Bacterial Enteritides of Poultry

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ABSTRACT Enteric bacterial infections in poultry pose a threat to intestinal health and can contribute to poor feed efficiency and livability of a flock. A variety of enteric bacterial diseases are recognized in poultry. Three of these bacterial diseases, necrotic enteritis, ulcerative enteritis, and spirochetosis, primarily infect the intestine, whereas other bacterial diseases, such as salmonellosis, colibacillosis, mycobacteriosis, erysipelas, and fowl cholera, affect a variety of organ systems in addition to the intestine. Diagnosis of bacterial enteritis requires monitoring of clinical signs in the flock and proper use of diagnostic methods such as necropsy, histopathology, bacteriology, and serology.

(*Key words*: poultry, enteritis, intestine, disease, bacteria)

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INTRODUCTION

Poultry must have a healthy and functional intestinal tract to maintain the excellent feed efficiency that is required by modern production standards. It is estimated that feed costs for poultry comprise roughly 66% of the total production costs, whether costs are for a dozen eggs, broiler chickens, or turkeys (L. Schrader, 1997, Purdue University, Department of Agricultural Economics, Krannert Building, West Lafayette, IN, 47907, personal communication). Low-grade damage to the intestinal tract by pathogenic bacteria may cause poor feed efficiency and decreased rate of gain to escalate the total production costs. In addition, more severe enteric damage by bacterial infection will cause overt illness and high mortality in a poultry flock.

Diagnosis of bacterial infections in poultry often requires the following procedures: 1) evaluation of flock history and signalment, 2) physical examination of live birds, 3) gross necropsy to identify organ lesions, 4) histopathologic examination of tissues, 5) bacterial culture and antibiotic sensitivity, and 6) serology. Certain enteric bacterial infections may be diagnosed in the field based on flock history and physical examination of sick birds. However, in most cases a definitive diagnosis should be attained by submitting dead and moribund birds to a veterinary diagnostic laboratory where more specific testing can be performed. In bacterial diseases that affect many organ systems, a cursory examination and diagnosis in the field may inadvertently overlook the intestinal tract. In fact, an intestine that is normal to the naked eye may have inflammatory lesions that can only be detected by light microscopic evaluation. Gross, histologic, or cytologic examination of the intestine is essential for determining the extent of intestinal damage, whereas bacterial culture and antibiotic sensitivity may be quite useful to direct treatment of the enteric disorder.

A variety of enteric bacterial infections pose a serious threat to gastrointestinal health and overall flock livability. Three of these bacterial diseases, necrotic enteritis, ulcerative enteritis, and spirochetosis, are primarily enteric diseases, whereas the other bacterial diseases described in this report, salmonellosis, colibacillosis, mycobacteriosis, erysipelas, and fowl cholera, affect a variety of organ systems with the alimentary tract being involved to an equal or lesser extent. This report will emphasize the enteric aspects of the bacterial infections.

CLOSTRIDIOSIS

Clostridium species are large, 3 to 4 μm long, anaerobic, Gram-positive bacilli that produce spores. Clostridia are widely distributed in soil and fresh water. Many of the pathogenic clostridia are normal inhabitants of the intestine of animals. Exotoxins are important virulence factors in clostridial diseases. *Clostridium perfringens* Types A–E release exotoxins in the alimentary tract (enterotoxins) that are absorbed into the

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Abbreviation Key: FT = fowl typhoid; PD = pullorum disease; SE = *Salmonella enteritidis*; UE = ulcerative enteritis.

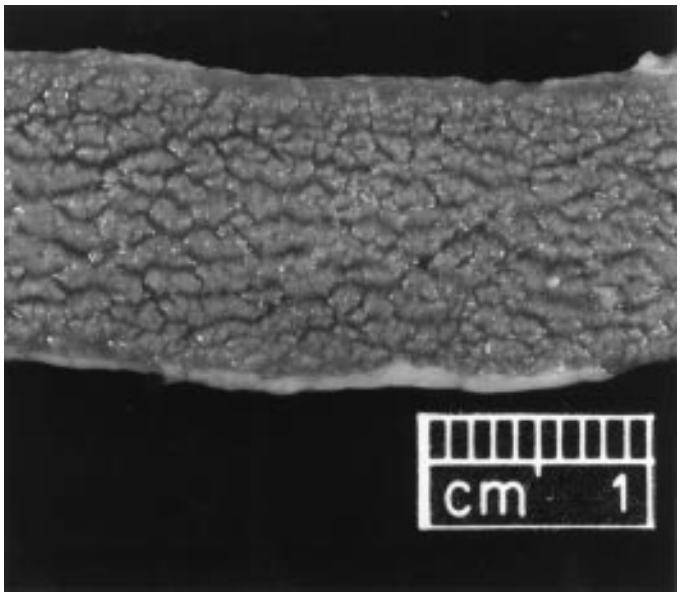

FIGURE 1. Opened small intestine of a 4-wk-old broiler chicken with necrotic enteritis caused by *Clostridium perfringens.* Note the roughened, irregular fibrinonecrotic material (pseudomembrane) that lines the mucosa of the intestine.

bloodstream to produce toxemia. Histotoxic clostridia such as *Clostridium colinum*, the agent that causes ulcerative enteritis, are invasive and produce moderately potent toxins (Quinn *et al.*, 1994a).

Necrotic enteritis of poultry is caused by toxigenic *Clostridium perfringens* Type A or C. Alpha toxin, produced by *C. perfringens* Types A and C, has been shown to produce the enteric lesions of necrotic enteritis when administered to broiler chicks (Al-Sheikhly and Truscott, 1977). *Clostridium perfringens* also produces beta toxin that may play a role in necrotic enteritis (Kohler *et al.*, 1974). Outbreaks of necrotic enteritis in poultry are most common in broiler chickens, but the disease has also been reported in commercial layers raised on the ground (Chakraborty *et al.*, 1984), cage-reared replacement pullets (Broussard *et al.*, 1986), and turkeys (Gazdzinski and Julian, 1992). *Clostridium perfringens* may often be a normal inhabitant of the intestinal tract (Shapiro and Sarles, 1949) and other factors promote microorganism overgrowth and toxin production in the gut. For example, necrotic enteritis is often preceded by or associated with enteric coccidial infection (Al-Sheikaly and Al-Saieg, 1980; Shane *et al.*, 1985). Also, high levels of wheat or fishmeal in the ration may predispose birds to develop necrotic enteritis (Branton *et al.*, 1987, Truscott and Al-Sheikhly, 1977).

Clinical signs described with necrotic enteritis include depression, ruffled feathers, diarrhea, huddling, anorexia, sternal recumbency, and a sudden rise in flock mortality (Long, 1973). Gross lesions are usually restricted to the small intestine, particularly the jejunum and ileum and less often the duodenum and cecum. The intestine is distended by gas and dark brown fluid. A discontinuous to diffuse layer of tan to grey, friable, fibrinonecrotic material (pseudomembrane) is adhered to the mucosa (Figure 1). Rare focal lesions may be observed (Hemboldt and Bryant, 1971; Shane *et al.*, 1985). Histopathologic evaluation of affected intestine reveals the fibrinonecrotic material to consist of necrotic villi, fibrin, and necrotic inflammatory cells. Large Gram-positive bacterial rods with or without other bacteria are usually scattered throughout the areas of necrosis. A sharp line of demarcation occurs between the necrotic and viable tissue (Riddel, 1987). Electron microscopic evaluation of lesions indicated that *C. perfringens* was intimately associated with the inflammation, but the microorganism did not invade viable tissue. These findings were consistent with enterotoxin release playing a primary role in lesion pathogenesis (Kaldhusdal *et al.*, 1995).

Ulcerative enteritis (UE), which is caused by *Clostridium colinum*, is a highly fatal enteric disease that primarily affects captive quail, but the disease has also been reported in other birds including chickens, turkeys, and pheasants (Berkhoff, 1985; Ononiwu *et al.*, 1978). Young quail, from 4 to 12-wk-old, are most susceptible. Acute mortality as high as 80 to 90% is not uncommon in a covey of captive Bobwhite quail. Quail may die in 1 to 3 d after experimental infection (Kondo *et al.*, 1988). It has not been possible to experimentally reproduce UE in chickens, which raises questions about what other factors (e.g., feed, crowding, other infectious agents) act to promote the disease in poultry (Bickford, 1985). Birds may die in good physical condition without premonitory signs, whereas others may appear weak and depressed with ruffled feathers and diarrhea. Intestinal lesions are widespread throughout the duodenum, jejunum, ileum, and cecum and consist of multifocal to coalescing 1 to 5 mm diameter white punctate mucosal ulcers that can usually be observed on the serosa (Figure 2). The ulcers often perforate the intestinal wall to produce peritonitis and intestinal adhesions (Kondo *et al.*, 1988). Hepatic and splenic lesions usually consist of well-demarcated, multifocal to coalescing, yellow foci of necrosis that are scattered throughout the organs. Histopathologic evaluation of affected intestine often reveals well-demarcated areas of coagulative necrosis of villi with infiltrates of heterophils and mononuclear cells. A margin of epithelioid macrophages separates necrotic and viable tissue. The necrosis often extends deep into the tunica muscularis and may ulcerate through the serosa. Colonies of large Gram-positive bacterial rods are numerous throughout the lesion. Foci of coagulative necrosis in the liver and spleen contain fibrin thrombi and colonies of Gram-positive bacteria.

COLIBACILLOSIS

Colibacillosis is an infectious disease affecting a wide variety of birds in which *Escherichia coli* is a primary or secondary pathogen. *Escherichia coli* is a Gram-negative,

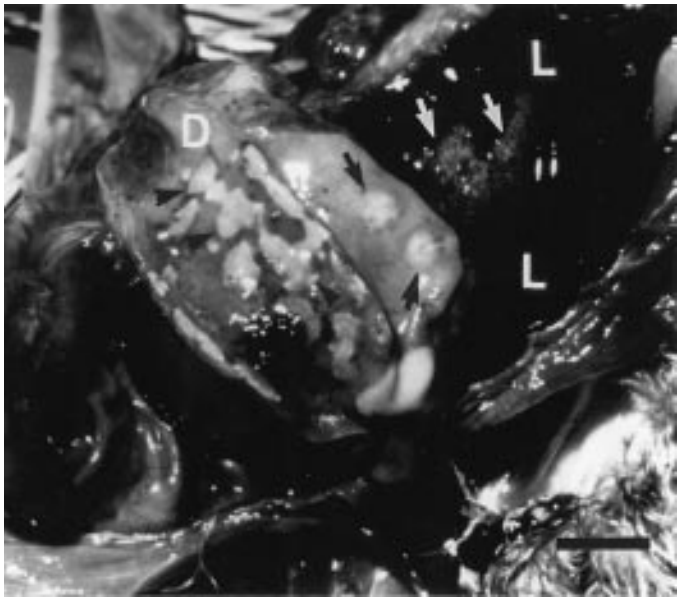

FIGURE 2. Duodenal loop (D) and liver (L) of a 5-wk-old bobwhite quail with ulcerative enteritis (UE) caused by *Clostridium colinum.* Note the pale, well-demarcated, punctate necrotic foci (black arrows) on the serosa of the unopened portion of the duodenum. The pale necrotic foci on the mucosa of the open portion of the duodenum are coalesced (black arrowheads). The liver (L) contains a pale region of necrosis (white arrows) that is typical of UE (black bar scale = 0.5 cm).

medium-sized (2 to 3 μm long) rod that is widespread in nature and is a normal inhabitant of the intestinal tract of poultry (Gross, 1994). Pathogenic serotypes of *E. coli* can often be isolated from the intestinal tracts of healthy poultry, which supports the claim that *E. coli* is often a secondary or opportunistic pathogen. Feces and dust in the poultry house are an important source of pathogenic *E. coli* (Gross, 1994). Both ingestion and inhalation of *E. coli* are potential routes of infection. A wide variety of pathogenic *E. coli* have been identified by somatic O antigen serotyping, but the most common are Serotypes O1, O2, and O78. Many pathogenic *E. coli* isolates are not among the established serotypes and are classified as "untypeable" (Heller and Drabkin, 1977). The major disease syndromes in colibacillosis of poultry are yolk sac infection, respiratory disease complex (airsacculitis, perihepatitis, pericarditis), acute septicemia, salpingitis, peritonitis, synovitis, osteomyelitis, cellulitis, and enteric coligranuloma. Young birds with little resistance to infection will acutely die from septicemia, but older chickens are often resistant and survive the initial septicemic lesions (Nakamura *et al.*, 1985); however, *E. coli*-associated septicemic peritonitis in adult laying hens can cause significant mortality (personal observation). Coligranuloma is a rare condition that may be observed in adult chickens that are found dead and in poor physical condition. Gross findings in coligranuloma consist of firm, white to yellow nodules in the mesentery and wall of the intestine (Ashton, 1990). The nodules are granulomas from which *E. coli* may sometimes be isolated. Coligranuloma can be differentiated from mycobacteriosis by microscopy and bacterial culture.

ERYSIPELAS

Erysipelas is caused by *Erysipelothrix rhusiopathiae*, a slender, Gram-positive rod. The organism is hardy and can survive for years in contaminated soil. *Escherichia rhusiopathiae* is a zoonotic agent that can cause a skin rash (erysipeloid) and systemic disease in humans (Mutalib *et al.*, 1993), Erysipelas in poultry is diagnosed most often in turkeys over 12 wk of age and in broiler breeders, although other chickens, ducks, and game birds may be affected. The infection is transmitted between birds by contamination of breaks in the skin or mucous membrane through fighting, pecking, vaccine needles, etc. (Stuart, 1990). Affected birds may die rapidly and diarrhea may be observed. Gross necropsy reveals septicemic lesions with hemorrhages in the skin, muscles, epicardium of the heart, liver, and mesentery. Splenomegaly is often observed. Petechial hemorrhages may cover the serosa of the gizzard and intestine, and the luminal surface is congested and contains abundant mucus. Histologic examination reveals edema and hemorrhage of the lamina propria of villi, which contain fibrin thrombi and Gram-positive bacterial colonies in capillaries (Bickford *et al.*, 1978).

FOWL CHOLERA (PASTEURELLOSIS)

Fowl cholera is a severe systemic bacterial infection that affects chickens, turkeys, ducks and game birds. Death is usually caused by bacteremia and endotoxemia, especially in acute cases. Birds may die without clinical signs, but may be depressed with cyanosis and diarrhea. Birds that survive may develop chronic, localized infections in joints, wattles, footpads, sinuses, middle ear, bones, sternal bursae, and other tissues. *Pasteurella multocida* is the causative agent of fowl cholera. *Pasteurella multocida* is a small (less than 2 μm long), Gram-negative, nonmotile rod or coccobacillus that varies in virulence depending on the strain (Rhoades *et al.*, 1989). Sixteen distinct strains, which are serotyped according to the type of surface lipopolysaccharide, have been described (Brogden *et al.*, 1978). Unlike most bacterial diseases discussed in this review, but similar to mycobacteriosis and coligranuloma, fowl cholera is most common in adult or young adult birds. Gross lesions are septicemic in character and consist of hemorrhages and small necrotic foci scattered throughout the liver and other viscera. A necrofibrinous pneumonia is often present in turkeys (Glisson, 1996). Marked mucus accumulation and congestion of the small intestine may be observed. Histopathologic evaluation of the intestine may reveal only marked mucosal congestion (Rhoades, 1964); however, infiltrates of heterophils in the lamina propria and bacteria in small blood vessels of villi may sometimes be observed (personal observation).

MYCOBACTERIOSIS

Avian mycobacteriosis (avian tuberculosis), a chronic disease that affects a wide range of birds, is caused by *Mycobacterium avium*, of which Serovars 1 and 2 are most commonly isolated from birds. *Mycobacterium avium* is an acid-fast, nonmotile, aerobic rod that is long-lived in soil and dried feces (Thoen *et al*., 1981). Serovars of the *M. avium* complex have also caused disease in nonhuman primates, cattle, and swine (Thoen and Himes, 1986). *Mycobacterium avium* is primarily transmitted via the fecal-oral route and has zoonotic potential, based on the isolation of this agent from humans with acquired immunodeficiency syndrome (Falkinham, 1994). *Mycobacterium avium* has a long incubation period, and for that reason the disease is most commonly diagnosed in adult birds. Although avian mycobacteriosis is often a disease of captive birds (aviaries, zoos) is it relatively rare in commercial poultry in the United States, largely because of the limited life span (2 yr or less) of commercial poultry combined with effective management practices such as thorough cleaning and disinfection, in-door confinement, all-in all-out bird movement within houses, and raising of adult and young poultry in separate facilities (Jordan, 1990, Mutalib and Riddel, 1988). Avian mycobacteriosis in poultry is usually a chronic disease and is characterized by progressive loss of condition. Birds will continue to lose body weight despite having normal feed consumption. Gross necropsy often reveals the birds to be emaciated with atrophy of breast muscle and no internal body fat. The liver, spleen, intestines, and bone marrow may contain variably-sized white to grey, caseous nodules (tubercles). The tubercles can be scattered along the serosa of the entire intestinal tract, often penetrating the full thickness of the intestinal wall (Mutalib and Riddel, 1988). Ulceration of the intestinal wall by a tubercle permits *Mycobacterium* rods to spill into the gut lumen and hence contaminate the feces. Histologic evaluation of affected tissues is effective for making a rapid and definitive diagnosis. The tubercles consist of one or more discrete granulomas that often infiltrate all layers of the intestinal wall. The granulomas often have necrotic centers and are separated from normal tissue by fibrous connective tissue. Acid-fast bacilli can be observed in the necrotic center and in the cytoplasm of epithelioid macrophages that comprise the granuloma (Riddel, 1987).

SALMONELLOSIS

The genus *Salmonella* contains over 2,000 serotypes that have been classified by somatic (O), flagellar (H), and capsular (Vi) antigens. *Salmonella* is a Gram-negative, 2 to 3 μm long, nonsporulating rod that grows well on brilliant green and MacConkey agar. Isolation of the agent is often enhanced by incubating samples in selective enrichment media prior to culture (Quinn *et al*., 1994b). The intestinal tract is one of the most common sites of *Salmonella* colonization and transmission is often by the fecal-oral route. *Salmonella* infections can be systemic (bacteremia) and are at times accompanied by enteric lesions. Disruption of the normal intestinal microflora by antibiotic therapy or feed deprivation, and concomitant coccidial infection can increase the host's susceptibility to *Salmonella* infection (Hikasa *et al*., 1982; Porter and Holt, 1993). *Salmonella*-induced enteric lesions of poultry are often associated with three diseases: pullorum disease, fowl typhoid, and paratyphoid infections. In these diseases the enteric lesions are often prominent in the cecum, which is the best site for bacteriologic isolation of enteric *Salmonella.*

Pullorum disease and fowl typhoid can be described together because the history, clinical signs, lesions, and testing procedures are relatively similar for both. Pullorum disease (PD) is caused by *Salmonella pullorum* and fowl typhoid (FT) is caused by *Salmonella gallinarum*. These microorganisms cause systemic disease in a wide range of domestic poultry, including chickens, turkeys, ducks, and other gallinaceous birds. In the U.S. breeding flocks are serologically monitored for pullorum-typhoid through the National Poultry Improvement Plan and there is mandatory disease reporting and quarantine of affected flocks (Anonymous, 1994). Such programs have drastically reduced the incidence of pullorum-typhoid disease in the U.S. *Salmonella pullorum* and *S. gallinarum* were traditionally distinguished from paratyphoid *Salmonella* by virtue of the strong host-specificity for birds and the nonflagellated, nonmotile characteristics of the former; however, *S. pullorum* has been isolated from a variety of domestic mammals and recent work indicates that *S. pullorum* can exhibit motility and produce flagella under specific culture conditions (Holt and Chaubal, 1997). Pullorum disease is most lethal in young birds 3 wk old or less with minimal affects on adults, while the mortality observed in FT affects young birds and persists out to adulthood (Pomeroy and Nagaraja, 1991). Birds that recover from PD and FT can become chronic carriers and transmit the infection to progeny through the eggs (transovarian transmission). Infected progeny can readily spread the infection horizontally through contaminated feces. Birds hatched from infected eggs may die shortly after hatch and will appear depressed and weak with white, chalky material adhering to the vent (Hinshaw *et al*., 1926). Other signs, such as respiratory distress, lameness and blindness, may occur depending on the body systems that are infected. Adult birds from flocks with PD or FT generally show no clinical signs, but they may exhibit depression, ruffled feathers, pale and shrunken combs, decreased egg production, decreased fertility and decreased hatchability, especially in the case of FT (Pomeroy and Nagaraja, 1991). Gross lesions of PD and FT consist of peritonitis and variably-sized, white, raised caseous nodules or gray, necrotic foci in the lung, heart, spleen, liver, gizzard and kidney. Swollen joints, most

commonly the hock, will contain yellow, fibrinous fluid. Typhlitis, characterized by caseous, white to tan material in the lumen of the cecum, is the most common enteric lesion (Ashton, 1990). The typhlitis is characterized by marked mucosal and submucosal necrosis with the caseous cecal cores consisting of necrotic epithelium and debris, fibrin, heterophils, mononuclear cells, and bacterial colonies (Shivaprasad, 1997). An enteritis characterized by necrosis of villus tips and massive infiltration of the lamina propria by inflammatory cells has been described in adult chickens infected with PD (Riddel, 1987).

Most serotypes of *Salmonella* are placed in the paratyphoid group which is described as the *Salmonella* serotypes other than *S. pullorum*, *S. gallinarum*, and *S. arizona* (Ashton, 1990). The paratyphoid *Salmonella* are motile and infect a wide variety of host species. Examples of paratyphoid *Salmonella* include *S. typhimurium*, *S. enteritidis*, *S. montevideo*, and *S. heidelberg*. Paratyphoid infections have received significant attention over the last decade because poultry constitute a significant source of *Salmonella* that can induce foodborne illness in humans (St. Louis *et al.*, 1988). Although industry monitoring of paratyphoid *Salmonella* for purposes of food safety are emphasized, it is often forgotten that these agents can also pose a significant threat to poultry health. Paratyphoid infections can be transmitted by various routes including fecal-oral, fecal soiling of egg shells, and contaminated feed. Mice are a common reservoir of paratyphoid infection. *Salmonella enteritidis* (SE) has a distinction among the paratyphoid group by being egg-transmitted *via* deposition into the egg yolk prior to lay, resulting in the production of SE-contaminated eggs (Gast and Beard, 1990). Intestinal colonization by paratyphoid *Salmonella* usually results in invasion of the gut wall and dissemination to internal organs (Brown *et al.*, 1976). Paratyphoid infections in young birds often result in systemic infections with high mortality. Birds may die without showing signs or may appear depressed with closed eyes, ruffled feathers, fecal-soaked vent feathers, discolored yolk sacs, and profuse diarrhea. Adult birds appear to be rather resistant to paratyphoid infections and may harbor *Salmonella* in soft tissues without showing clinical signs (Brown *et al.*, 1976). Adult hens that are infected with SE will appear healthy and continue to shed SE in the feces (Holt and Porter, 1993). Recent studies showed that SE infection in White Leghorn hens could cause mild enteritis and typhlitis, and that feed deprivation exacerbated the degree of intestinal inflammation (Porter and Holt, 1993). Gross lesions of paratyphoid infection may include white necrotic foci scattered throughout a variety of internal organs with fibrinonecrotic and hemorrhagic exudate in the cecum (Figure 3). The cecal exudate can be observed in both salmonellosis and coccidiosis (*Eimeria tenella*). The histologic lesions of enteric paratyphoid infections are similar to those described for pullorum-typhoid, although the intestinal lesions will be mild in carrier birds that have minimal to no clinical signs.

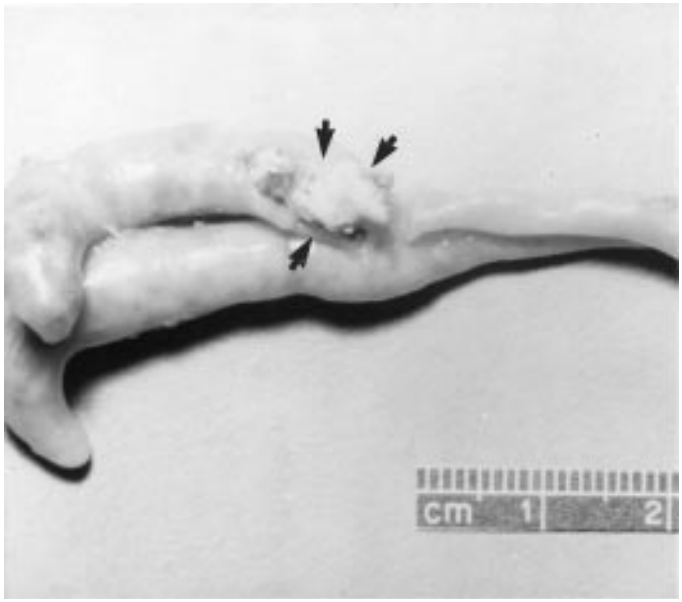

FIGURE 3. Cecum from a 5-wk-old pullet with fibrinonecrotic typhlitis. The lumen contains fibrinonecrotic exudate (arrows), which is commonly observed in both salmonellosis and coccidiosis (*Eimeria tenella*).

SPIROCHETOSIS

Spirochetes are slender, motile, flexible, unicellular, and helically coiled bacteria that range from 0.1 to 3.0 μm in width (Quinn *et al.*, 1994c). Both pathogenic and nonpathogenic spirochetes appear to inhabit the intestinal tract of birds (Buckles *et al.*, 1997). Two genera of spirochetes have been associated with enteric disease in fowl. The first form is a weakly β-hemolytic spirochete, representing a new species in the genus *Serpulina*, that has been associated with an infectious typhlitis in commercial laying hens and broiler chickens (Dwars *et al.*, 1992; Swayne *et al.*, 1992). The chicken-origin spirochetes shared similar surface antigens with and were morphologically similar to *Serpuline hyodysenteriae*, the causative agent of swine dysentery (Davelaar *et al.*, 1986). Recent evidence indicated that the chicken-origin spirochetes were primary pathogens for chickens and that the microorganisms were different from *S. hyodysenteriae* by being weakly β-hemolytic, having alpha-galactosidase, and not producing indole. In addition, the chicken-origin spirochetes were distinguished from *S. hyodysenteriae* by rRNA gene restriction pattern analysis and multilocus enzyme electrophoresis (Swayne *et al.*, 1995). The chicken-origin spirochetes have been isolated from the intestine of birds with retarded growth rate, diarrhea, feces-stained egg shells and vents, and decreased egg production (Dwars *et al.*, 1992; Griffiths *et al.*, 1987, Swayne *et al.*, 1992). A fecal-oral transmission of *Serpulina* is likely (Quinn *et al.*, 1994c), although it has

not been clearly established how long the chicken-origin spirochetes are viable outside the host. Gross lesions are often limited to the cecum which contains light brown, frothy fluid (Trampel *et al.*, 1994). Mild lesions consist of a dense, uniform layer of spirochetes covering the apical surface and brush border of cecal enterocytes. More severe cecal lesions consist of lymphocyte and plasma cell infiltrates in the lamina propria, variable degrees of epithelial cell necrosis and hyperplasia, crypts distended with sloughed epithelial cells and spirochetes, and infiltrates of a mixed population of inflammatory cells (Swayne *et al.*, 1992; Trampel *et al.*, 1994). Similar lesions have been described in a spirochete-associated necrotizing typhlocolitis of rheas; however, the relationship between the chicken-origin and rhea-origin spirochetes has not yet been established (Sagartz *et al.*, 1992). A second form of spirochetosis in poultry, which has been described in chickens, turkeys, and ducks, is a tick-transmitted (*Argas* sp.) bacteremic disease caused by *Borrelia anserina* (Quinn *et al.*, 1994c). The systemic infection is characterized by splenomegaly, hepatomegaly, and meningoencephalitis with mild enteric involvement. Bile-stained feces have been reported in field cases and green, mucoid diarrhea in chickens has been produced by an intramuscular injection of infected blood (Bandopadhyay and Vegad, 1984).

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