**Pasteurellosis and haemorrhagic septicaemia**

**Introduction**

Pasteurellosis in ruminants is usually regarded as the respiratory disease caused by *Mannheimia (Pasteurella) haemolytica*, but *Pasteurella multocida* also causes significant disease in ruminants. Respiratory infections are common in South Africa, and haemorrhagic septicaemia is a problem in border areas. Game animals suffer from pneumonic *Pasteurella multocida* infections in the same way as farmed ruminants, but to a lesser extent, as pasteurellosis is a disease of intensification. Haemorrhagic septicaemia in buffaloes has recently been detected in South Africa, which is concerning, as many game farms are found in border areas. The mortality rate for Haemorrhagic Septicaemia in non-endemic areas can be from 20 – 98%.

The whole *Pasteurella* group has undergone a taxonomic revision in recent years. This revision went hand in hand with the detection of toxins and other virulence factors, as all were based on elucidating various molecular differences and similarities. These changes have resulted in a better understanding of the pathogenesis, as well as in improved methods of control.

**Aetiology and epidemiology**

Using molecular methods, it has been found that the *Pasteurella* group is far more divergent than originally thought, and the members do not form only one genus. *Pasteurella multocida* was described first, and it therefore keeps the name *Pasteurella*. Most of the original *Pasteurella haemolytica* strains are now known as *Mannheimia*, which has been split into *M. haemolytica*, *M. granulomatis*, *M. glucosida*, *M. ruminalis* and *M. varigena*. Sheep were the likely original hosts of *Mannheimia haemolytica*, and only certain clones spread to other ruminants. The strains previously known as *P. haemolytica* T strains are now known as *Bibersteinia trehalosi* and they are pathogenic in sheep. They include the old serotypes 3, 4, 10 and 15. The leukotoxin mosaic is found in some of these species, especially *M. haemolytica*, *M. glucosida* and *B. trehalosi*.

The virulence of the *Pasteurella / Mannheimia / Actinobacillus / Haemophilus / Histophilus* group depends on the capsule [especially *Pasteurella* and some *Haemophilus* strains], lipopolysaccharide, outer membrane protein toxins [especially for Porcine Atrophic Rhinitis], RTX [repeats in toxin] families of toxins, neuraminidase, glycoproteins and proteases.

The primary virulence factor varies with the animal species concerned. The most important RTX toxin is leukotoxin produced by *Mannheimia haemolytica*. The capsule is the main virulence factor in *Pasteurella multocida*. Some of the components of the capsules are similar to normal host molecules. The microbe employs microbial mimicry of normal host molecules, so that the capsule is not recognized by the immune system, and antibodies are not produced.
*Pasteurella multocida* is divided into 5 capsular types, A, B, D, E and F [The original C was not a separate type, and so it is left out]10. Typing according to the capsule is more useful for distinguishing pathogenic mammalian strains, and somatic typing is more useful for typing pathogenic avian strains, but both may be combined to identify especially haemorrhagic septicaemia strains3. Somatic types run from 1 – 16. Somatic type 2 is associated with haemorrhagic septicaemia.

All strains are potentially pathogenic in all animals, but in practice, most disease syndromes are associated with certain capsular or somatic types. Ribotyping holds promise for more accurate typing in the future7.

*Pasteurella multocida* is found less frequently than *M. haemolytica* in both cattle and sheep [unless vaccinated], and is usually due to type A in ruminants. Type D is also present in ruminants, but is far less common. If animals are effectively vaccinated to protect them from *M. haemolytica* infections, *P. multocida* infections become correspondingly higher. A study during 2011 yielded *M. haemolytica* in 23% of cases, and *P. multocida* in 33% from feedlot cattle that had mostly been vaccinated with a leucotoxin vaccine11.

Types B and E are associated with Haemorrhagic Septicaemia, which causes losses in cattle and water buffalo in tropical Africa and Asia3. Water buffaloes are more severely affected. The strains are mostly B:2 and E:2. Only type B is found in Asia, and type E is more common than B in Africa. Other ruminants such as bison, fallow deer, pigs, horses, donkeys, Indian elephants and camels may rarely become infected with Haemorrhagic Septicaemia3.

Of the two types causing haemorrhagic septicaemia, types B and E are rarely isolated in South Africa, but are present in Namibia and Zimbabwe3,9,13. The cases that have occurred in South Africa have generally been in border areas, in cattle [E]3 and buffaloes [B]. There have been several cases of type B in buffaloes in Limpopo, North West and Gauteng since 2008 (J Steyl, University of Pretoria, pers. Comm., 2011). As haemorrhagic septicaemia is so rare in South Africa, it is listed as reportable (not notifiable) to the State. Types A and D are common and are not reportable. Reportable diseases are ones not normally present in South Africa, which could cause serious losses in livestock.

Transmission of *P. multocida* is via aerosols or contact with especially nasal discharges and saliva, but urine, faeces and milk can also be responsible10.

**Clinical Signs**

*Mannheimia haemolytica*, *Pasteurella multocida* and *Histophilus somni* are present in small numbers as part of the bacterial flora of the upper respiratory tract. The animal’s normal bodily defenses keep these bacteria in check10,11. Pneumonia may follow, when the defence mechanisms are compromised. This can include damage to the cell linings of the upper respiratory tract by viruses such as IBR, PI3 and
BRSV. Damage to the tracheal lining could also occur due to inhaled irritants such as exhaust fumes or dust. The respiratory defense mechanism can also be depressed due to immuno-suppression associated with BVD. 

Initial signs of bronchopneumonia in feedlot cattle and enzootic pneumonia in calves affected by types A and D are vague and are often limited to a slight depression and lack of interest in eating. As the disease progresses, these symptoms intensify. Dyspnoea, pyrexia and coughing occur. The nasal discharge changes in consistency from thin and clear to thick yellow and viscous. The laboured breathing and associated pain causes the calf to stand with its elbows positioned away from the chest wall and reluctant to move. To ensure a successful treatment outcome, it is imperative that the animals are identified and treated early in the disease process.

Peracute cases of haemorrhagic septicaemia result in sudden death. Acute cases show dyspnoea, fever, depression, profuse salivation and nasal discharge. Subacute cases are characterized by subcutaneous swelling of the submandibular region, which may extend down towards the brisket.

The bacteria first multiply in the tonsils, resulting in necrosis and oedema of the area. From there, they spread septicaemically via the circulation.

**Diagnosis**

The respiratory system is principally affected in cases of pneumonic pasteurellosis. The interlobular septae are distended, and grey and red hepatisation is seen. Other associated signs such as pleuritis and congestion may be found.

Pathological signs in haemorrhagic septicaemia include widespread petechiae and ecchymoses, as well as severe congestion and oedema. Pneumonic signs are also found in subacute and chronic cases.

Diagnosis is mainly reliant on the isolation of the causative organism, from the affected lung or a trans-tracheal aspirate, in the case of pneumonic pasteurellosis and from the affected organs in haemorrhagic septicaemia. Blood cultures from live animals seldom yield results, as the septicaemia is terminal. Nasal swabs are unsatisfactory as the strains of Pasteurella carried on the mucous membranes of the upper respiratory tract are not necessarily the same as those causing the disease.

Serotyping of isolates is generally not necessary in cases of pneumonic pasteurellosis, as knowing the serotype is of little value in controlling the disease.
Serotyping is crucial in cases of haemorrhagic septicaemia, to confirm the presence of either types B or E, and subsequent vaccine selection.

**Control**

Simple toxoid vaccines for *P. multocida* protect well, as those vaccines are based on the capsule of *P. multocida*. *Pasteurella multocida* usually has a prominent capsule.

The only vaccine available in South Africa which contains *P. multocida* is the Onderstepoort Biological Products *Pasteurella* vaccine for cattle. It contains types A, D and E, as well as *Pasteurella haemolytica*, now known as *Mannheimia haemolytica*, type A1. Type B is not included in the vaccine, and if it were to infect cattle in South Africa there would be no real cross-protection from the existing vaccine. The use and the degree of protection afforded by the OBP vaccine in wildlife is unknown.

Leukotoxin based vaccines, which include most of the presently available cattle *Pasteurella* vaccines, protect cattle well against nearly all *Mannheimia haemolytica* infections. Leukotoxin vaccines do not protect against *P. multocida* infections. Leukotoxin vaccines are vaccines such as Bovitect, Bovivax, Leukopast, One Shot Ultra 7 and Pastvac.

*Pasteurella multocida* is generally sensitive to most commonly used antibiotics, such as penicillin, amoxicillin, cephalosporins, florfenicol, macrolides, sulphonamides and fluoroquinolones.

One exception is tetracycline. There is significant resistance to tetracyclines amongst *Pasteurella multocida* isolates from feedlot cattle with pneumonia. This amounted to 59% resistance in isolates made during 2011. Tetracycline resistance has not been seen in isolates from other sources, such as calf pneumonia, sheep pneumonia and haemorrhagic septicaemia.

**References**


8. DeAngelis P L, Gunay N S, Toida T, Mao W, Linhardt R J, 2002 Identification of the capsular polysaccharides of Type D and F \textit{Pasteurella multocida} as unmodified heparin and chondroitin, respectively. Carbohydrate Research 337:1547-1552


