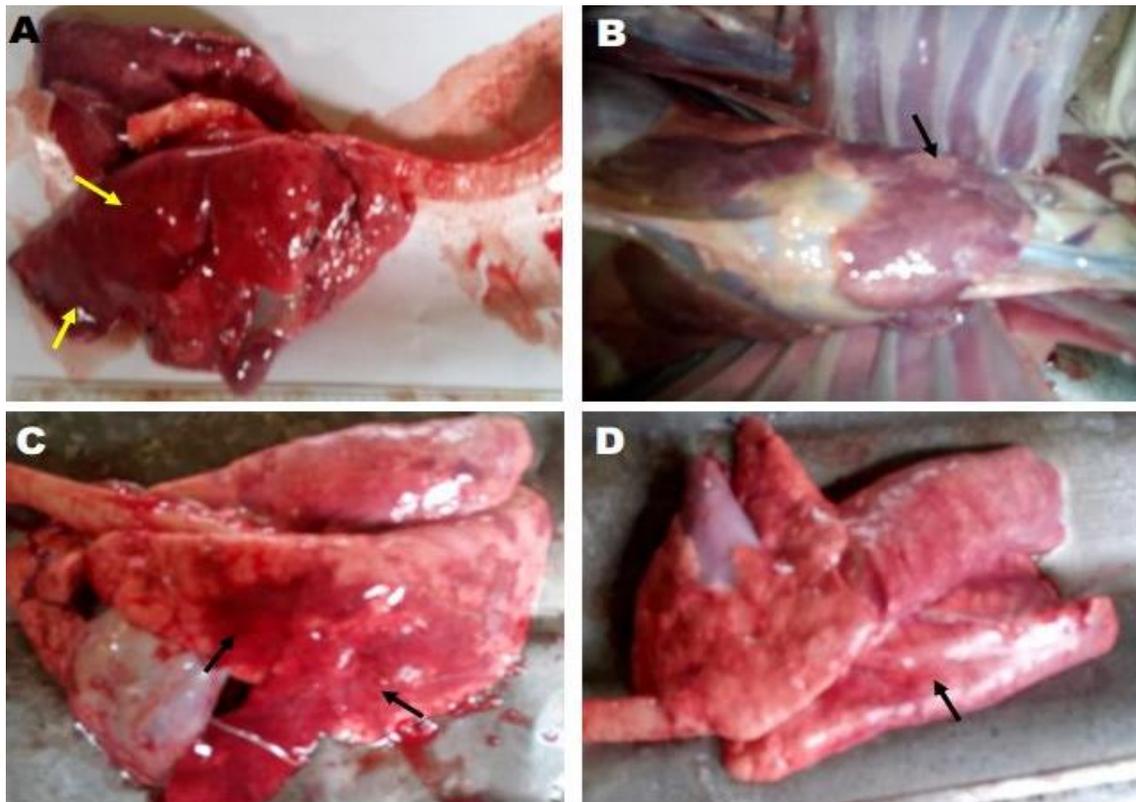


Figure 2: Gross lesions in lungs of kids experimentally infected with the local isolates of *Mycoplasma* species (Panels A, B, C, and D). A) Severe congestion (arrows) in lungs of kid experimentally infected with *M. ovipneumoniae*, 10 days post-infection (PI). B) Adhesion of pleural surface to the thoracic wall (arrow) in kid experimentally infected with *M. ovipneumoniae*, 12 days PI. C) Congestion and edema (arrows) in lungs of kid experimentally infected with *M. ovipneumoniae*, 12 days PI. D) Mild edema and congestion in lungs of kid infected with *M. arginini*, 12 days PI.

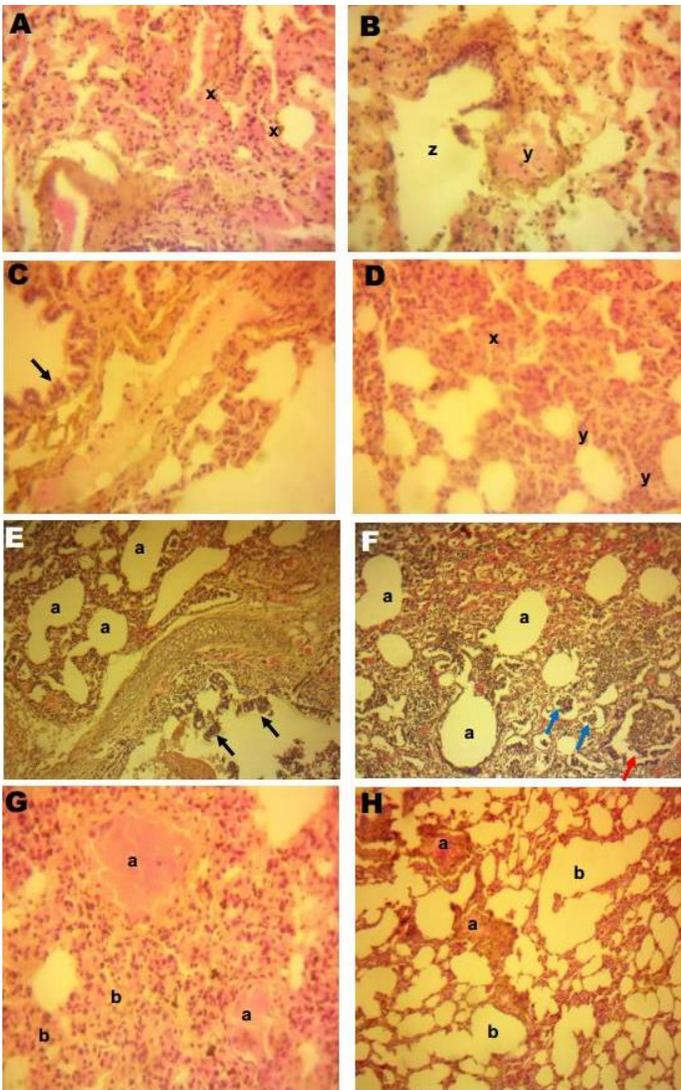


Figure 3: Histological lesions in lungs of kids experimentally infected with the local isolates of *Mycoplasma* species (Panels A-H). Group EG1: Interstitial pneumonia involving edema with infiltration of leukocytes in the lungs of infected goat with *M. ovipneumoniae* (Panels A, B and D). A and D– alveoli containing edema fluid with infiltration of leukocytes (x). B– extended alveolus (z) with lymphocyte infiltration and alveolus (y) containing edema fluid and few leukocytes. C) Bronchopneumonia involving edema and thickening of the bronchial epithelium (arrow) due to infiltration of neutrophils. D) thickening of intraalveolar septa due to infiltration of neutrophils (y). (H&E 400x). Group EG2: Lungs of experimentally infected goat with *M. ovipneumoniae* (Panels E and F). E) emphysema (a), bronchopneumonia involving infiltration of the bronchial epithelium with neutrophils (b) and necrosis of the epithelial cells of the bronchus (black arrow). (H&E 40x). F) infiltration of alveoli (blue arrow) and bronchiole (red arrow) with neutrophils. (H&E 100x). G) EG3: Acute interstitial pneumonia in the lungs of goat infected with *M. arginini*. Note: hyperaemia of pulmonary capillaries (a), severe infiltration of leukocytes in the interstitial tissues and alveoli (b). (H&E 400x). : H) Group EG4: Lungs of experimentally infected goat with *M. arginini*. Note: mild pneumonitis involving Congestion (a) and distended alveoli (b) with mild infiltration of the interstitial tissues with leukocytes. (H&E 100x).

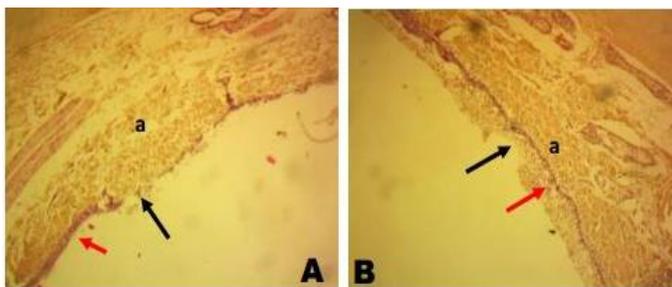


Figure 4: Trachea of goat experimentally infected with local isolates of *Mycoplasma* species (Panels A and B). Note: tracheitis with leukocyte infiltration (red arrow), disruption of the columnar epithelium of the trachea (black arrow), and edema of lamina propria (a). (H&E 100x).

Histologic lesions observed in the lungs of the experimentally infected goats ranged from mild to severe bronchitis and bronchiolitis involving mild to severe edema, congestion, emphysema and infiltration of lung tissues with leukocytes (**Figure 3, Panels A-H**). No histologic lesions were observed in the control groups. The histopathological changes observed in the lungs of goats in group EG1 were characterized by mild interstitial pneumonia occurring in foci and mild edema and

congestion with focal infiltration of leukocytes (**Figure 3, Panels A, B**). Alveoli containing edema fluid with infiltration of leukocytes and areas of emphysema were observed. Thickening of bronchial epithelium, which was heavily infiltrated with neutrophils, and mild infiltration of bronchiole and lamina propria with neutrophils and mononuclear inflammatory cells were also observed in this group (**Figure 3, Panels C, D**).

In group EG2, the lesions comprised of broncho-pneumonia, bronchiolitis and emphysema. The bronchial epithelium, bronchiole and alveoli were infiltrated with neutrophils with necrosis of the epithelial cells of the bronchus (**Figure 3, Panels E, F**).

The histologic lung lesions observed in goats in group EG3 included acute interstitial pneumonia, hyperaemia of pulmonary capillaries, edema, congestion, emphysema and severe infiltration of leukocytes in the interstitial tissues and alveoli (**Figure 3, Panel G**).

In group EG4, the histologic lung lesions observed in the goats were mild pneumonitis involving congestion, distended alveoli and mild infiltration of the interstitial tissues with leukocytes (**Figure 3, Panel H**).

The histologic lesions observed in the trachea of the experimentally infected goats comprised of mild and severe tracheitis. The lesions were characterized by infiltration with leukocytes and disruption of the pseudostratified ciliated columnar epithelium, necrosis and edema of the lamina propria of the trachea (**Figure 4, Panels A, B**).

DISCUSSION

The pathogenicity assay, using goats (kids), showed that the strains of *M. ovipneumoniae* and *M. arginini* tested were pathogenic. Clinical signs and mild to severe pneumonic lesions were observed in all the experimentally infected kids. The pathogenicity of *M. ovipneumoniae* recorded in this study agrees with reports of [Goltz et al. \(1986\)](#), [Azizi et al. \(2011\)](#) and [Black et al. \(1988\)](#). The response of goats with mild fever, 4 days PI in group infected with *M. ovipneumoniae* trans-tracheally, agrees with the report of [Goltz et al. \(1986\)](#) who inoculated kids endo-bronchially with *M. ovipneumoniae*.

The mild conjunctivitis observed in some of the experimentally infected goats in this present study supports the reports of [Nicholas \(2002\)](#), [McAuliffe et al. \(2005\)](#), and [Sharif and Muhammad \(2009\)](#) that many *Mycoplasma* species are associated with respiratory diseases, arthritis, mastitis, and conjunctivitis.

Gross and histological lung lesions, *viz.*, congestion, edema, bronchitis, bronchiolitis, emphysema and alveolitis, seen in our experimental goat kids were similar to the findings of [Goltz et al. \(1986\)](#); they reported development of clinical signs of disease with severe respiratory tract lesions in goats inoculated endobronchially with *M. ovipneumoniae*. Variation in susceptibility in the experimentally infected goats observed in this study paralleled the report of [Goltz et al. \(1986\)](#). However, the mortality

recorded in this present study was not recorded in the study of [Goltz et al. \(1986\)](#). This difference could be attributed to the degree of virulence of our *M. ovipneumoniae* strain. The results of [Azizi et al. \(2011\)](#) and [Black et al. \(1988\)](#) further suggested that *M. ovipneumoniae* is an important agent in respiratory diseases of SR. [Azizi et al. \(2011\)](#), in their study of the role of *M. ovipneumoniae* in pneumonic lungs of slaughtered sheep associated with *M. ovipneumoniae* reported the following pathological findings which agree with our findings: suppurative broncho-pneumonia, fibrinous broncho-pneumonia and interstitial pneumonia. [Meigs \(2010\)](#) similarly reported that *M. ovipneumoniae* infection in lambs caused infiltration and expansion of bronchial/bronchiolar submucosa by dense cuffs of moderate to large numbers of lymphocytes with macrophages, plasma cells, eosinophils and neutrophils. The presence of inflammatory cells within the respiratory epithelium, adjacent pulmonary interstitium, parenchyma and in the alveolar spaces were observed by [Meigs \(2010\)](#).

The findings in this study disagree with the reports of [Martrenchar et al. \(1995\)](#) and [Thiaucourt et al. \(1994\)](#) that *M. ovipneumoniae* was not pathogenic. In their work on the isolation and experimental study of *M. mycoides* subsp. *mycoides* LC and *M. ovipneumoniae* in goats in northern Cameroon, [Martrenchar et al. \(1995\)](#) reported that *M. ovipneumoniae* was not pathogenic despite its multiplication in the upper respiratory tract (nasal sinuses of goats) following experimental infection. However, they acknowledged that the type of strain, the inoculation route, previous exposure, the concentration and nature of the inoculum are factors that affect pathogenicity ([Martrenchar et al., 1995](#)).

The goats inoculated with *M. arginini* developed clinical signs similar to those of *M. ovipneumoniae*. Though fever was not observed in any of these groups, a goat in the group infected with *M. arginini* trans-tracheally developed hypothermia and died 6 days PI. This was in contrast to the report of [Zamri-Saad et al. \(1994\)](#) that the body temperature of all the goats inoculated with *M. arginini* remained normal throughout the study period. The gross and histologic lesions observed in goats inoculated with *M. arginini* in this study agree with the reports of [Zamri-Saad et al. \(1994\)](#) where slight congestion and moderate edema were recorded. They reported moderate bronchiolitis consisting of accumulation of a mixture of mononuclear cells and neutrophils in the subepithelial layer; the bronchiolar associated lymphoid tissue (BALT) was slightly hyperplastic and edema fluid was found in most alveoli. The interalveolar septa were thickened due to hyperaemia and the presence of neutrophils and mononuclear cells. [Zamri-Saad et al. \(1994\)](#) also

demonstrated that *M. arginini* can produce only mild lesions in the lung of goats and thus this may not lead to a serious disease. According to [Zamri-Saad et al. \(1994\)](#), the organism appeared to be eliminated early as a result of the inflammatory response following the infection. The findings in the group inoculated with *M. arginini* intravenously, in this study, agree with [Zamri-Saad et al. \(1994\)](#). In this group, mild lesions were observed in the lungs and only one goat died in this group 5 days PI. [Goltz et al. \(1986\)](#) also observed pathological lesions in the respiratory tract of goats inoculated with *M. arginini* in their study but did not attribute the lesions to *M. arginini*. They observed leukocytosis and increased fibrinogen level in the experimental goats and attributed these to pathophysiological response to the challenge. In this present study, the group inoculated trans-tracheally with *M. arginini* did not only show clinical signs of the respiratory tract, 2 out of the 4 goats in this group died within the experimental period suggesting that *M. arginini* is pathogenic. Of the many *Mycoplasma* species, *M. arginini* is regarded as non-pathogenic ([Jones, 1985](#); [Azizi et al., 2011](#)) which prevails in goats and sheep and is isolated from various sites ([Jones, 1985](#)).

In this study, it was observed that mortality was high in the trans-tracheally infected goats than in the intravenously inoculated groups irrespective of the *Mycoplasma* species used. This finding suggests that the route of infection increases the virulence of *Mycoplasma* species. The deposition of *Mycoplasma* strains directly into the respiratory tract of the experimental goats might have enhanced the virulence of these strains in the respiratory system; while intravenous inoculation exposed these organisms to destruction by the body defence system before they could get to the respiratory system to initiate pneumonic lesions. However, this contradicts the report of [Martrenchar et al. \(1995\)](#) who recorded absence of pathogenicity for *M. ovipneumoniae* despite infecting the experimental goats intravenously and trans-tracheally.

In the present study, similar pathologic lesions were observed in the trachea irrespective of the route of experimental infection and the *Mycoplasma* species used as inocula. None of the earlier researchers documented any pathology in the trachea following experimental infection with *Mycoplasma* species.

CONCLUSION

In conclusion, this is the first report on the pathogenicity of mycoplasmas in small ruminants in Benue State, North-central Nigeria. The demonstration of the pathogenicity of local isolates of *M. ovipneumoniae* and *M. arginini* strains from small ruminants in the study area, contrary to

the reports of many researchers in different parts of the world, should generate strong interest in research in the epidemiology, pathogenicity and pathogenesis of these species of *Mycoplasma* in Nigeria.

CONFLICT OF INTEREST

None of authors have any conflict of interest.

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CONTRIBUTIONS OF AUTHORS

Akwuobu, Chinedu Adivo: Carried out the field and laboratory works. Chah, Kennedy Foinkfu and Oboegbulem, Stephen Ike: were the supervisors. Rabo, Jude S.: was the pathologist that read the histopathology slides

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