

# [Molecular detection of virulent mannheimia haemolytica and pasteurella multocida ...](https://assignbuster.com/molecular-detection-of-virulent-mannheimia-haemolytica-and-pasteurella-multocida-in-lung-tissues-of-pneumonic-sheep/)

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Detection of diverse serotypes of B. trehalosi has been attained from sheep with systemic pasteurellosis. Susceptibility findings of pasteurellae were more or less similar to the findings reported as earlier. Reduced responsiveness of aminoglycosides (amikacin and gentamicin) might be due to their selective therapeutic uses for clinical cases.

Virulence-associated genetic elements among M. haemolytica might be homologous to their PHSSA gene. In stressful milieu, the PHSSA gene after expression induces the commensal or non-pathogenic M. haemolytica to be pathogenic to sheep and hence, the PHSSA is reportedly supposed to be a species-specific and virulence-linked component of these bacteria. The gene Rpt2 encoding for methyltransferase has been analyzed and found with the multiple and variable quantum repeats of CACAG which could have potentiated the pathogenic ability of carrier bacterial serotypes. Variability in Rpt2 sequences might have resulted in the genesis of functionally diverse methyltransferase which possibly promoted the virulence of M. haemolytica serovars. Proteolytic enzymes and outer structures such as adhesin, capsular-lipopolysaccharide and the OMPs peculiar to these important microbes help their colonization and exhibit the host defense evasion to initiate the lung pathology. Thus, the Rpt2, PHSSA and PlpE are reportedly the crucial genetic components that supplement to the infection progression ability of carrier bacterium and these bacterial genetic constituents aid to encounter the sheep with advanced and overt pneumonia. Likewise, the lipopolysaccharide moiety of the capsule produced by P. multocida resists the phagocytosis and exhibits the pneumonia progression by lowering the host immunocompetency. Pasteurellae lipopolysaccharide constitutes a shielding antigen and stimulates the humoral immunity of the diseased or infected animals. Thus, the P. multocida requires a unique lipopolysaccharide moiety of the capsule for its expansion in-vivo to establish the clinical bronchopneumonia in sheep.

Diverse sequences of OMP genes might have terminated into the translation of altered surface-exposed epitopes. The Omp87 gene encodes a structurally and functionally significant outer protein specific to P. multocida. This protein also acts as an adhesin needed for bacterial colonization on the mucosa of the pulmonary system. By potentiating the pathogenic capability of P. multocida and their colonization on the pulmonary mucosa, these OMPs lead to the progression of bacterial enzootic pneumonia in sheep. Bacterial OMPs enable the ionic transport or entry of energy molecules across their membrane, in summation to their important role in colonization, host defense evasion and productive infection. The OmpH is a unique protein specific to all serovars of P. multocida. Variable sequences of OmpH gene and their translation into the diverse surface epitopes are significantly paramount to P. multocida adaptation to its host environment. These bacterial surface markers are exposed to different selection pressures or environmental insults and modulated themselves to safeguard the host bacterium under harsh environment or changing ecological niches. Therefore, the genes encoding pathogenicity markers were targeted to assess the intra-species heterogeneity and for establishing the epidemiological relationship.

## Conclusions

Acute fibrinous-bronchopneumonia was predominantly found to be connected with high mortality of lambs and adult sheep. Virulent M. haemolytica or P. multocida isolates sensitive to commonly used antimicrobials were recovered from the pulmonary tissues or the heart blood of sheep and lambs with divergent pneumonia. Different molecular elements accountable for the invasive role of the causative bacterial agents were detected which might have affected the prognosis of ovine fatal pneumonia.