

Clostridial Diseases of Cattle and Sheep

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Introduction

The *Clostridium* species affecting cattle and sheep are large, Gram-positive, rod-shaped, spore-bearing bacteria. Many clostridia are saprophytes, which normally grow in soil, water and decomposing plant and animal material. Other species, such as *C. perfringens*, are normal inhabitants of the intestines and, after the death of the animal, rapidly invade the blood and tissues playing a major role in decomposing the carcass. These post-mortem invaders must be distinguished at post-mortem examination from those organisms causing primary clostridial infections.

Diseases Caused by Pathogenic Clostridia

Quinn and others (2000) divided the pathogenic clostridia affecting cattle and sheep into the following groups: neurotrophic clostridia, histotoxic clostridia and clostridia which produce enterotoxins (Table 1).

Neurotrophic clostridia

C. tetani and *C. botulinum* produce powerful neurotoxins giving rise to the diseases tetanus and botulism, respectively.

Histotoxic clostridia

The exotoxins produced by these clostridia produce local tissue necrosis and systemic toxæmia. Examples

include *C. chauvoei*, the major cause of blackleg, and *C. novyi* type B, which causes black disease.

Clostridia which produce enterotoxins

C. perfringens species cause enterotoxaemia and enteropathy. Examples include *C. perfringens* type D, which causes pulpy kidney disease and *C. perfringens* type B, which causes lamb dysentery.

There has been an increase in recent years in submissions to DARD's Veterinary Sciences Division (VSD) of carcasses affected by clostridial diseases (Figure 1).

Neurotrophic Clostridia

C. tetani

Tetanus occurs sporadically in cattle and sheep of all ages. Tetanus endospores are present in the environment and may enter surgical wounds, such as after castration or docking, or traumatised tissues, such as those of the genital tract following dystocia. An idiopathic form of tetanus also occurs, in which the organism multiplies in the intestinal tract following a dietary imbalance or change. The incubation period may be from a few days to several weeks.

The clinical presentation of tetanus includes early stiffness and reluctance to move. Bloat, a raised tailhead and trismus develop and the third eyelid

prolapses, particularly when the head is moved. In the terminal stages of the disease the animal becomes

recumbent and develops tetanic spasms and opisthotonus.

Table 1. Ruminant Diseases Caused by Pathogenic Clostridia (after Quinn and others, 2000)

Clostridium Species	Disease
Neurotoxic clostridia <i>Clostridium tetani</i> <i>Clostridium botulinum</i>	Tetanus Botulism
Histotoxic clostridia <i>Clostridium chauvoei</i> <i>Clostridium septicum</i>	Blackleg Malignant oedema Braxy (sheep) Big head of rams
<i>Clostridium novyi</i> type A <i>Clostridium novyi</i> type B <i>Clostridium haemolyticum</i> (<i>C. novyi</i> type D) <i>Clostridium sordellii</i>	Black disease (necrotic hepatitis) Bacillary haemoglobinuria Gas gangrene Abomasitis
Enterotoxaemia <i>Clostridium perfringens</i> Type B (<i>C. welchii</i>) <i>Clostridium perfringens</i> Type C (<i>C. welchii</i>) <i>Clostridium perfringens</i> Type D (<i>C. welchii</i>)	Lamb dysentery Struck Pulpy kidney

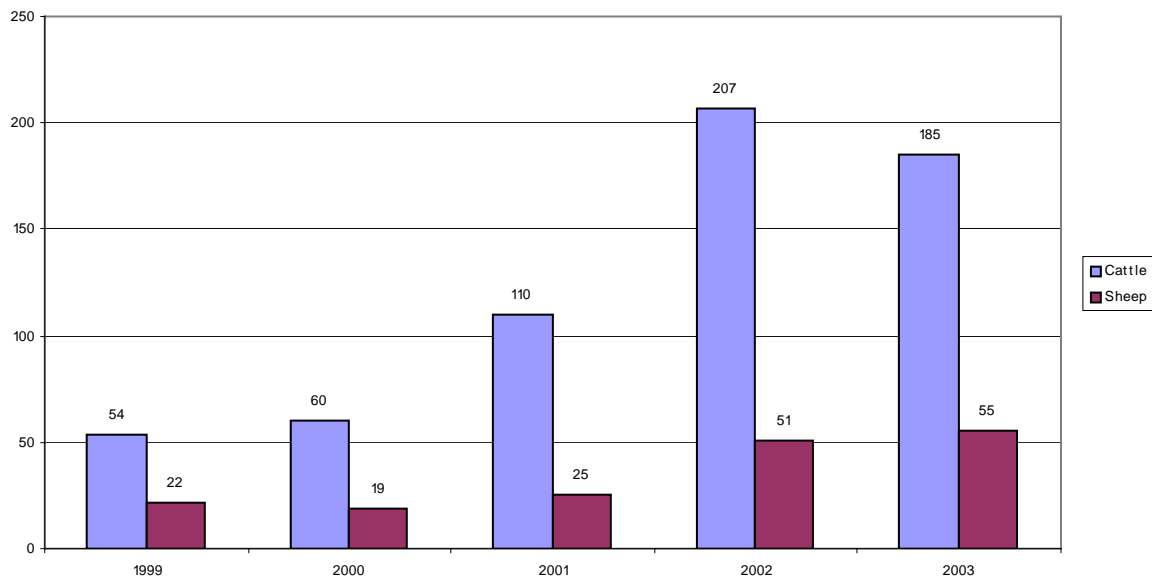


Figure 1. Clostridial diseases of cattle and sheep confirmed post mortem at VSD

C. botulinum

There has been a major increase in the number of suspected cases of botulism in cattle in Northern Ireland in recent years. Botulism is a neuro-paralytic disease caused by the toxins of *Clostridium botulinum*, which is usually fatal. These bacteria are commonly found in the environment and will grow in decaying carcasses and vegetable matter. There are seven different types of botulinum toxin (types A-G). Type C or D toxins cause most cases of botulism in cattle and as little as 10µg of these toxins are lethal to a cow (Gregory and others, 1996). Type D is the most common botulinum toxin type associated with bovine botulism in Northern Ireland.

Botulism can affect cattle of all ages. If large amounts of toxin have been ingested the animal may be found dead without showing signs of illness. However, signs of illness usually become apparent within 24 hours to 7 days of ingesting botulinum toxin. The main clinical feature is lack of muscle tone resulting in progressive muscle weakness.

In the early stages of the disease, affected cattle may stagger about, have hind leg stiffness and be reluctant to move. They occasionally become aggressive and may attempt to charge people in close proximity. Muscle weakness usually affects the hindquarters first before progressing to the forequarters, head and neck. Affected animals may be found lying on their chest with the head turned towards the flank (similar to cows with "milk fever"). A few cattle may develop paralysis of the tongue muscles resulting in inability to chew or swallow, drooling of saliva or, less commonly, protrusion of the tongue from the mouth. Breathing eventually

becomes difficult when the chest or diaphragm muscles become paralysed and most affected animals either die or are euthanased. The clinical signs most commonly recorded in recent cases of bovine botulism in Northern Ireland are listed in Table 2 (Karina Wrigley, personal communication).

Botulism does not produce pathognomonic lesions and therefore cannot be diagnosed by gross post-mortem or histopathological examinations. However, necropsy may help with differential diagnosis. The standard diagnostic test for botulism is the mouse bioassay that is used to detect toxin in faeces or gastro-intestinal contents of affected animals.

Outbreaks of botulism in Northern Ireland have been reported in cattle grazing pasture on which poultry litter had been spread (McIlroy and others, 1987) and in cattle fed ensiled poultry litter (McLoughlin and others, 1988). Recent outbreaks of bovine botulism in Northern Ireland have been reported where broiler poultry litter had been spread on pasture, used as bedding in cattle houses or stacked on the farm adjacent or close to pasture where cattle were grazing. The presence of the carcasses of birds that have died during production is regarded as the likely source of botulinum toxins. Scavengers may gain access to this material after it has been stacked outside or spread on pasture. It is speculated that even small fragments of carcasses transferred onto pasture or silage by scavenger animals, such as foxes, dogs or crows, can pose a risk to grazing cattle.

Table 2. Clinical signs of bovine botulism recorded on 30 Northern Ireland farms

Clinical signs	Number of farms (n=30)
Progressive flaccid paralysis	29
Lateral recumbency	29
Sternal recumbency	27
Difficulty rising	16
Ataxia	12
Posterior weakness	5
Tongue paralysis	6
Difficulty in swallowing/excessive salivation	6
Lethargy	5
Aggression	3
Staring eyes	2

Careful disposal of animal or bird carcasses and poultry litter is necessary to minimise the risk of botulism to cattle. Poultry carcasses should be promptly removed from the chicken house and disposed of by incineration, or rendering (as required by EU Regulation No. 1774/2002). Following removal of the broiler crop, all poultry house doors should be kept closed until the litter is removed. The litter should not be removed from the house until it can be loaded directly onto covered vehicles or immediately covered. At no time should it be accessible to dogs, foxes, crows or other scavengers that may carry carcasses onto adjacent pasture or into cattle housing. Washings from poultry houses and yards should be collected in tanks rather than be allowed to flow onto adjacent land.

The results of investigations by VSD staff suggest that poultry litter should not be spread on agricultural land that is to be grazed, or from which silage

or hay is to be harvested, in the same year. This is because fragments of carcasses may persist on pasture for a considerable time. If poultry litter must be spread, it should be deep-ploughed into arable ground. If this is not an option and litter must be disposed off by spreading on pasture, cattle should not have access to the treated fields for at least several months. However, there is no guarantee that the treated fields would then be safe for cattle and it is important to remember that scavenger animals and birds to neighbouring fields may transport fragments of carcasses on pasture.

Vaccination may protect cattle grazing potentially contaminated pasture. Spreading poultry litter on a windy day may also pose a risk of contaminating adjacent fields. Any animal or bird carcasses, or portions of carcasses, visible on pasture or in cattle houses, should be promptly removed. Even small fragments of carcasses may be dangerous to cattle and should be disposed of by incineration or rendering. In any case, it is an offence to leave carcasses or part carcasses on any land, and any person discovered so doing may be prosecuted.

No vaccine is available under general licence in the UK for the protection of cattle against botulism. However, a vaccine against types C and D botulism is available under “special treatment authorisation” (STA) from the Veterinary Medicines Directorate (VMD). A recent survey by VSD staff of veterinary surgeons that have used this vaccine in Northern Ireland indicates that it is effective in controlling botulism in cattle. Veterinary surgeons who wish to consider vaccination of clients' herds may apply to the VMD for an STA to obtain and use this vaccine. It is

important to remember that two doses of vaccine are required at an interval of 4-6 weeks. Cattle receiving only one dose are not fully protected.

The risk to humans from cases of botulism in cattle appears remote. However, invoking the precautionary principle, the Food Standards Agency requests a voluntary ban on the sale for human consumption of all milk and meat from the affected group of animals until 14 days after the last case.

Histotoxic clostridia

C. chauvoei

Blackleg (gangrenous myositis), caused by *C. chauvoei*, is the most common clostridial disease seen in cattle carcasses submitted to VSD for post-mortem examination. Gangrenous myositis occurs less commonly in sheep. However, *C. chauvoei* commonly causes a post-parturient metritis of sheep.

The majority of blackleg cases occur in young cattle (3 months to 2 years of age) at grass during the summer months (Figure 2). Most cases are found dead. Cattle examined before death are usually lame and depressed. Crepitation is present over affected muscle masses, which are initially warm and painful; later becoming cold and insensitive. Death usually occurs within 12-24 hours,

following signs of systemic toxæmia: dyspnoea, recumbency and coma.

At post-mortem examination lesions are principally found in the large muscle masses of the fore- and hindquarters. However, lesions are commonly found in other muscles such as the masseter, intercostal, psoas, tongue, diaphragm and heart. There is a characteristic rancid odour from recently dissected lesions. Visceral lesions due to *C. chauvoei* infection, such as pericarditis and pleurisy, are frequently seen. Malone and others (1986) reported visceral lesions only in 7 out of 29 cases of *C. chauvoei* infection of cattle diagnosed over a two-year period (Table 3). *C. chauvoei* may be identified in the lesions by the fluorescent antibody technique.

C. septicum

Although other histotoxic clostridia may be involved, *C. septicum* is primarily associated with malignant oedema in cattle and sheep. Signs of malignant oedema include subcutaneous accumulation of fluid, dullness and inappetence. The lesion may be associated with a wound or may occur following intramuscular injections (Harwood, 1984).

C. septicum also causes an abomasitis of sheep, known as braxy. It is not commonly diagnosed at VSD.

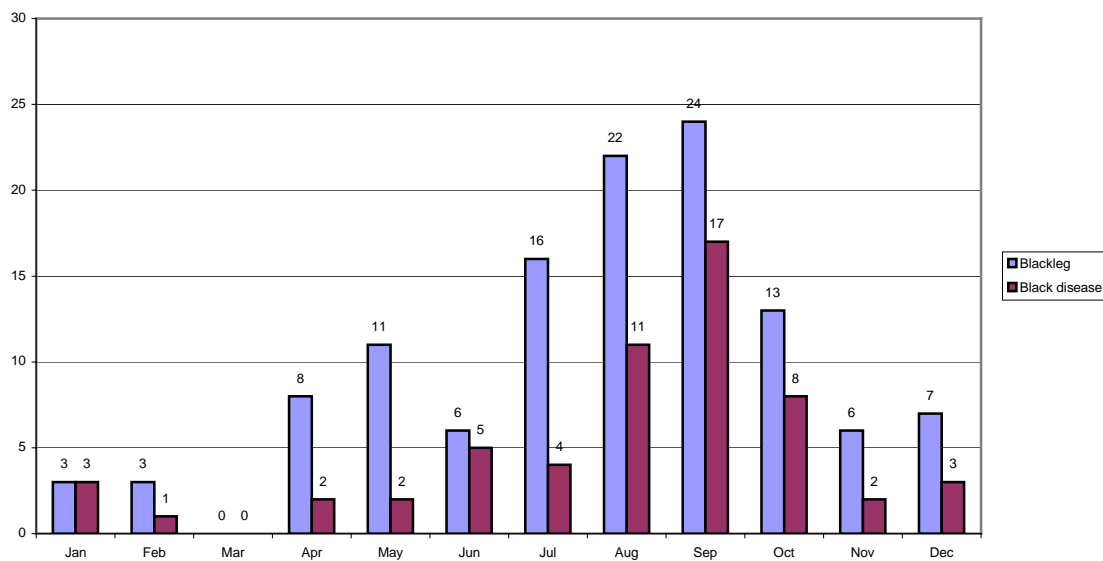


Figure 2. Clostridial diseases of cattle confirmed *post mortem* at VSD in 2003

Sites of <i>C. chauvoei</i> lesions	Number of cases	Percentage
Muscle only	14	48.3
Muscle and pericardium	8	27.6
Pericardium only	6	20.7
Meninges only	1	3.4
Total	29	100

Table 3. Sites of lesions in 29 cases of *C. chauvoei* infection in cattle

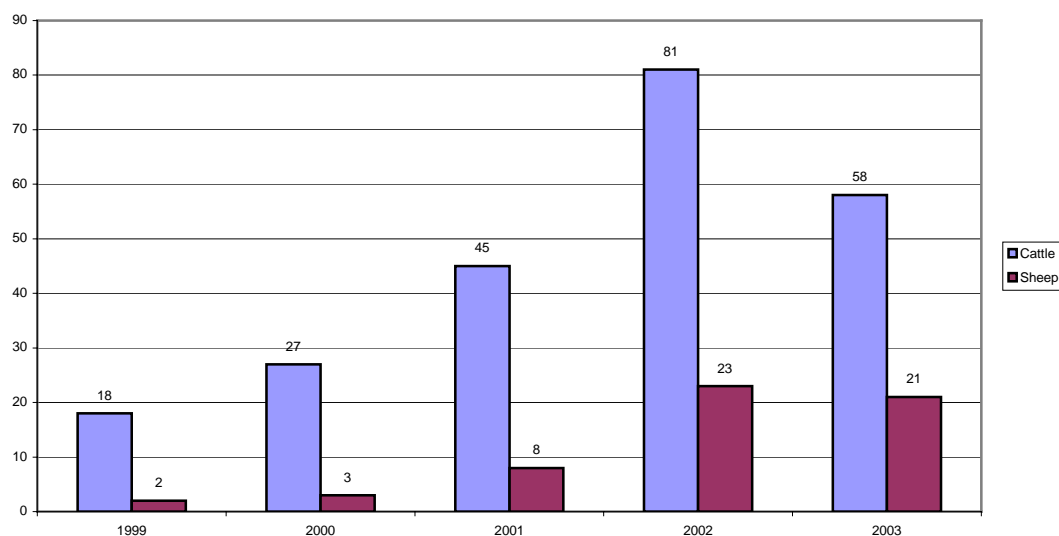


Figure 3. Black disease cases diagnosed at VSD 1999-2003

C. novyi type D

C. novyi type D causes bacillary haemoglobinuria of cattle. This disease is present in the southwest of Ireland, but is rare in Northern Ireland. Affected cattle may be found dead. Clinical disease is characterised by the presence of dark red urine and fresh blood in the rectal contents. If standing the animal will have an arched back and be reluctant to move, grunting when forced to do so. Jaundice is not a constant finding. Breathing is distressed and the pulse is weak. The animal becomes recumbent and death follows within 24 hours. On post-mortem examination all cases have a mahogany-coloured liver in which there is a least one focal area of necrosis. The urine is dark red and there is blood in the faeces. Jaundice is present in all cases, but the intensity varies from slight to pronounced. Widespread ecchymotic haemorrhages are present on the omentum and subcutaneous tissues, pleura and in the submucosa of the stomach and intestines. *C. novyi* may be identified in the liver lesion by the fluorescent antibody technique. However, in order to differentiate *C. novyi* type D from *C. novyi* type B, toxin-antitoxin tests are required (Power and others, 1987).

C. sordellii

C. sordellii was traditionally associated with gas gangrene, a wound infection of cattle and sheep. Recently, it has also been associated with acute abomasitis in young lambs, 3-10 weeks of age, and sudden death and abomasitis in finishing lambs of 6-12 months of age (Lewis and Naylor, 1998). It has also more recently been identified in cases of fatal metritis in ewes (Clark, 2003) and several laboratories have identified this organism in cases of bovine abomasitis.

Post-mortem lesions of *C. sordellii* abomasitis vary depending on the age of the sheep. In lambs 3-10 weeks of age the abomasum is partially distended and displaced. The abomasal wall is thickened due to a combination of emphysema and oedema. Erosions and congestion are present in the abomasal mucosa. The main feature of *C. sordellii* abomasitis in lambs 4-6 months of age is abomasal congestion with some ulceration. The carcasses appear toxæmic. Post-mortem findings in older ewes are less specific. There is a variable degree of abomasitis, but in some cases there is an intense peritonitis, blood-tinged fluid in the abdominal cavity and a perforated abomasal ulcer. *C. sordellii* may be identified in the abomasum by the fluorescent antibody technique.

Enterotoxaemia

C. perfringens Type B

Lamb dysentery, caused by *C. perfringens* Type B, is infrequently diagnosed at VSD. The disease is seen in lambs, usually less than 2 weeks of age, and is characterised by a haemorrhagic enteritis. Lambs are usually found dead. Affected lambs show signs of abdominal pain and have fluid, bloodstained faeces. Definitive diagnosis is by demonstration of the clostridial beta toxin by ELISA in intestinal contents.

C. perfringens Type C

Struck, caused by *C. perfringens* Type C, causes sudden death in adult sheep at pasture. It has not been reported in Northern Ireland.

Clostridium perfringens Type D

Pulpy kidney disease, caused by *C. perfringens* Type D, is the most common clostridial enterotoxaemia of sheep diagnosed in Northern Ireland.

The disease has also been reported in cattle.

Although reported in all ages of sheep, the disease is most common in growing lambs 1-3 months of age and in finishing lambs over 6 months of age. The majority of cases are found dead. On post-mortem examination the intestines are usually congested and the kidneys soft and friable in appearance. A hydropericardium, containing a fibrin clot, and glycosuria are present. A minority of cases develop central nervous system signs: ataxia (progressing to recumbancy), opisthotonus, convulsions and death. In these cases focal symmetrical encephalomalacia is seen on histopathological examination of the brain.

Clostridial enterotoxaemia is more difficult to diagnose in cattle than in sheep. Clinically, sudden death is common, but diarrhoea/dysentery and profound depression occur in the early stages of the disease. At post-mortem examination the carcass is usually in good condition, but rapidly decomposes. Bloat is usually present and the intestinal contents may be haemorrhagic. Further evidence of clostridial enterotoxaemia may be obtained by demonstration of the epsilon toxin by ELISA in the intestinal contents. Focal symmetrical encephalomalacia also occurs in cattle.

Conclusion

There appears to be an increase in incidence of clostridial disease within recent years in Northern Ireland. Many of these diseases have a distinct geographical distribution. The economic importance of correct vaccination against common diseases such as blackleg, black disease and clostridial enterotoxaemia is self-evident. Vaccines are now also available for the emerging clostridial

diseases of botulism and *C. sordellii* infection.

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