Pathogenic anaerobic bacteria and the environment

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Summary: The distribution of pathogenic anaerobes in the environment and the relationship with diseases in animals are discussed. A distinction between the spore-bearing anaerobes (Clostridia) and the Gram-negative non-spore-forming anaerobes is necessary. The main habitat of clostridia is the soil but they are also found in dust, sewage, rivers, lakes, sea water, milk, vegetables, fresh meat, fish, insects and the intestinal tract. The Gram-negative non-spore-forming anaerobic bacteria are also widely distributed among animals, principally on mucous membranes of the alimentary tract.

After a general introduction and a section on the isolation of anaerobes, the various diseases caused by clostridia (botulism, tetanus, blackleg, malignant oedema, infectious necrotic hepatitis, enterotoxaemia and gas gangrene) and Gram-negative anaerobes (infections due to Fusobacterium and Bacteroides spp., such as diphtheria, footrot, etc.) are discussed. In particular, information concerning the reservoir of the causative agent and the mode of transmission is presented.

KEYWORDS: Clostridium - Disease - Environment - Gram-negative anaerobes - Pathogenic anaerobes - Reservoirs - Transmission.

INTRODUCTION

Anaerobic bacteria are widely distributed in the environment. In discussing the relationship between pathogenic anaerobes and the environment, a distinction should be made between the spore-bearing anaerobes (genus Clostridium) and the non-spore-forming anaerobes (in particular those involving Gram-negative bacteria).

The main habitat of clostridia is undoubtedly the soil. Organisms of the resistant spore form live for long periods in the soil and may be acquired by susceptible animals and Man from the soil or vegetation, through wound infection, or by mouth (12). Some of them appear to be common inhabitants of the intestinal tract of animals. For example, C. perfringens is uniformly present in faeces.

It has been held by some that the intestinal tract is the main habitat of certain of the anaerobes and that their presence in the soil is due to faecal contamination. It is more likely that the primary habitat of most spore-bearing anaerobes is the soil; that they are ingested frequently with fodder; and that some of them have adapted themselves temporarily or permanently to a life in the intestinal tract (32).

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However, the species of clostridia associated with animal infections have been isolated from a wide spectrum of environmental sources, namely: dust, sewage, rivers, lakes, sea water, milk, vegetables, fresh meat, insects and fish, in addition to soils and the intestinal tract. Therefore, clostridia of veterinary importance can be expected to be recovered from any clinical specimen contaminated by soil, dust or faeces. As members of the normal or transient flora of the respiratory and genito-urinary tracts, they may be expected to be recovered from any body fluid or tissue contaminated, colonised, or infected by these endogenous sources.

Sources other than clinical specimens from which clostridia of greatest significance to the veterinary field have reportedly been isolated are indicated in Table I.

### Table I

**Non-clinical sources of isolation of clostridia**

(4, 6, 12, 20)

<table>
<thead>
<tr>
<th>Species</th>
<th>Faeces</th>
<th>Isolated from</th>
<th>Soil/water</th>
<th>Marine sediment</th>
<th>Food</th>
</tr>
</thead>
<tbody>
<tr>
<td>C. bifermentans</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C. botulinum</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>C. butyricum</td>
<td>+</td>
<td>+</td>
<td></td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>C. carnis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>C. chauvoei</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>C. colinum</td>
<td>+ (bird)</td>
<td></td>
<td></td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>C. difficile</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C. fallax</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C. histolyticum</td>
<td></td>
<td>+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C. novyi</td>
<td>+</td>
<td>+</td>
<td></td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>C. perfringens</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>C. septicum</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>C. sordellii</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>C. spiroforme</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>C. sporogenes</td>
<td>+</td>
<td></td>
<td>+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C. tetani</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td>+</td>
</tr>
</tbody>
</table>

In the sporing stage, all members of the genus Clostridium have a pronounced but variable resistance to heat, drying and disinfectants. Therefore, they are able to survive in the environment under unfavourable conditions. In the vegetative state, clostridia are about as resistant to physical and chemical agents and unfavourable environmental conditions as non-sporing aerobes.

Although clostridia usually lead a saprophytic existence, several species are causally related to well-recognised diseases in animals and Man. Some clostridia produce powerful exotoxins which are responsible for the specific pathogenicity of the organisms. Others may produce less potent toxins but, in addition, have the power of invading and multiplying in the tissues, causing wound infections such as gas gangrene (12).
Infections involving Gram-negative, non-spore-forming, anaerobic bacteria are also widespread among animals and Man. Their sporadic occurrence, variability in localisation and bacterial population and pronounced tendency to include admixtures of other microbial agents perhaps account for the absence of a collective name. The term necrobacillosis has been used for infections with *Fusobacterium necrophorum*, formerly *Sphaerophorus necrophorus*. There are two genera under discussion: *Fusobacterium* and *Bacteroides*, both of which consist of uniformly Gram-negative members that require strict anaerobic conditions for propagation. They are widely, and perhaps universally, distributed among animals, principally on mucous membranes of the alimentary tract. Therefore, they must be assumed to exist wherever their hosts are found, but no firm figures on carrier rates are available. Most of the agents are acquired promptly after birth. An extrinsic source directly related to a particular episode of disease caused by these organisms usually cannot be identified, except in a handful of traumatic exposures. Another major exception is footrot in sheep, in which *B. nodosus* is acquired through direct or indirect contact with infected animals. There is evidence that *F. necrophorum*, an important pathogen in herbivores and swine, is capable of surviving outside an animal host for more than eight weeks and possibly for several months, even in soil (2).

**ISOLATION**

A large variety of media and procedures have been described for cultivation of clostridia and Gram-negative anaerobes. When culturing clinical specimens, primary emphasis should be placed upon the proper selection and collection of specimens to avoid the normal and transiently colonising flora. Although clostridia are frequently found in mixtures with other anaerobic and facultative organisms, special enrichment or selective media are not usually necessary for their recovery. For polymicrobial infections in which clostridia may play a significant role, if the specimen truly represents the invading edge of the infected tissue, the clinically significant clostridia are usually sufficiently abundant to be easily detected and isolated on non-selective, anaerobic blood agars. Isolation of clostridia from faeces or other normal flora and soils can be problematic. *C. tetani* may produce disease in very small numbers in wounds and may be extremely difficult to recover or separate from mixed populations, even with the most effective procedures. In this case and in the isolation of *C. botulinum*, enrichment and selective procedures can be helpful. Knowledge of the normal flora of various regions may also allow one to judge more readily whether a given isolate is significant. Table II indicates the incidence of certain anaerobes as normal flora at various sites and provides taxonomic information of the various anaerobes (29).

**CLOSTRIDIAL DISEASES AND THE ENVIRONMENT**

Domestic animals, however well kept, exist under what would be regarded as conditions of extreme squalor for Man. They are intimately exposed to micro-organisms in the soil and in their own dejecta to a degree that humans only experience
<table>
<thead>
<tr>
<th></th>
<th>Clostridium</th>
<th>Gram-positive</th>
<th>Non-spore-forming bacilli</th>
<th>Gram-negative</th>
<th>Coeci</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Actinomyces</td>
<td>Bifidobacterium</td>
<td>Bacteroides</td>
<td></td>
</tr>
<tr>
<td>Upper respiratory tract</td>
<td>0</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Mouth</td>
<td>±</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Intestine</td>
<td>2</td>
<td>±</td>
<td>2</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Genito-urinary tract</td>
<td>±</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

0 : not found or rare
± : irregular
1 : usually present
2 : usually present in large numbers
where hygiene is exceptionally poor. Because pathogenic clostridia are common in the soil and in the dejecta of animals, domestic animals suffer more than Man from clostridial infections. Diseases such as gas gangrene, clostridial enterotoxaemias, tetanus, botulism, etc., are widespread among domestic animals and result in serious losses to the stock owner (28).

**BOTULISM**

Botulism is a serious disease of animals and Man, caused by ingestion of food in which *C. botulinum* has grown and produced toxin (pre-formed toxin). It is characterised by weakness, paresis and paralysis because the botulinum toxin affects the nervous system. The seven official toxin types are A, B, C, D, E, F and G. Man and animals have highly divergent sensitivities to these toxin types. In Man, outbreaks are caused by type A, B, or E and a few by type F. Toxin types C and D are responsible for nearly all cases of botulism in animals, but rare cases of type B have also been reported (12, 18, 19).

**Reservoir of the agent**

*C. botulinum* is widely distributed in soil, water, marine sediments and food. However, the distribution of toxin types may differ from one area to another. Type A is widespread in North America but is rarely found in Europe. Types B and C are reported on all continents although knowledge of the situation in Asia, Africa and South America is scarce. Type D is historically associated with botulism in cattle and sheep in Southern Africa and Australia but, since the 1970s, several type D outbreaks have occurred in Western Europe. Type E occurs mainly in marine environments of the Northern Hemisphere whilst limited distribution of type F is reported (Denmark and the United States of America). Type G is the most recently recognised type of *C. botulinum*. The first isolate was obtained in 1969 from a soil sample in Argentina. Later, type G was isolated in Switzerland during a study of human autopsy specimens but has not been clearly implicated as the cause of botulism in Man or animals (7, 27). No thorough world-wide survey has ever been made on the incidence of *C. botulinum* and current knowledge of its occurrence derives from the results of a number of investigations of smaller areas (13, 21, 26). Results from some countries are shown in Table III. In general, the incidence and distribution of the various toxin types is greatly influenced by the isolation procedure and by the types of material and ecosystems investigated.

Little effort has been made to establish a relationship between the properties of the soil and the presence of *C. botulinum*. Meyer and Dubovsky considered that *C. botulinum* type A inhabited primarily virgin soil, being replaced by type B on cultivation (22). The occurrence of type A strains seems to be associated with soils which are neutral to alkaline in reaction and of low organic content. Although the factors governing the incidence of other types are not clear, it does seem that the occurrence of types E and F is often associated with soil which is moist, or even wet, much of the time.

The occurrence of *C. botulinum* in soil may be inversely related to the occurrence of organisms which inhibit its growth. Some soil or sediment specimens may be highly inhibitory to type E strains. Some strains of *C. perfringens* and *C. sporogenes* from
TABLE III

Geographical distribution of *C. botulinum* (13, 21, 26)

<table>
<thead>
<tr>
<th>Country</th>
<th>Number of samples</th>
<th>Percentage of samples with type indicated</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>A</td>
</tr>
<tr>
<td>Sweden (1963)</td>
<td>281</td>
<td>0</td>
</tr>
<tr>
<td>Russia (1966)</td>
<td>2,851</td>
<td>0.7</td>
</tr>
<tr>
<td>United States (1967)</td>
<td>1,414</td>
<td>0.2</td>
</tr>
<tr>
<td>Indonesia (1973)</td>
<td>196</td>
<td>1.5</td>
</tr>
<tr>
<td>Great Britain (1977)</td>
<td>664</td>
<td>0</td>
</tr>
<tr>
<td>Netherlands (1970-1988)</td>
<td>3,267</td>
<td>0.09</td>
</tr>
<tr>
<td>Denmark (1980)</td>
<td>342</td>
<td>0</td>
</tr>
<tr>
<td>Faroe Islands (1930)</td>
<td>98</td>
<td>0</td>
</tr>
<tr>
<td>Iceland, Greenland (1980)</td>
<td>121</td>
<td>0.8</td>
</tr>
<tr>
<td>Bangladesh (1980)</td>
<td>12</td>
<td>0</td>
</tr>
</tbody>
</table>

* No attempt was made to distinguish between types C and D.

Soil are inhibitory for *C. botulinum* types A, B and F but not for types C and D strains. Such inhibiting strains were not found in soil samples in which strains of susceptible types of *C. botulinum* had been demonstrated (26).

Soil and water infected with *C. botulinum* are the sources of contamination for foodstuffs. The organism may also be found in the intestinal tract and perhaps other organs of healthy animals. It does no harm there, but, if the animals die, it may multiply and produce toxins in their carcasses. Soil and water may remain infectious for many years (at least twenty) because the spores are highly resistant. The existence of *C. botulinum* types C and D in an area would depend upon the incidence of botulism in animals there and upon carcass material left behind (12, 19).

Mode of transmission

Botulism is transmitted by the ingestion of food or food-related material in which botulinum toxin has been produced. Serious outbreaks have occurred in horses, ruminants, mink and many bird species, in particular poultry, waterfowl and pheasants. Horses and ruminants are highly susceptible to toxin types C and D; rare cases of type B have also been diagnosed (18, 19). Bird and mink are not susceptible to toxin type B or D; type C toxin is usually responsible for outbreaks of botulism in these animals.

Cattle contract botulism type C or D in two ways, either by eating carcasses or chewing bones contaminated with botulinum toxin (so-called "lamsiekte"), or by eating hay or silage which has been accidentally contaminated with toxin by the carcass of a small animal (cats, fowl or rats). The lamsiekte type of botulism is often a problem...
in those areas of the world where the soil is deficient in phosphate, where long periods of drought, either in the winter or in the summer, render the pasture poor, where some small animal is comparatively frequent and where \textit{C. botulinum} type C or D is present in the soil or is carried by the animals themselves. Some cases result from cattle drinking water which has become toxic from the decomposition of the carcass of a small animal. Spores of \textit{C. botulinum} are usually consumed with the toxic material and, after the animal dies of botulism or any other disease, the carcass will be invaded by this organism, toxin will be produced in the decomposing musculature and the carrion will pose a special hazard to other ruminants. Many spores will also be formed and will heavily pollute the soil under and around the carcass (19, 26).

In Western Europe (the Netherlands, Belgium, France and Great Britain) a different mode of transmission has developed since 1975, in which toxic poultry litter is the source of toxin types C and D. It is assumed that the extensive contamination of the environment with \textit{C. botulinum} type C, caused by botulism in waterfowl, has also led to the infection of poultry houses. In large poultry flocks, it is unavoidable that dead animals are overlooked during inspections, especially when they are hidden in the litter. Toxin type C concentrations can be very high in chicken carcasses. Toxin production is favoured by the high temperatures in chicken houses, commencing with approximately 33°C in the first week and being slowly reduced after a few weeks to 22°C. Thousands of chickens can be intoxicated in a few days. In turn, the outbreaks of botulism type C in poultry have led to cases of botulism in cattle. These cases can be explained by the following routes of transmission (14):

- The litter has been used as bedding in cubicles for cattle. The intake of toxic carcass material is favoured by the fact that the litter harbours spoiled chicken meal.

- The litter has been used as a fertilizer on grassland. Toxic parts of the chicken carcasses can be ingested accidentally by cattle during grazing or after harvesting of the grass for hay or silage.

- The litter is stored outside the chicken house. If this is not covered up carefully, birds (crows, magpies), dogs and other carnivorous animals may transfer toxic parts of carcasses to neighbouring pastures and, in this way, cattle can be intoxicated.

Outbreaks of botulism type D in cattle are caused by the same mode of transmission. However, there is one important clinical difference. Like other birds, chickens are not susceptible to type D toxin and this implies that, contrary to contamination with type C, the poultry farmer has no complaints. In this situation, it is often difficult to convince the owner of the poultry that his farm is responsible for the cases of botulism type D in cattle (14).

Rare cases of botulism type B have also been reported in cattle and horses. The outbreaks are caused by feeding a toxic ration and are associated with the contamination of those environments with \textit{C. botulinum} type B. In some cases, animals were fed grass or alfalfa silage of bad quality. In 1977, extensive investigations in the Netherlands showed that a striking outbreak of botulism type B in cattle was caused by supplementary feeding of toxic wet brewers' grains (5, 16, 17, 18).

Botulism type C in birds, in particular waterfowl, is a world-wide problem. There is no doubt that the disease has killed many millions of aquatic birds. Various species of birds are suspected of disseminating several types of \textit{C. botulinum} spores by way of their excreta and birds might carry \textit{C. botulinum} type C from an epizootic area
to a previously uncontaminated area. The incidence of *C. botulinum* type C is highly influenced by the history of avian botulism and the type of ecosystem. Because botulism in waterfowl is caused by the ingestion of pre-formed toxin, it follows that the toxin must be present in the environment in sufficiently high concentrations. These are found in putrid carcasses of waterfowl and other birds and in maggots of zoophagous flies. Predisposing environmental factors are periods of hot, dry weather, low water levels and an abundance of rotting vegetation (11, 23). Temperature increase in water habitats as a result of thermal pollution has precipitated outbreaks among waterfowl (11, 15).

**TETANUS**

Tetanus is an infectious disease of animals and man induced by the toxin of *C. tetani* which grows in injured tissues and results in toxaemia. It is characterised by painful, spasmodic contractions of voluntary muscles caused by the tetanus toxin being absorbed through the motor endplates in the muscles and apparently passing along the motor nerves to the spinal cord. The bacterial infection itself remains localised. The most susceptible animal, calculated on the amount of toxin/g of body weight is the horse. Ruminants and pigs are more resistant, while birds and cold-blooded animals are highly resistant.

**Reservoir of the agent**

*C. tetani* is widely distributed. Under suitable conditions spores can survive for very many years in soil and dirt, whereas spores present on the surface, as on roads, are subject to relatively rapid destruction or modification. Reinfection of soil, however, continuously takes place through animal excreta. It is generally admitted that *C. tetani* is found far more commonly in horse manure than in the faeces of other animals, but reliable figures are scarce. The spores are highly resistant when protected from light and heat (19, 32).

**Mode of transmission**

Natural infection commonly occurs in animals suffering from wounds contaminated with dirt containing *C. tetani*. As this organism is present in large numbers on the surface of the soil, particularly in garden earth, street dust, hay and manure, contamination of wounds no doubt occurs with comparative frequency; fortunately, however, infection is relatively rare. The presence of a suitable accessory factor enables the spores to germinate and multiply in the tissues; this probably depends on the production of tissue debris in the necrosed parts, with sufficiently low oxygen tension to allow propagation of *C. tetani*. Tetanus is not uncommon in animals subjected to the cruder forms of farm surgery (castration), docking, shearing and injuries from nails, punctures from sharp instruments, parturition, vaccination, etc.

*C. tetani* may grow and produce toxin in the rumen of cattle, resulting in an occasional case of endogenous tetanus (28).

**BLACKLEG**

*C. chauvoei* is most often the cause of blackleg of cattle and sheep. It is a myonecrosis, the essential feature being an involvement of muscles, characterised by the development of crepitating muscular swellings. Generally, young animals are affected.
Reservoir of the agent

*C. chauvoei* has a world-wide distribution. The diseased carcass is responsible for infecting the soil with *C. chauvoei* and for spreading the disease, chiefly by means of skins and meat. By allowing the decay of parts of the carcass of an animal which has died from blackleg, the surrounding areas become contaminated; the result is that stables, sheds, pastures and drinking pools are transformed into reservoirs of infection. Once an environment is contaminated, the infection is maintained by resistant spores which persist for a number of years. This fact explains the regional distribution of blackleg. The spores of *C. chauvoei* are highly resistant and they can retain their vitality and virulence for several years outside the body, but there is no evidence that the organism can multiply in the soil (19). Birds of prey fed with animals which have died from blackleg may serve as vectors in the spread of *C. chauvoei* spores over more than 2,500 kilometres (3).

Mode of transmission

The exact manner in which transmission usually occurs under natural conditions is not yet clear. It is believed that the organisms enter through the digestive tract and, after gaining access to the blood stream, are deposited in various tissues throughout the body. In cattle, the great majority of lesions apparently develop spontaneously, unassociated with wounds or other injuries. In sheep, serious outbreaks usually follow farm operations involving the handling of the animals for shearing, dipping, docking and castration.

MALIGNANT OEDEMA

This is an acute, highly fatal disease of horses, cattle, sheep, goats and swine caused by *C. septicum*, generally associated with a wound infection. The disease is characterised by oedematous and emphysematous swellings of the affected parts. “Braxy” is a type of gas gangrene of the abomasum of sheep due to the action of *C. septicum*, principally occurring in North-Western Europe.

Reservoir of the agent

*C. septicum* is widely distributed in cultivated soil and is a common inhabitant of the alimentary canal of animals and Man in an infected environment. It is not known whether the agent multiplies in the soil or whether it merely exists there as spores formed by the vegetative organisms in the intestinal tract of animals, in particular herbivorous animals (19).

Mode of transmission

Infection ordinarily occurs through contamination of non-aerated wounds, but many cases cannot be accounted for in this manner. Wounds caused by nails, splinters, fractures, castration, shearing, docking, parturition and unsanitary vaccination may become contaminated with soil containing the spores and are liable to infection.

“Braxy” probably results from the infection of the digestive tract. The ingestion of frozen grass or roots is thought to be a contributing factor.
BLACK DISEASE
(INFECTION NECROTIC HEPATITIS)

An infectious disease of sheep and occasionally of bovines, black disease is caused by a localised infection of the liver by \textit{C. novyi}. The organism usually occurs in necrotic areas of the liver and apparently is always associated with the invasion of the liver by immature wandering flukes (\textit{Fasciola hepatica}). Black disease is caused by \textit{C. novyi} type B, while \textit{C. novyi} type A may cause gas gangrene in Man and animals ("big head" in rams), type C is associated with a form of osteomyelitis in cattle and type D with bacillary haemoglobinurea in cattle (12, 19).

Reservoir of the agent

\textit{C. novyi} is found in the soil of environments where the disease occurs, namely: Australia, New Zealand, Germany, England, United States of America and Iran (1, 19). Many apparently healthy sheep in infected areas are carriers of \textit{C. novyi} spores present in the liver.

Mode of transmission

The spores are probably ingested and absorbed from the intestinal tract. When necrotic liver tissue is present due to the action of immature flukes, the spores vegetate and produce the powerful toxin which causes death.

ENTEROTOXAEMIA

This sub-section includes a number of acute toxaemic diseases caused by \textit{C. perfringens} involving the intestinal tract. The various enterotoxaemias differ in their clinical manifestations and pathology. The classical lethal toxins, alpha, beta, epsilon and iota, are mainly responsible for the pathogenesis of the diseases caused by \textit{C. perfringens} and it is on the identification of these toxins that the subdivision of the species into five main types, A, B, C, D and E, is based (28). \textit{C. perfringens} type A has been reported as causing a fatal enterotoxaemia of calves and sheep (yellow lamb disease), type B is the cause of a haemorrhagic and rapidly fatal enterotoxaemia of lambs (lamb dysentery), type C causes a disease of sheep known as "struck" and a necrotic enteritis of young piglets, and type D causes an often fatal enterotoxaemia in ovines (pulpy kidney disease or "overeating disease"). Type E is of no pathogenic importance. In ovines, type D is responsible for by far the largest proportion of losses due to infection or intoxication with \textit{C. perfringens}.

Reservoir of the agent

\textit{C. perfringens} is found in the soil of environments where the disease occurs. Type A is ubiquitous and has been isolated from soil, faeces, dust and food. The spores of some varieties are particularly heat resistant. Types B and type C have a restricted distribution and type D occurs in most parts of the world in soil and manure on infected farms. Certain parts of a farm appear to be more dangerous than others. Healthy sheep may serve as carriers, spreading the disease to areas which were regarded as clean (19).
Mode of transmission

The spores of *C. perfringens* are ingested and the organism is present in the intestinal tract. It is believed that the lamb in particular usually contracts the infection from the teat of a ewe, the teat being contaminated either by the faeces of the ewe harbouring *C. perfringens* or by infected soil. A number of predisposing factors are responsible for the sudden and rapid multiplication of *C. perfringens* in the bowel and for the massive production of toxin in a very short time.

GAS GANGRENE

Gas gangrene is a clostridial myonecrosis, the essential feature of which is muscle involvement. Gas gangrene often follows infection of contusions or deep wounds which have been contaminated with soil or faeces. The disease may be caused by a single species of *Clostridium*, but generally clostridia are present either in combination with aerobic organisms, anaerobic streptococci or other *Clostridium* spp. (32). Besides *C. chauvoei, C. septicum, C. novyi, C. perfringens* and *C. sordellii*, some clostridia of lesser importance can also be isolated: *C. histolyticum, C. fallax, C. bifermentans* and *C. sporogenes*.

Reservoir of the agent

The spores of these clostridial spp. are distributed world-wide and exist in the soil and in the intestinal tract of Man and animals.

Mode of transmission

The infection usually occurs through non-aerated wounds caused by nails, splinters, compound fractures, castration, docking, shearing, unsanitary vaccination or parturition.

DISEASES CAUSED BY NON-SPORE-BEARING ANAEROBES AND THE ENVIRONMENT

Infections involving Gram-negative, non-spore-forming, anaerobic bacteria are widespread among animals. There are two important genera, *Fusobacterium* and *Bacteroides*. They are principally distributed on mucous membranes of the alimentary tract and therefore the relationship with the environment is quite different from that with clostridia.

*FUSOBACTERIUM NECROPHORUM*

*Fusobacterium necrophorum* is the cause of calf diphtheria and liver abscesses in cattle and other ruminants. The organism is also involved in foot lesions of cattle, which begin as an interdigital dermatitis, and in footrot (epidermatitis contagiosa digitalis) of sheep.
Reservoir of the agent

*F. necrophorum* is found in animals as a commensal organism which colonises the mucous membranes, especially those of the alimentary tract and adjacent structures, e.g., teeth and tonsils. The organism has been encountered in faeces of normal animals and is capable of surviving outside the animal host for more than eight weeks and possibly for several months, even in soil (10).

Mode of transmission

*F. necrophorum* is a commensal inhabitant of mucous surfaces of animals and is acquired promptly after birth. An extrinsic source directly related to a particular episode of disease caused by this organism cannot usually be identified, except in a handful of traumatic exposures.

**BACTERIOIDES SPP.**

Purulent and necrotising processes in animals, regardless of host species and anatomical location, appear to be favourable subjects for colonisation by *Bacteroides* spp. and may at least be complicated by their presence. *B. melaninogenicus* is by far the most common member of the genus found in infections. Footrot in sheep is the result of the combined or sequential effort of *F. necrophorum* and *B. nodosus*. *Bacteroides* spp. and *F. necrophorum* are also isolated in cases of summer mastitis in cattle (31).

Reservoir of the agent

*Bacteroides* spp. are found as commensal organisms which colonise the mucous membranes and are acquired promptly after birth, as is the case with *F. necrophorum*. In contrast with *F. necrophorum*, *B. nodosus* is not present in healthy flocks of sheep which are free from footrot. Pastures are free from footrot infection approximately two weeks after the removal of the infected flock (8).

Mode of transmission

*Bacteroides* spp. colonise the mucous membranes soon after birth. In footrot, *B. nodosus* is the only organism in the lesion that must be transferred from one animal to another to transmit the disease. However, its establishment in the epidermis is preceded by a colonisation of the stratum corneum by *F. necrophorum* apparently derived from faeces (25). The most common predisposing factors for footrot transmission are moisture, warmth and foot injuries (8, 9). The occurrence of summer mastitis is related to the foraging activities of *Hydrotaea irritans* (24, 30).

CONCLUSION

Diseases caused by spore-bearing anaerobes are mainly contracted through the environment because pathogenic clostridia are common in the soil and in the dejecta of animals. Organisms of the resistant spore form live for long periods in the soil
and may be acquired by susceptible animals through wound infection or by mouth. As an exception, botulism is caused by the ingestion of toxic food or by eating parts of carcasses or by chewing bones contaminated with *botulinum* toxin. Transmission of clostridial diseases through direct contact has no significance.

The relationship with the environment is quite different in infections caused by non-spore-forming anaerobic bacteria. They are commensal inhabitants of mucous surfaces and are acquired promptly after birth. In footrot, *B. nodosus* is transferred from one animal to another to transmit the disease.

The most important methods of control against anaerobes are:

- the destruction of carcasses of domestic and wild animals so that viable spores or toxic elements will not contaminate the environment;
- good farm management, including hygienic conditions, good nutrition and the reduction of predisposing environmental factors;
- the vaccination of susceptible animals and the distribution of sufficient colostrum to new-born animals.

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**BACTÉRIES ANAÉROBIES PATHOGÈNES ET ENVIRONNEMENT. — J. Haagsma.**

Résumé: Cet article traite de la distribution des bactéries anaérobies pathogènes dans l'environnement et de leurs rapports avec les maladies animales. Il convient d'établir une distinction entre les anaérobies sporulées (clostridies) et les anaérobies non sporulées à coloration de Gram négative. La principale source des clostridies est le sol, mais on les trouve aussi dans la poussière, les eaux usées, les cours d'eau, les lacs, l'eau de mer, le lait, les légumes, la viande fraîche, le poisson, les insectes et le tube digestif. Les anaérobies non sporulées à coloration de Gram négative sont aussi largement répandues parmi les animaux dont elles colonisent surtout les muqueuses digestives.

Après une introduction générale et une partie consacrée à l'isolement des anaérobies, l'auteur discute des différentes affections provoquées par les clostridies (*botulisme*, tétanos, infections à *Clostridium chauvoei*, œdème malin, hépatite nécrotique infectieuse, entérotoxémie et gangrène gazeuse) et par les anaérobies à coloration de Gram négative (infections à *Fusobacterium* et à *Bacteroides* telles que diphtérie, piétin, etc.). Il fournit notamment des informations relatives aux réservoirs de ces agents pathogènes et à leur mode de transmission.

BACTERIAS ANAEROBIAS PATÓGENAS Y MEDIO AMBIENTE. – J. Haagsma.

Resumen: El artículo trata de la distribución de las bacterias anaerobias patógenas en el medio ambiente y de su relación con las enfermedades animales. Es necesario diferenciar los anaerobios esporágenos (clostridios) de los anaerobios no esporágenos gramnegativos. El principal habitat de los clostridios es el suelo, pero también se los encuentra en el polvo, las aguas residuales, los ríos, los lagos, el agua de mar, la leche, las legumbres, la carne fresca, el pescado, los insectos y el tracto gastrointestinal. Los anaerobios no esporágenos gramnegativos también se encuentran muy difundidos entre los animales, de los que colonizan sobre todo las mucosas digestivas.

Después de la introducción general y una parte consagrada al aislamiento de los anaerobios, el autor comenta las diferentes afecciones provocadas por los clostridios (botulismo, tétanos, infecciones por Clostridium chauvoei, edema maligno, necrobacilosis hepática, enterotoxemia y gangrena gaseosa) y por los anaerobios gramnegativos (infecciones por Fusobacterium y por Bacteroides, como difteria, pedero – también llamado dermatitis interdigital ovina, etc.), suministrando informaciones relativas a los reservorios de estos agentes patógenos y a su modo de transmisión.

PALABRAS CLAVE: Anaerobios gramnegativos - Anaerobios patógenos - Clostridium - Enfermedades - Medio ambiente - Reservorios - Transmisión.

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REFERENCES


