

# Role of endotoxin in the pathogenesis of haemorrhagic septicaemia in the buffalo<sup>\*1</sup>

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The pathogenesis of haemorrhagic septicaemia in buffalo infected with *Pasteurella multocida* is poorly understood. However, the characteristic of sudden onset leading to the rapid death of infected animals is similar to that seen in other clinical conditions known to involve endotoxic shock. The objectives of the work were to assess the contribution of endotoxaemia to the disease's pathogenesis and to characterize the pathophysiological reaction, including the acute phase response, of buffalo to experimental infection with *P. multocida* serotype B:2, the bacterium responsible for the disease in Asia. After intranasal infection of eight buffaloes with a culture of a field isolate of *P. multocida* serotype B:2, three animals succumbed to the disease at 26–30 h post-infection (p.i.) and five survived. Rectal temperatures of infected animals rose to a peak at about 10 h p.i. and surviving animals showed a second peak in rectal temperature at 36 h p.i. Endotoxin was present only in serum of non-surviving animals 3–5 h before death or killing during which time concentrations increased rapidly, correlating with the development of overt clinical signs and reductions in rectal temperature, concentrations of white blood cells, serum thyroxine, iron, copper and zinc, an increase in serum haptoglobin and cortisol and the detection of a low-grade bacteraemia. A strong acute phase response was maintained in surviving animals with a progressive rise in serum haptoglobin over 96 h p.i. a slow rise in the serum copper concentration from 24 h p.i. and an increase, from about 65 h p.i. in serum  $\alpha_1$ -acid glycoprotein. The findings demonstrate that a progressive endotoxaemia and associated sequelae correlates with the development of overt haemorrhagic septicaemia disease and sudden death in buffalo.

Microbial Pathogenesis (2001) 30 p171-178