CLOSTRIDIUM PERFRINGENS TYPE A IN A FREE-RANGING FAWN? -- *Clostridium perfringens* is a pathogenic, anaerobic bacterium (Kummeneje and Bakken 1973, Smith 1975) commonly located in soil, water, feces, domestic animal feed, and animal gastrointestinal tracts (Niilo 1980, Niilo 1993, Songer 1996, East et al. 1998). Five bacterial types have been identified (i.e., A, B, C, D, and E) and are differentiated by their ability to produce four major lethal toxins: alpha, beta, epsilon, and iota (Smith 1975). Type A is most widespread in the intestines of warm-blooded animals and in soils (Songer 1996). Alpha toxin is produced primarily by type A (Russell 1970). Although type A has been studied extensively, its role in pathogenesis of diseases is not fully understood (Niilo 1980) and diagnosing type A induced disease can be difficult. Types B, C, D, and E are less common in the intestinal tract and can be located in areas where disease produced by these organisms is occurring continually at low levels within a population (Niilo 1980). Type B produces alpha, beta, and epsilon toxins, can cause lamb dysentery, and is relatively rare in North America (Songer 1996). Type C produces alpha and beta toxins and type D produces alpha and epsilon toxins. Type C and D infections were confirmed in several domestic animals (e.g., cattle [*Bos taurus*], sheep [*Ovis aries*], horses [*Equus caballus*], chickens [*Gallus gallus*], and dogs [*Canis lupus*]) worldwide and typically are associated with sudden changes in diet or from continuous feeding of high levels of feed concentrates. Type E is the only strain producing iota toxin and is an uncommon cause of diseases originating in the intestine (i.e., enteric diseases; Songer 1996). Further research is needed to clarify the role of iota toxin in the pathogenesis of intestinal infections.

The general mode of entry for toxin producing strains of *C. perfringens* is ingestion of contaminated food or water (Niilo 1993). In the presence of a high concentration of carbohydrates in the digestive tract of the animal, the bacteria multiply rapidly, and release a toxin (Russell 1970, Kimberling 1988) causing inflammation and hemorrhaging in the intestine (i.e., enteritis). Death of the host quickly follows, usually within 6 to 12 hours (Smith 1975, Kimberling 1988). All toxin producing strains of *C. perfringens* type A, B, C, D, and E can cause death (Songer 1996).

Enteritis and enterotoxemia are common enteric diseases caused by *C. perfringens* that can lead to death of the host. Enteritis is the inflammation of the intestine and enterotoxemia is the inflammation and hemorrhaging of the intestine with the release of a lethal toxin (i.e., alpha, beta, epsilon, and iota). *Clostridium perfringens* induced enteric diseases have been reported in domestic sheep (type A-E; Kimberling 1988, De La Rosa et al. 1997), horses (type A-C; East et al. 1998), alpacas (*Lama pacos*; type A; Ellis 1997), cattle (type A-E; Griner and Bracken 1953, Songer 1996), poultry (type A and C; Long et al. 1974), and swine (*Sus scrofa*; type A and C; Olubunmi and Taylor 1985). In addition, *C. perfringens* induced enteric diseases have been reported in captive animals such as chital (*Axis axis*;

type C; Arora 1987), Siberian ibex (*Capra siberica*; type A; Russell 1970), and fallow deer (*Dama dama*; type D; English 1985). *Clostridium perfringens* induced enterotoxemia has been diagnosed in free-ranging mule deer (*Odocoileus hemionus*; type C; Wyoming Game and Fish Department 1982), and caribou (*Rangifer tarandus*; type A; Kummeneje and Bakken 1973). We were unable to locate a documented case of *C. perfringens* type A in free-ranging white-tailed deer (*Odocoileus virginianus*).

On 19 February 2001, a nine-month-old radiocollared female white-tailed deer was located dead near Lamberton, Minnesota. The animal was part of a cooperative white-tailed deer study conducted by the Minnesota Department of Natural Resources and South Dakota State University (SDSU). The white-tailed deer was lying on its side on a snow-packed trail on property where the landowner provides supplemental feed (i.e., corn [*Zea mays*]) for white-tailed deer during winter. Ambient temperature was 0°C. However, the animal carcass had not begun to freeze as the joints were flexible. We estimated that the animal was located within 12 hours of death. Loose feces were located in the immediate area and the animal showed no signs of external trauma except for a minor abrasion over the right abdominal wall that likely occurred when the animal was captured approximately one month earlier.

The fawn was transported immediately to Ron Kuecker, D.V.M., Windom, Minnesota, and a necropsy revealed bloody fluid in the abdominal cavity and upper small intestine, a blood tinged, liquid abomasal content, and petechial hemorrhage on the heart. The rumen and reticulum were compacted with corn. Preliminary diagnosis was acute hemorrhagic enterotoxemia caused by *C. perfringens*.

The following day, the partially-frozen carcass was submitted to the Animal Disease Research and Diagnostic Laboratory (ADRDL) at SDSU to confirm preliminary diagnosis. Gross necropsy revealed moderate to regionally severe diffuse mucosal hemorrhage throughout the small intestine. No other significant histologic lesions were observed in the tissues and organ systems examined. Bacteriological tests (i.e., anaerobic cultures, polymerase chain reaction [PCR] assays) indicated the presence of *C. perfringens* type A in three sections of small intestine. Polymerase chain reaction assays are used to detect the presence of specific toxin's genes in affected animals, and have been a successful method for determining the potential ability of an isolate to produce enterotoxin (Songer 1996). No pathogenic aerobic organisms were cultured from the lung, liver, kidney, or intestine. Immunohistochemical staining of the intestine was negative for bovine viral diarrhea (BVD).

Clostridium perfringens type A induced enterotoxemia was the suspected cause of death of the animal. However, autolysis of the gastrointestinal tract prevented histologic evaluation necessary to confirm this diagnosis. The intestinal hemorrhage and enteritis were believed to be caused by *C. perfringens*, but we

were unable to test for the presence of the lethal alpha toxin that would have confirmed enterotoxemia.

Although confirmation was not possible, necropsy, bacteriological tests, and pathological results suggested *C. perfringens* type A induced enterotoxemia. Ancillary evidence at the mortality site was concurrent with other documented cases of enteritis caused by *C. perfringens* including: a young animal in good condition (Russell 1970, English 1985, Bildfell et al. 2001), insult altering rich diet (Kummeneje and Bakken 1973, Niilo 1980, Songer 1996), diarrhea present at site of mortality (Blackwell et al. 1991, East et al. 1998), sudden death (English 1985, Songer 1996), significant intestinal hemorrhage (Kummeneje and Bakken 1973, Niilo 1980, 1993), abundant presence of a potentially lethal toxin producing strain of *C. perfringens* (Kummeneje and Bakken 1973, Songer 1996, Ellis 1997), and lack of alternative causes of mortality.

Confirming *C. perfringens* type A as the primary causative agent is difficult. First, younger animals are generally more susceptible to enteric diseases because of an absence of well-established intestinal microenvironment (Songer 1996), which creates a defense against colonization of newly introduced *C. perfringens* (Kimberling 1988). Second, *C. perfringens* is present commonly in the intestinal tracts of warm-blooded animals and generally is innocuous. It also is a frequent postmortem invader of the gut and other tissues (Songer 1996). Type A in particular is isolated easily from tissues, effusions, and intestinal tracts of dead animals. Within a few hours post-mortem, the bacteria rapidly multiply masking the presence of other potential causes of death (Niilo 1980). Although we used PCR (Meer and Songer 1997) assays to identify the *C. perfringens* as type A, it was not possible to test for the actual presence of toxin due to the elapsed time between death of the fawn and initiation of PCR tests (~24 hours). Toxins produced by *C. perfringens* are undetectable in the intestinal contents within a few hours after death (Kummeneje and Bakken 1973). Third, severe autolysis of the fawn's tissue cells prevented a thorough histological examination and might have obscured enterotoxemia symptoms such as enteric lesions (Songer 1996).

We were unable to find a documented case of *C. perfringens* type A enteric disease in free-ranging white-tailed deer. Nevertheless, due to the ubiquity of the bacteria in the environment, such events likely occur. *Clostridium perfringens* induced illnesses might be more common in northern latitudes where supplemental feeding of single source diets (e.g., corn) occurs following severe winter storm events. Due to incomplete understanding of the role of *C. perfringens* in pathogenesis, precautions in diagnosing type A diseases, and limited clinical history associated with free-ranging animals, a confirmed case of enteritis or enterotoxemia in a wild animal might continue to be a rare occurrence. We recommend that *C. perfringens* induced enteric disease should be considered as a cause of death anytime an ungulate dies suddenly and an insult altering rich diet is involved.

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