International Dairy Topics

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Clostridium perfringens enterotoxaemia

Introduction

The enterotoxaemia caused by the bacterium Clostridium perfringens type C often has a fatal outcome in dairy calves. Enteric diseases caused by Clostridium perfringens A, B and D have been seen in calves but are very rare.

It is the neonatal animal that is typically affected by this disease, but losses in older animals, usually up to three months of age, can be important.

The cause

Clostridium perfringens is a Gram positive anaerobic bacterium that is part of the normal intestinal flora. Intakes of large amounts of soluble carbohydrate and/or protein favour a rapid multiplication in the numbers of this bacterium in the intestinal lumen and the development of a type C enterotoxaemia. The rapid explosion in bacterial numbers causes the production of lethal exotoxins that cause the majority of the tissue damage (lesions) seen in the enterotoxaemia.

The actual trigger for the production of large bacterial numbers is hard to specifically define but is linked to the 'pushing' of the calves' nutrition. Feeding of large volumes of milk replacer, especially as a few large meals, is a good way to induce Clostridium perfringens enterotoxaemia. Other risk factors include heavy grain feeding, foraging on grain crops, sudden access to high quality forage or overfeeding following a period of starvation/hunger.

The major lethal toxin is the β-toxin, although variable amounts of the α-toxin are also produced.

Pathology

The β-toxin kills the enterocytes of the small intestine and this then gives this toxin access to the deeper layers of the intestinal walls where it induces extensive submucosal necrosis and haemorrhaging into the intestinal lumen. The α-toxin is a phospholipase and it destroys the lecithin within host cell membranes and some intracellular components. Terminally, these toxins enter the bloodstream.

β-toxin is a protein that can be inactivated by exposure to trypsin. Its effects in neonates can therefore be made worse because of low pancreatic production of trypsin or the presence of trypsin inhibitors in colostrum.