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EPIZOOTIC NECROTIC ENTERITIS IN WILD GEESE

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ABSTRACT: Outbreaks of a disease characterized by severe necrotic enteritis occurred among Canada geese (*Branta canadensis*), lesser snow geese (*Anser caerulescens caerulescens*), Ross' geese (*A. rossi*), and white-fronted geese (*A. albifrons*) on lakes in Saskatchewan and Manitoba during the autumn of 1983, 1984 and 1985. Ducks using the lakes were apparently not affected. Lesions in the geese closely resembled those described in enteritides in other species associated with the proliferation of *Clostridium perfringens* in the small intestine. *Clostridium perfringens* was present in large numbers in the affected areas of the intestine of the geese; other pathogens were not identified. It is hypothesized that an abrupt change in diet as geese begin to feed on grain disrupts the intestinal microflora, allowing *C. perfringens* to proliferate in the upper small intestine. Toxins produced by the bacteria then cause mucosal necrosis. Protease-inhibitory substances in some grains might also have a role in the disease.

Key words: Wild geese, enteritis, *Clostridium perfringens*, Saskatchewan, Manitoba, pathology, migration.

INTRODUCTION

Severe fatal enteritis characterized by extensive, grossly visible mucosal necrosis is an uncommon disease in birds. Two well-defined diseases of this type that occur in gallinaceous birds are associated with proliferation of *Clostridium* spp. in the intestine. "Necrotic enteritis" is a disease of growing broiler chickens that has been reproduced by feeding a diet containing *Clostridium perfringens* type C and type A, or by intraintestinal inoculation of the organism (Long and Truscott, 1976; Al-Sheikhly and Truscott, 1977). "Ulcerative enteritis" or "quail disease" caused by infection with *C. colinum* occurs in many species including bobwhite quail (*Colinus virginianus*), domestic chickens and turkeys, and is transmitted through spores passed in the droppings (Peckham, 1978). Occasional cases of "necrotic enteritis" have been reported in domestic ducks (Leibovitz, 1973) and captive geese (Wobeser, 1981). Jasmin et al. (1972) reported an outbreak of disease involving several species of waterfowl in Florida, in which many of the birds had "mucoid necrotic enteritis" that was thought to have been caused by *C. perfringens* type C. A similar "enterotoxemia" caused by *C. perfringens* has been diagnosed in wild ducks in the Rhine area of Germany (Neumann, 1983). Siegfried and Brand (1982) reported necrotizing enteritis in Canada geese (*Branta canadensis*) in Wisconsin and Illinois. This report describes three outbreaks of a disease characterized by necrotic enteritis that occurred in wild geese in western Canada during 1983–1985.

CASE HISTORIES

The first of the three outbreaks occurred on Antelope Lake, a shallow saline wetland with a surface area of approximately 1,000 ha in southwestern Saskatchewan (Fig. 1). The history of this outbreak was determined in retrospect because the extent of the problem was not recognized at the time. In early November 1983, a resident of the area noted dead geese on the lake and contacted the Department of Parks and Renewable Resources (DPPR). An officer visited the site and concluded that the dead birds were the result of crippling during the hunting season which was still in progress in the area. The area of lake examined and the number of dead birds examined at that time is not known; no birds were necropsied. In mid-February, 1984, staff of Ducks Unlimited Canada (DU), who

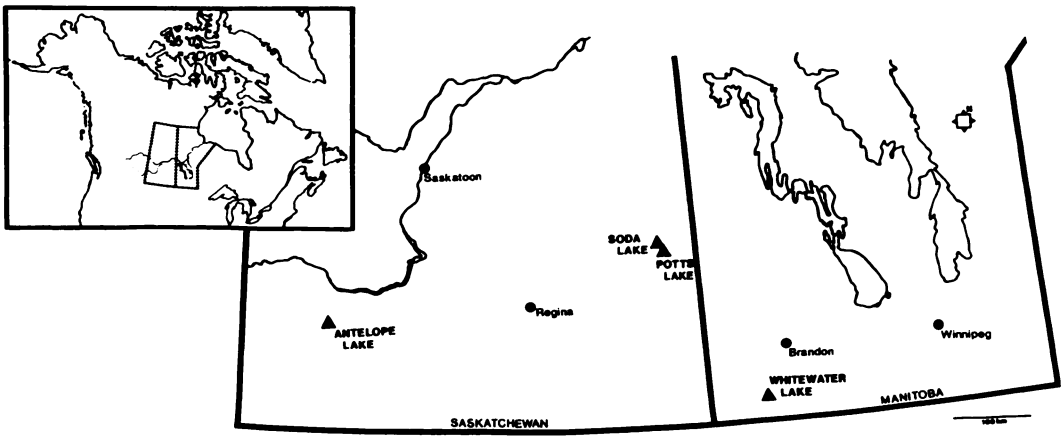

FIGURE 1. Map of southern Saskatchewan and Manitoba showing location of three outbreaks of necrotizing enteritis in geese.

were placing nesting structures for geese on Antelope Lake reported many dead geese frozen in and on the ice. We visited the lake on 18 February 1984 and a sample of 32 birds consisting of 18 Canada geese, 8 white-fronted geese (*Anser albifrons*), 5 Ross' geese (*A. rossi*) and a pintail (*Anas acuta*) was collected. The dead birds were concentrated in the northeastern corner of the lake where most were frozen into the ice, but some were present on the surface of the ice or on the open shoreline. Many carcasses had been fed on by scavengers. Through interviews with local residents we learned that dead birds were first seen about 7 November, and birds continued to die until 2 December when the lake froze completely and geese left the area. The presence of dead geese on top of the ice is evidence that birds were dying as the lake froze. In the spring of 1984, DU personnel collected and disposed of the carcasses of approximately 800 geese from Antelope Lake. Canada geese comprised 80% of these birds, with the remainder consisting of approximately equal numbers of Ross' geese, white-fronted geese and lesser snow geese (*Anser caerulescens caerulescens*).

The second outbreak occurred in 1984 on two small saline lakes (Soda, Potts) in eastcentral Saskatchewan (Fig. 1). These lakes are approximately 130 and 60 ha in area, respectively, and are located about 7 km apart. The outbreak on Soda Lake began prior to 26 September when it was reported to the regional biologist, DPRR. On 28 September all dead geese on the lake were collected and submitted to the Western College of Veterinary Medicine (WCVM) for examination. These consisted of 128 lesser snow geese, seven Ross' geese and three white-fronted geese. Two other lesser snow geese, one Ross' goose and one white-fronted goose appeared sick, but were able to fly. Approximately 300 lesser snow geese and 200 Canada geese, as well as 300–400 ducks comprised of approximately equal numbers of lesser scaup (*Aythya affinis*) and mallards (*Anas platyrhynchos*) that appeared normal were present on the lake.

Mortality probably began about the same time on nearby Potts Lake. On 5 October when the lake was visited, more than 200 dead geese were present along the shoreline. Discrete "splashes" of fecal material that were red-black and strongly suggestive of partially digested blood were seen in several places among the normal goose excreta on the shoreline. Dead birds were comprised of approximately equal num-

bers of Canada geese and lesser snow geese, with only a few dead Ross' geese and white-fronted geese seen. About 50 apparently healthy Canada geese were present as well as ducks of several species including blue-winged teal (*Anas discors*), gadwall (*A. penelope*) and northern shoveler (*A. clypeata*). The latter were feeding actively on suspended material in the water among the goose carcasses. Four sick Canada geese were unable to fly. Three of these stood at the water's edge in a "trance-like" state, and one was seen to drink repeatedly. When these birds were approached closely, they roused suddenly and began to swim away slowly; flight was not attempted. The other sick bird was swimming slowly about 2 m from shore, with the bill in the water and the eyes closed. These birds were collected and necropsied immediately.

The third outbreak occurred on Whitewater Lake, a major staging area for geese in southwestern Manitoba (Fig. 1) in 1985. This is a very large, shallow, saline lake with a surface area of approximately 9,000 ha. In the autumn of 1985, the area was used by about 250,000 geese, predominantly lesser snow geese. As was the case at Antelope Lake, this outbreak was not recognized until after freeze-up when live birds had left the area. During a retrospective investigation by staff of the Manitoba Department of Natural Resources (DNR), a hunter was interviewed who had observed "a lot" of dead geese on the shore of one area of the lake on 19 and 26 October. He recalled that there was a dead goose "every 20 feet along the shore of a bay." He had skinned a few of these geese and found that some had been shot, whereas others were in good body condition with the "crop" full of grain that smelled "rotten." A nearby resident farmer had observed many dead geese on the shore 16–17 November when he drove around the lake. Carcasses were most numerous on the north and east sides of the lake where there was "a carcass every 10–15 feet and occasionally in groups of four or five," but dead birds were seen all around the lake. He collected about 40 of the dead frozen lesser snow and Canada geese with the intention of having some mounted. The carcasses were placed outdoors where they remained frozen. On 10 December, a technician of the DNR visited the farmer in response to a belated report of geese dying on the lake, and a number of the frozen goose carcasses were collected. The technician drove along about 10 km of the north shore of Whitewater Lake and observed approximately 100 remnants of goose carcasses. Most had been fed on by scavengers; a few that were undisturbed were collected. The dead birds were lying on the exposed shoreline between the marginal vegetation and the water's edge and were not frozen into the mud. A sample of 18 carcasses (13 lesser snow geese and five Canada geese), comprised of birds from the farmer's yard and those collected on the lake shore was submitted to the WCVM.

MATERIALS AND METHODS

The type of analyses used varied among the outbreaks because of the quality and type of specimens available. In all instances, geese were necropsied. Body condition, presence and type of food, and gross lesions were recorded. Portions of major body organs collected from a subsample of birds from each lake were preserved in 10% neutral buffered formalin, embedded in paraffin, sectioned at 5 μm and stained with hematoxylin and eosin for histological evaluation. Gram stain was used on some sections. Portions of intestine, liver and occasionally lung from a subsample of birds from each lake were submitted to the Department of Veterinary Microbiology, WCVM for bacteriological examination. All such specimens were cultured aerobically on 5% sheep blood agar (BA) and on MacConkey agar at 37 C. Specimens from the intestine were also cultured anaerobically (BBL Gas Pak System, Becton Dickinson and Co., Cockeysville, Maryland 21030, USA) on BA at 37 C. *Clostridium perfringens* was identified on the basis of anaerobiasis, morphology on Gram-stained smears, presence of a double zone of hemolysis on BA and reaction when inocu-

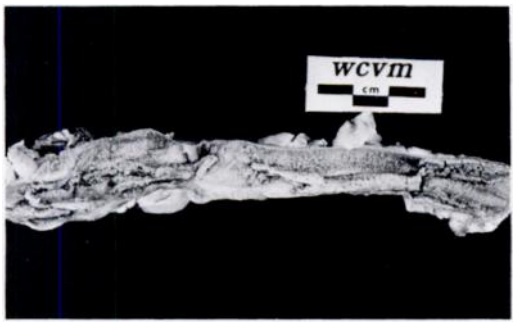

FIGURE 2. Intestine of affected Canada goose. The mucosal surface is covered by adherent necrotic material.

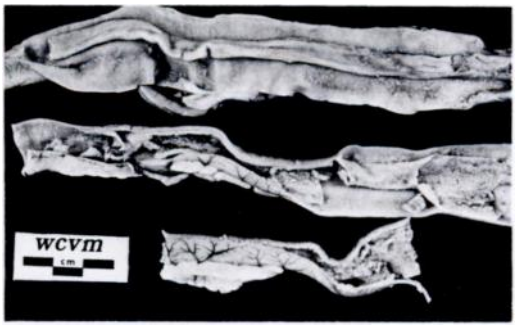

FIGURE 3. Intestine of affected white-fronted goose. Adherent fibrino-necrotic material covers the mucosal surface, forming a complete cast in one segment.

lated into litmus milk. Portions of intestine from a subsample of geese from each lake were submitted to the diagnostic parasitology laboratory, Department of Veterinary Microbiology for examination for helminths and coccidia. A sample of winter wheat collected 6 October in a field between Soda and Potts Lakes that had been used extensively by feeding wild geese, and a pooled sample of winter wheat from the esophagus of geese found dead on Soda Lake were screened for tricothecene mycotoxins using a bio-assay technique (Hayes and Schiefer, 1979).

RESULTS

Necropsy findings

Necropsies were performed on 32 birds from Antelope Lake in 1983. One Canada goose had gunshot wounds. No significant gross lesions were found in the pintail; cause of death of this bird was not determined. A few of the remaining birds had blood-tinged subcutaneous fluid over the abdomen, and many had edematous lungs, but these may have been an artifact of freezing. All birds were in moderate to excellent body condition. The upper alimentary tract was empty in five geese; two Canada geese and one white-fronted goose had sedge material in the esophagus, and the remaining 22 birds had intact kernels of wheat in the esophagus and proventriculus. In 17 of these birds the wheat was of the Durum type, two others had winter wheat, and three had hard red spring wheat. Lesions in the intestine were similar in type in all the geese, but were most severe in white-fronted geese and least severe in Ross' geese. The small intestine was swollen and turgid in all birds, and was very friable when handled. The content had a characteristic foul smell and was usually grey-tan or blood-tinged. In Ross' geese there were usually only focal areas of mucosal necrosis in the distal third of the small intestine, together with the presence of bloody fluid. Canada geese had a focal to diffuse layer of adherent tan-yellow necrotic debris covering the mucosa of the mid and lower small intestine (Fig. 2). In many white-fronted geese and a few Canada geese an adherent fibrino-necrotic cast was present in the lower small intestine (Fig. 3). The colon and ceca were grossly normal and empty. Two Canada geese, one Ross' goose and one white-fronted goose had intact kernels of wheat throughout the small intestine.

Necropsies were performed on 40 geese from Soda Lake in 1984, including representatives of all three species. Twenty-seven of these birds had winter or Durum wheat in the esophagus and proventriculus and all were in moderate to excellent body condition. Almost all birds had roughening and thickening of the areas of the gizzard between the koilin pads, and grain awns were found embedded in this area in a few. The intestinal lesions were as de-

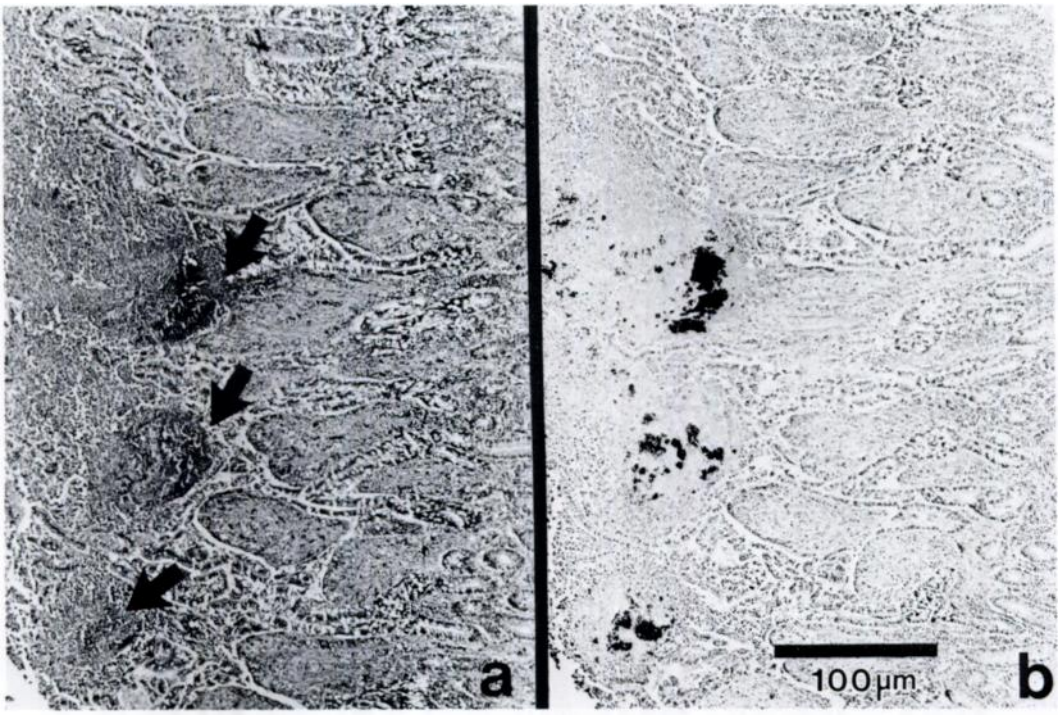

FIGURE 4. Portion of small intestine from affected Canada goose. (a) Three small foci of necrosis are evident on the lumen end of villi (arrows). Hematoxylin and eosin. (b) Duplicate section showing presence of Gram positive bacteria in these foci. Brown and Brenn stain.

scribed in birds from Antelope Lake, and variation in severity among species followed a similar pattern. Ross' geese had the least obvious lesions, with the mid to lower small intestine having a rough granular appearance with relatively little adherent necrotic material. In snow geese the mucosa of the mid to lower small intestine was obviously necrotic, while in white-fronted geese there was usually a fibrino-necrotic cast that occasionally extended proximally to the duodenum.

The geese found dead and necropsied in the field at Potts Lake in 1984 had similar lesions to those described above. The four sick Canada geese (two adult and two juvenile) collected were all in good body condition. Two had winter wheat in the esophagus. In one, the entire small and large intestine was hyperemic, with excessive fluid and fibrin casts present from the duodenum to the level of the cecum. The colon was empty and the mucosa appeared normal. In the other three birds, there were multiple foci of necrotic material on the duodenal mucosa, with excessive fluid and fibrino-necrotic debris or casts in the lower small intestine.

The geese from Whitewater Lake in 1985 were also in good body condition. Seventeen of the 18 birds had food in the upper alimentary tract; in 11 this was winter wheat, one had white beans, four had a mixture of winter wheat and barley and one had barley and beans. Seven birds had intact kernels of wheat in the lower small intestine. All had necrotic enteritis, involving the mid and distal portions of the small intestine. In three of five Canada geese and two snow geese, there was a fibrino-necrotic membrane lining the intestinal lumen.

Microscopic findings

The predominant lesion in geese from all lakes was necrosis of epithelium of the small intestine. This change was usually restricted to the lumenal surface and involved the tips of the villi. In a few instances the necrosis extended the full depth of the mucosa. The change was not uniform, usually only involving focal areas consisting of one or a few adjacent villi. In these foci, the lumenal surface of the villus was usually covered with amorphous eosinophilic debris containing many large bacteria; with bacteria and necrosis extending in the lamina propria of the villus (Figs. 4, 5). The distal portions of adjacent epithelium-covered villi were often dilated because of engorgement of the capillaries. The lamina propria contained small numbers of mononuclear cells and heterophils. The bacteria on the surface and within the necrotic villi were consistently large Gram-positive bacilli. In those birds that had a grossly visible diphtheritic membrane or cast lining the intestine, the microscopic lesions were similar to those described. The villi were usually short in these areas with the overlying membrane adherent to villi from which the luminal epithelium was absent. The casts contained many Gram-positive bacteria.

One Canada goose found sick at Potts Lake had focal areas of necrosis of villar epithelium with bacterial colonization as described above in the upper small intestine, but also had a different lesion in the distal small intestine. In this area the mucosa of the villi was totally absent and the stroma of the villi had collapsed. Epithelium was still present in the crypts. The surface layer of the collapsed stroma was necrotic, and this was overlain by a dense layer of single-celled protozoa that appeared to have flagellae. Gram-positive bacteria were relatively sparse in the lumen of the intestine in this area.

Significant lesions were not found other than in the intestine. Because of the condition of the specimens available from Antelope and Whitewater lakes, microscopic examination of tissues from birds from these lakes was limited. However, despite artifact caused by freezing and thawing, the lesions in six geese examined from Antelope Lake and four from Whitewater Lake were essentially identical to those found in birds from Soda and Potts lakes.

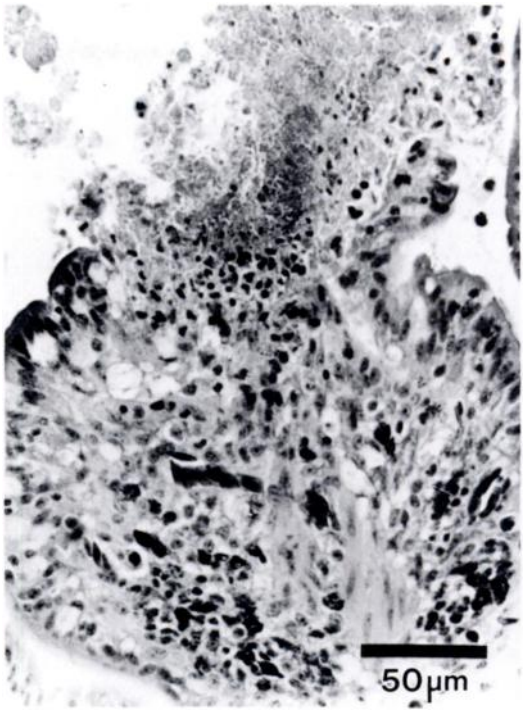

FIGURE 5. Tip of a single villus (Canada goose) with necrosis and bacterial colonization. Hematoxylin and eosin.

Microbiologic findings

As might be expected from the type of specimens available for culture, a variety of bacteria were isolated from the tissues of these geese. Cultures of lung and liver usually yielded few to many colonies of bacteria including *Aeromonas* sp., *Streptococcus* sp., *Escherichia coli*, and/or *Pseudomonas* sp. These were considered to be contaminants and no specific etiologic agent was identified from these tissues.

Routine aerobic culture of the mid-small intestine also yielded a variety of bacteria; usually a combination of *E. coli*, *Streptococcus* sp., *Aeromonas* sp. and *Pseudomonas* sp. Eleven geese from Antelope Lake were examined microbiologically including three Ross' geese, five Canada geese and three white-fronted geese. *Clostridium perfringens* was not isolated from the Ross' geese, but was isolated in moderate numbers on anaerobic cultures from four of the Canada geese, and from two of the white-fronted geese.

Four of the "fresher" specimens received from Soda Lake were cultured. Aerobic cultures of intestine yielded bacteria of the same genera as described above. Additionally, *Plesiomonas shigelloides* was isolated in moderate numbers from the intestine of three birds. Organisms suggestive of *Clostridium* sp. were evident in Gram-stained smears of intestinal content from all of these birds and *C. perfringens* was isolated as a heavy growth on anaerobic cultures from these birds.

Clostridium perfringens was isolated in large numbers from the intestine of three of the four sick geese collected at Potts Lake. An organism identified as a Non 01, non-enterotoxigenic strain of *Vibrio cholerae* was isolated on aerobic culture from the intestine of two of these birds, in addition to the same genera of bacteria described from geese from other lakes.

Intestines from four geese from Whitewater Lake were examined. All had moderate to large numbers of Gram-positive rods present on smears, and *C. perfringens* was isolated from all.

None of the *C. perfringens* isolates from geese were identified as to toxin type.

Parasitologic findings

There were no consistent parasitologic findings. One of four geese examined from Soda Lake had a small number of unsporulated oocysts of an unidentified coccidium in its excreta. Renal coccidiosis and cysts of *Sarcocystis* sp. in skeletal muscle were found microscopically in a few geese.

Other analyses

Trichothecene toxin was not identified in grain collected in the field, or from the esophagus of geese from Soda Lake.

DISCUSSION

The pathology observed in the 50 wild geese examined is very similar to that reported in necrotic enteritis of fowl (Helmboldt and Bryant, 1971). Also, there are many similarities to enteritis described in several mammalian species in association with *C. perfringens* infection such as necrotic enteritis of baby pigs (Bergeland, 1981), enteritis necroticans or "Pigbel" of humans (Lawrence and Walker, 1976), hemorrhagic necrotizing enteritis of foals (Sims et al., 1985), and hemorrhagic enterotoxemia of lambs (Niilo, 1986). Common to these enteritides is proliferation of a toxigenic strain of *C. perfringens* in the small intestine, with epithelial necrosis beginning at the tip of villi, and with subsequent necrosis and bacterial invasion then proceeding deeper into the mucosa. The nature of the disease, the intestinal lesions, and the presence of large numbers of *C. perfringens* in the intestine of the geese, lead us to believe that this bacterium has a causative role in the disease of geese. However, demonstration and characterization of toxin in the intestine of affected birds, and experimental reproduction of the disease will be required to confirm the etiology. Siegfried and Brand (1982) also found clostridial organisms in association with necrotic enteritis in Canada geese of the Mississippi Valley Population, but did not identify the organism.

The epizootiology and pathogenesis of enteritis associated with *C. perfringens* are not fully understood, but seem to involve conditions that allow the organism *C. perfringens* to colonize and proliferate in the small intestine, produce necrotizing toxins,

and invade damaged mucosa. *Clostridium perfringens* is described as being "ubiquitous in nature and is part of the normal intestinal microflora of healthy animals" (Gillespie and Timoney, 1981, p. 208), but the occurrence of toxigenic strains and their distribution along the length of the intestine is less clear. Shane et al. (1984) found that *C. perfringens* was infrequent in the small intestine of chickens, and this agreed with earlier studies which indicated that while *C. perfringens* was consistently present in the ceca of chickens it occurred only sporadically higher in the intestine (Timms, 1968; Barnes et al., 1972). The occurrence and prevalence of *C. perfringens* in the intestine of normal wild geese is unknown, so one can only speculate whether there had been proliferation of a pre-existing organism or establishment of a new organism in the small intestine of the affected birds. Experiments in mice (Hudault et al., 1983) and sheep (Niilo and Cho, 1985) suggest that it is difficult for *C. perfringens* to colonize the small intestine of an animal with an established microflora.

A number of mechanisms have been proposed to explain the proliferation of *C. perfringens* and the mucosal damage caused by toxins in the various forms of necrotizing enteritis. Many of these involve sudden changes in diet and/or decreased levels of proteolytic enzymes in the intestine. The latter is thought to be important because the necrotizing beta toxin of *C. perfringens* type C is readily inactivated by trypsin. The disease in pigs and foals is limited to neonates; it has been suggested that neonatal piglets may be made susceptible to infection because of trypsin inhibitors in colostrum (Griner, 1963), and Bergeland (1972) referred to earlier studies which suggested that decreased proteolytic enzyme activity in the intestine as a result of other enteric infections might allow *C. perfringens* to increase in numbers and toxin to accumulate. Enteritis necroticans in humans is associated with sudden "dietary overindulgence." In Papua New Guinea this disease is thought to be a result of a diet that is very low in protein, with subsequent low pancreatic trypsin activity, and composed largely of sweet potato (*Ipomoea batatas*) which contains heat-stable trypsin inhibitors. Together, these result in inadequate proteolysis of beta toxin produced following overindulgence on *C. perfringens* contaminated food, usually at a "pig feast" (Lawrence and Walker, 1976). These authors were able to reproduce a similar disease in guinea pigs by intragastric inoculation of a culture of *C. perfringens* together with raw soybean flour which contains trypsin inhibitors, whereas inoculation of the organism alone had no effect. Niilo (1986) produced fatal hemorrhagic enteritis by dosing lambs via the intraduodenal route with *C. perfringens* type C together with soybean flour. Without soybean flour the bacteria did not cause fatal disease.

Management stress, changes in diet, alterations in feeding programs and subclinical coccidiosis have been listed as factors that may predispose chickens to necrotic enteritis (Shane et al., 1985). Decreased intestinal motility may also promote proliferation of *C. perfringens* and allow toxin accumulation (Shane et al., 1985).

Some of the factors discussed above seem to be present in wild geese during autumn migration. The species involved in these outbreaks breed in the Arctic, and consume diets obtained largely through grazing, or grubbing for aquatic roots and tubers. The diet changes abruptly when these birds reach the agricultural area of the prairie provinces, and is then comprised almost entirely of the seeds of agricultural crops. During a study of *C. perfringens* type D enterotoxaemia in sheep, Bullen (1963) found that a sudden change to a wheat diet allowed large amounts of semi-digested food to escape into the small in-

testine. Introduction of *C. perfringens* into the duodenum following the dietary change resulted in extremely rapid proliferation and toxin production by the bacterium. The presence of undigested wheat in the lower small intestine of several of the dead geese suggests that a similar phenomenon may have occurred in the birds.

Plant protease-inhibitors might also play a role. Although these compounds are usually associated with legumes, trypsin-inhibitors have also been found in various cereal grains including wheat and barley (Liener and Kakade, 1969). Beans were found in the upper alimentary tract of some of the geese from Whitewater Lake, and a number of other legume crops, including lentils and field peas, are grown on the prairies, so that geese might have access to plants with relatively large amounts of protease-inhibitors.

Enteric coccidiosis has been shown to be an important predisposing factor for necrotic enteritis in chickens (Shane et al., 1985). However, we found coccidia in only one goose.

Leibovitz (1973) described necrotic enteritis in domestic ducks as well as in captive mallards, black ducks (*A. rubripes*), Canada geese and domestic geese. The pathology of this disease was very similar to that observed in the wild geese. However, clostridia were apparently not observed, nor was the intestinal content cultured for anaerobic bacteria. Large numbers of flagellates identified as *Trichomonas* sp. and *Hexamita* sp. were found in the intestine, and were "considered as contributing to the pathogenesis" of the enteritis. Flagellates were obvious in the intestine of one goose among 50 examined in this study, but their significance is unclear. A specific search was not made for flagellates, and freezing would probably have obscured infection had flagellates been present in many of the geese.

The overall significance of this disease for goose populations is unknown. The outbreaks described were of sufficient magnitude that it seems unlikely that such die-offs have been a common occurrence on the prairies in the past without having been recognized. Consequently, this suggests that the disease may be a relatively new phenomenon. We hypothesize that the disease is the result of an abrupt dietary change resulting in disruption of the intestinal microflora allowing *C. perfringens* to proliferate and produce toxin in the small intestine. Trypsin inhibitors in the diet might have a role in lowering the ability of the bird to deal with toxins. If this hypothesis is correct, one should be able to identify some new factor responsible for the occurrence of the disease in recent years. Geese have been consuming waste cereal grains during autumn migration through Saskatchewan and Manitoba for many years. However, cropping practices have changed recently, with a marked increase in the amount of winter wheat that is grown. Legume crops such as lentils, peas and beans are also relatively recent additions, but whether these changes have any relation to the disease is speculative at present.

ACKNOWLEDGMENTS

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