Poult enteritis complex

H.J. Barnes (1), J.S. Guy (2) & J.-P. Vaillancourt (1)

(1) Department of Farm Animal Health and Resource Management, College of Veterinary Medicine, North Carolina State University, 4700 Hillsborough Street, Raleigh, North Carolina 27606, United States of America
(2) Department of Microbiology, Pathology and Parasitology, College of Veterinary Medicine, North Carolina State University, 4700 Hillsborough Street, Raleigh, North Carolina 27606, United States of America

Summary
Poult enteritis complex (PEC) is a general term that encompasses the infectious intestinal diseases of young turkeys. Some diseases, such as coronavirus enteritis and stunting syndrome, are relatively well characterised, while others, such as transmissible viral enteritis, poult growth depression and poult enteritis mortality syndrome, remain ill-defined. All forms of PEC are multifactorial, transmissible and infectious. Salient clinical features include stunting and poor feed utilisation that result from enteritis. In the more severe forms, runting, immune dysfunction and mortality are reported. Gross and microscopic lesions of enteritis are present in all forms but tend to be non-specific. Other lesions may be present, depending on the agents involved. The basic pathogenesis involves the following:

a) alteration of the intestinal mucosa, generally by one or more viruses infecting enterocytes;
b) inflammation;
c) proliferation of secondary agents, usually bacteria.

Non-infectious factors interplay with infectious agents to modulate the course and severity of disease. Diarrhoea is believed to be primarily osmotic because of maldigestion and malabsorption, but may also have a secretory component. Transmission is primarily faecal-oral. No public health significance is recognised or suspected. Prevention is based on eliminating the infectious agents from contaminated premises and preventing introduction into flocks. This is accomplished by an effective cleaning, disinfection and biosecurity programme. All-in/all-out production or separate brooding and finishing units are helpful. Control may require regional co-ordination among all companies producing turkeys, especially if the production is highly concentrated, and a quarantine programme for more severe forms of PEC. No vaccines or specific measures for controlling the organisms involved in PEC are available. Treatment is supportive for the viral component, while antibiotics, especially those with efficacy against Gram positive bacteria, may help to reduce the impact of bacterial infections. Evidence suggests that PEC occurs wherever turkeys are raised commercially, but this is not well documented and distribution of the various organisms that have been associated with PEC is largely unknown. The disease causes enormous economic loss, mostly from failure of the turkey to reach its genetic potential.

Keywords
Description of the disease

Poult enteritis complex (PEC) is a general term for a group of multifactorial, transmissible, infectious diseases of young turkeys less than six weeks of age. These diseases are characterised by clinical signs of intestinal disease (enteritis), moderate to marked growth depression (stunting), retarded development (runting), impaired feed utilisation, and secondary nutritional deficiencies. Mortality is typically nil to low, but can exceed 10% in the more severe forms. Immune dysfunction generally occurs, which increases susceptibility of the flock to other infectious diseases.

PEC includes a number of described diseases and clinical intestinal infections of unknown cause. The characteristics evoke a production disease rather than a classical disease (Table I). The term is useful because of the limited knowledge about the causes and interactions of enteric pathogens and opportunists in young turkeys, limited availability of diagnostic procedures, lack of specific prevention methods, and the need for a generic approach to controlling all entities that comprise PEC.

Diseases encompassed by PEC include the following:
- coronaviral enteritis of turkeys (‘bluecomb’, infectious enteritis, transmissible enteritis) (69)
- cryptosporidiosis (41)
- malabsorption syndrome (80)
- poult malabsorption syndrome (73, 74, 75, 76)
- poult enteritis mortality syndrome (PEMS) (‘spiking mortality of turkeys’) (14)
- runting and stunting syndrome of turkeys (60)
- stunting syndrome (3, 4, 5, 10, 11)
- turkey viral enteritis (80).

Table I
Comparison between production and classical diseases

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Production disease</th>
<th>Classical disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cause</td>
<td>Complex, multifactorial; interactions between non-infectious and infectious factors</td>
<td>Simple, usually one, or less commonly, two agents; may be infectious or non-infectious</td>
</tr>
<tr>
<td>Clinical signs</td>
<td>Not present or mild and not considered significant</td>
<td>Usually present and readily recognised</td>
</tr>
<tr>
<td>Mortality</td>
<td>Typically nil to low</td>
<td>Variable, but usually present</td>
</tr>
<tr>
<td>Pathology</td>
<td>Not present or mild and non-specific</td>
<td>Typical lesions usually present</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Production below generic potential; inductive (few findings lead to generalities about flock)</td>
<td>Signs, lesions recognised, identification of cause; deductive (many findings lead to specific cause)</td>
</tr>
<tr>
<td>Treatment</td>
<td>Rarely possible, often too late, usually of little value</td>
<td>Often possible, moderate to high success</td>
</tr>
<tr>
<td>Prevention/control</td>
<td>Requires high biosecurity, excellent management, very good nutrition</td>
<td>Vaccines and medications often useful</td>
</tr>
<tr>
<td>Experimental reproduction</td>
<td>Difficult or impossible to reproduce and confirm experimentally, proven by Evans’ postulates (97)</td>
<td>Generally easy to reproduce experimentally, proven by Koch’s postulates</td>
</tr>
<tr>
<td>Methods of study</td>
<td>In flocks on farms, good records essential, emphasis on population medicine, epidemiology, etc.</td>
<td>Natural or experimental disease, emphasis on microbiology, pathology, toxicology, etc.</td>
</tr>
</tbody>
</table>

Several common names, such as ‘big bird – little bird syndrome’ have been used in the industry. Coccidiosis could also be included, but because of a well-recognized, specific disease, it will not be covered in this review (58). PEC is remarkably similar to a disease in chickens commonly referred to as running stunting syndrome (RSS) (65). The relationship between PEC in turkeys and RSS in chickens has yet to be completely clarified, but most evidence suggests these are two distinct syndromes (23, 90).

No specific cause of PEC has been identified. The basic pathogenesis involves damage to the intestinal mucosa, usually by one or more viruses, a host inflammatory response that may intensify the mucosal damage, and subsequent proliferation and possible colonisation by intestinal bacteria and protozoa. Lesions in the intestine are best described as catarhal enteritis (53). Impaired immunity is indicated by atrophy of lymphoid organs, fungal infections, especially crop mycosis caused by Candida albicans, and systemic bacterial infections such as colisepticcaemia. Non-infectious factors such as nutrition, environment and management can have a marked influence on the course and severity of the disease.

History

In the late 1940s and early 1950s, an acute diarrhoeal disease with high morbidity, and often high mortality, especially in young turkeys, occurred in flocks in several areas of the United States of America (USA). The disease was named ‘mud fever’ or ‘bluecomb’ because of the similarity to a disease in chickens. During the following two decades, bluecomb was a significant cause of economic loss in the turkey industry in Minnesota and the subject of intense research. The causative agent, a coronavirus, was eventually isolated from affected flocks and used to reproduce the disease, which prompted the name to be changed to ‘coronaviral enteritis of turkeys’.
However, while experimental infections reproduced the stunting, the mortality seen in natural infections was not typically reproduced (1). Diagnostic tests were developed and used to identify and eliminate all affected flocks in Minnesota, which was accomplished in 1976 (69). Subsequent outbreaks occurred in Quebec in the mid- to late 1980s (24), Indiana in the mid-1990s, and Virginia and North Carolina in the late 1990s (20). Sporadic cases involving only a few flocks and with limited spread as well as occasional outbreaks in which numerous flocks are affected and the disease spreads rapidly among farms continue to occur in some turkey producing areas of the USA.

Soon after the identification of coronaviral enteritis, the existence of other intestinal diseases in young turkeys became apparent, although these tended to be less severe than coronaviral enteritis. In the late 1970s and 1980s, a number of enteric viruses were identified from poults in the USA (40, 82, 84), United Kingdom (UK) (60, 63), France (9) and Germany (86) that were experiencing a RSS clinically similar to a disease in chickens. In addition to enteric viruses, Cryptosporidia were found to be associated with intestinal disease of poults (41).

In the 1990s, rotaviruses were associated with affected flocks in Israel (104). In the USA, a malabsorption syndrome of turkeys which was associated with skeletal abnormalities was identified in turkeys in the south-east of the country (73, 74, 75, 76), and a stunting syndrome was found to affect turkey flocks in the north-central USA (3, 4, 5, 10, 11). Cochlosoma was another protozoan linked with intestinal disease in young turkeys in California (21).

In 1991, a particularly virulent enteric disease characterised by a sharp peak of mortality emerged in an area of dense turkey production in western North Carolina. The disease was initially named 'spiking mortality of turkeys' because of the mortality pattern in affected flocks (Fig. 1), but was later termed 'PEMS', following the discovery of a milder clinical form of the disease in 1994 (14). In addition to high mortality, affected birds show immune dysfunction (51, 52, 78) and a variety of physiological abnormalities, including reduced body temperatures, reduced energy metabolism and hypothyroidism (28).

Poult enteritis mortality syndrome is readily reproduced by inoculating young poults with faeces or intestinal homogenates, or by placing susceptible birds in contact with litter from an affected flock, indicating that the most common mode of transmission is faecal-oral (17, 23). A number of viruses, including turkey coronavirus (TCV) and unidentified small round viruses, along with certain strains of Escherichia coli have been associated with the disease, but no specific agent has been present in all cases or has reproduced the disease experimentally (29, 30, 46, 48, 87, 106). Once a flock is affected with the disease, other conditions, such as colibacillosis, salmonellosis, rickets, and protozoal enterotyphlitis, occur more frequently.

The prevalence of PEMS increased from 1991 to 1996, when occurrence and severity reached a peak. Several flocks were destroyed after mortality exceeded 50%. One flock that was not destroyed experienced a total mortality of 96%. Since 1996, the number and severity of outbreaks have declined and no flocks in North Carolina have been destroyed.
By comparing hatchmates raised at the College of Veterinary Medicine of the North Carolina State University (NCSU) with those produced on commercial farms since the mid-1980s, a type of PEC has been identified that has been named 'poul growth depression'. A formal description of the disease has yet to be published. The disease is a mild intestinal disorder that causes a reduced growth rate between two and four weeks of age in most commercial flocks. Productivity lost during this period is never fully regained, so that affected flocks are typically 10%-15% lighter at processing or require between ten and fourteen days longer to reach market weight. Analysis of flock records from integrated companies across the USA suggests that this form of PEC is so ubiquitous that the disease is generally not recognised because of its insidious nature. The disease has been experimentally reproduced by exposing seven-day-old poults to an intestinal homogenate from affected birds; the disease developed in the first two flocks placed on new farms (H.J. Barnes, J.S. Guy and G.M. Elias, unpublished data).

Production records from a flock studied at the NCSU that did not experience poul growth depression, together with data regarding ten commercial flocks marketed at the same time, are summarised in Table II. Recent average data obtained from similar flocks produced throughout the USA are included for comparison (35). On average, the unaffected flock achieved a 17.7% better growth rate, reached market weight 13.5 days earlier, and each pound of turkey cost US$0.058 less per kg of feed to produce (US$0.026/lb). Note that growth and feed efficiency of the ten commercial flocks are comparable to average production in the USA and would be considered normal.

### Clinical signs

Flocks are typically affected between ten and twenty-eight days of age. The first indication of PEC is often hyperactivity with excessive vocalisation. Birds are described as being noisy and ‘marching’ as they move continuously along feed or water lines or the walls of the house. Feed consumption drops sharply; water consumption typically increases initially and then decreases in parallel with reduced food intake. During the next twelve to twenty-four hours, the flock becomes quiet and birds huddle together near heat sources. Birds consume litter and are seen pecking at feed and sorting out larger particles. Some flocks completely refuse to eat ('feed refusal'). In most instances the feed is satisfactory, moving the feed to another flock, testing the feed under controlled conditions, or sometimes just remixing and redistributing the feed to the affected flock results in consumption. Most flocks develop diarrhoea within 24 h. Diarrhoea primarily results from the osmotic effects of undigested, unabsorbed feed. Litter rapidly deteriorates, followed by declining air quality, and birds become progressively more soiled. Insect larvae multiply in wet litter, especially when ambient temperatures are high. Many birds will have faecal staining of vent and abdominal feathers and/or pasting around the vent with faeces and urates. Size variation among birds in the flock can be observed within a few days, and lack of uniformity is clearly evident one week after onset of clinical signs. Approximately 60%-70% of the stunting can be directly attributed to decreased feed intake. Feathers, especially those of the tail, become frayed and show segmental dysplasia ('stress bars'). The feathers may break off completely at these weakened sites, leaving the bird with partial or no tail feathers, or incompletely break.
Feathers develop segmental dysplasia ('stress bars'), either breaking off completely or remaining partially attached. The latter produces a 'helicopter' bird, as seen in this poult with experimental poult enteritis mortality syndrome. Clinical signs decrease within seven to ten days after the onset of disease but the lack of uniformity continues to increase and the appearance of the birds remains poor for the duration of the life of the flock (Fig. 6). Management intervention can shorten the duration of clinical disease and the impact of PEC on the flock. Secondary nutritional deficiencies such as osteoporosis ('brittle bones'), rickets, and/or encephalomalacia may occur (57, 100). Leg problems are greater at market-age in affected flocks (76). Susceptibility to coccidiosis in flocks with PEC may be increased if the disease is being controlled with a coccidiostat, due to decreased feed consumption. The lost growth potential of the flock will not be regained, although the rate of growth will eventually return to normal for most birds. Flocks affected by PEC have lighter weights, lower average daily gains, poorer feed conversion ratios, and a decreased percentage of white meat with a higher percentage of viscera and bone when marketed. This occurs because of the way in which nutrients are distributed to supply and demand organs and maximal growth rates of commercial turkeys. During periods of decreased nutrient intake, such as those which occur in flocks affected with PEC,
Fig. 6
Flock of older birds (10-11 weeks of age) with typical signs of poult enteritis complex (western North Carolina, 1996)
Note the rough overall appearance, poor feathering, faecal staining and lack of uniformity
Photo: courtesy of Mr T. Knapp

the limited nutrients are preferentially provided to the supply organs (viscera, support tissues) instead of demand organs (muscle, especially white muscle). Restoration of normal nutrition after recovery does not result in compensation for the lost production of muscle mass during this period because there is little or no ability to exceed the maximal growth rate of the commercial turkey. The net result is a decreased percentage of meat at processing, which is directly proportional to the severity of the episode of PEC in the flock. Lack of uniformity in affected flocks may create problems during processing and in fulfilling marketing contracts.

Pathology
Mildly affected poults may demonstrate few external signs, apart from stunting, dirty feathers, and/or excrement around the vent. Dehydration (skin folds along shanks, dark congested tissues, dry skin), weight loss, poor feathering (notched, frayed, brittle or broken, stress marks), faecal staining of feathers, and faeces and/or urates on feathers and around the vent are seen in more severely affected birds. Watery, yellow-brown, flocculent liquid may drip from the vent, especially when the carcass is handled, and the abdomen often appears distended (Fig. 7).

Internal lesions are also variable according to the severity of the disease. However, intestinal changes consisting of pale, thin-walled intestines distended with fluid and occasional gas or mucous casts, and caeca filled with watery brown fluid and gas are observed consistently, regardless of severity (Figs 8 and 9). Multiple, fluid-filled, saccular distensions of the small intestine alternating with contracted areas occur in more severely affected birds. The gastrointestinal tract contains little or no ingesta, but litter is found in the ventriculus, sometimes to the point of impaction. Soft brown to orange casts are occasionally present in the intestinal lumen, and rarely, caecal cores. The cloaca is often distended and a firmer mass of faeces and urates may be found just internal to the vent. Small erosions or ulcers of the ventriculus near the entrance of the proventriculus are common in both affected and unaffected young poults and should not be identified as a lesion of PEC.
Intestine of an eleven-day-old turkey poult affected by experimental poult enteritis complex, of unspecified type, day four post exposure

In more severely affected birds, a moderate to marked muscle atrophy occurs that is most apparent around the knee joint and along the keel (Fig. 10). If the bird is dehydrated, the muscles will be dry and dark. Fat stores are depleted but do not show prominent serous atrophy. Bones may be normal, osteoporotic (thin, brittle and easily broken when manipulated), or rachitic (soft and flexible with widened growth plates and easily cut). Kidneys are usually mildly to moderately swollen and urates may be visible in the ureters. In severely stunted birds, adrenal glands are prominent and lymphoid organs, especially the thymus and bursa of Fabricius, are atrophic (Fig. 11). Poults with coronavirus infection occasionally have caseous masses filling the lumen of the bursa of Fabricius. Abscesses of the yolk stalk remnant (vitelline or Meckel's diverticulum) occur more frequently in poults with PEC. Crop mycosis may be identified as a secondary complication related to immune dysfunction.

Microscopic changes are often not specific or even characteristic for different diseases within the scope of PEC. Intestinal lesions include villous changes (atrophy, blunting and fusion), alteration in the number of goblet cells, crypt epithelial hyperplasia, hyperaemia, protein loss and infiltration of the lamina propria with mixed inflammatory cells, especially macrophages (Fig. 12). Lesions tend to be less evident in experimental infections with viral agents alone. The lower small intestine and caecum are usually more severely affected than the duodenum. Heterophils are more numerous when bacterial infection is present. Free inflammatory cells, sloughed epithelial cells and proteinaceous exudate can accumulate in the intestinal lumen and support proliferation of intraluminal bacteria. Pancreatic acinar cells may undergo vacuolar degeneration and either be depleted or engorged with zymogen. The latter typically are located around islets. Changes in the liver consist of Kupffer cell hyperplasia, hepatocellular atrophy, and occasionally foci of necrosis and/or mononuclear cells.

Specific changes in the intestine include large, basophilic inclusions within the nuclei of enterocytes produced by Group 1 adenoviruses, adhering bacteria (Gram positive cocci, Gram positive or negative bacilli) covering villous surfaces, and attaching effacing lesions caused by enteropathogenic E. coli. Clostridia cause focal or diffuse coagulation necrosis and lysis of the mucosal surface. Lesions can be distinguished from autolysis by the presence of numerous large Gram positive bacilli and an inflammatory
Fig. 12
Jejunum from a three-week-old poult with poult enteritis complex
Lesions indicate acute catarrhal enteritis but are not specific for a causative agent. Increased goblet cells on the villous tips have been described as a lesion of turkey coronavirus enteritis, but infection with coronavirus could not be confirmed in this flock. x159; stain: haematoxylin and eosin.

In other tissues of severely affected birds, lymphoid depletion may occur in the spleen, bursa of Fabricius and thymus. Infection of the bursa of Fabricius by coronavirus causes degeneration, necrosis and inflammation of the epithelium, and is associated with lymphoid depletion of follicles (Fig. 13). Occasionally, fibrinoheterophilic, caseous exudate will fill the bursal lumen causing a ‘bursal core’ (Fig. 14) (49). Evidence of rickets may be found in bones, especially the proximal tibiotarsus. Cortical cell hyperplasia in the adrenal glands and histological indications of thyroid inactivity are evident in endocrine organs. Changes in the kidney are consistent with dehydration. Bacterial colonies are frequently numerous in yolk sac abscesses. *Candida* is present within the hyperplastic epithelium of the crop of birds with crop mycosis.

**Incidence and distribution**
Anecdotal evidence suggests that PEC has a world-wide distribution. However, the actual occurrence of intestinal diseases of young turkeys, and the infectious agents that are associated with them, remain largely undocumented. This may be due in part to the lack of diagnostic reagents and testing, the limited turkey production in many areas of the

Fig. 13
Bursa of Fabricius from a turkey with coronavirus enteritis
Degeneration, necrosis and sloughing of epithelial cells are visible together with an associated acute heterophilic inflammation. The interstitium is infiltrated by mixed inflammatory cells. Lymphoid follicles are depleted and a high level of apoptosis has occurred. x62; stain: haematoxylin and eosin.

Fig. 14
Bursal core in a four-week-old turkey from a flock experiencing poult enteritis complex
Although this flock was not tested for turkey coronavirus, the lesion is identical to that seen in experimentally infected poults.
world, the mild nature of most forms of PEC, or little awareness of the diseases that constitute PEC.

Significance
Although the relationships among the various disease entities included within PEC are largely unknown, infectious intestinal disease of young turkeys is clearly very common and a significant cause of economic loss. Calculations indicate that flocks infected with TCV have increased production costs of approximately US$0.044/kg-US$0.11/kg (2 cents/lb-5 cents/lb) (55, 83). Poult enteritis mortality syndrome devastated the turkey industry in North Carolina, causing an estimated loss of US$161 million between 1991 and 1997. Annual production of turkeys in the endemic area and State-wide substantially decreased during this period (Table III).

Although outbreaks with high mortality are dramatic and certainly costly, such episodes occur infrequently. Far greater loss results from the common, widespread occurrence of the mild forms of PEC, such as poult growth depression, which attract little attention (Table II). Based on a 10%-15% loss due to poult growth depression throughout the turkey industry of the USA, estimated potential losses to the USA turkey industry from poult growth depression would be between US$300 and US$400 million annually.

Research is continuing to identify new agents, define interactions between previously known agents, and develop diagnostic methods to determine what organisms are associated with the different forms of enteric disease. While progress is being made, the effort is small compared to the magnitude of the problem. The ability of a turkey to achieve its genetic potential with respect to growth and feed utilisation correlates directly with the health of the intestinal tract.

### Table III
Impact of poult enteritis mortality syndrome on turkey production in Union County, North Carolina, United States of America (USA) (endemic area), and State-wide between 1992 and 1997 (North Carolina Agricultural Statistics)

<table>
<thead>
<tr>
<th>Production variable</th>
<th>Union County (North Carolina)</th>
<th>State-wide (North Carolina)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Turkeys produced in 1992 (millions)</td>
<td>14.4</td>
<td>62</td>
</tr>
<tr>
<td>Turkeys produced in 1997 (millions)</td>
<td>5.4</td>
<td>53.5</td>
</tr>
<tr>
<td>Difference (millions)</td>
<td>-9</td>
<td>-8.5</td>
</tr>
<tr>
<td>Difference (%)</td>
<td>-62.5</td>
<td>-13.7</td>
</tr>
<tr>
<td>Proportion of total production in North Carolina in 1992 (%)</td>
<td>23.2</td>
<td>-</td>
</tr>
<tr>
<td>Proportion of total production in North Carolina in 1997 (%)</td>
<td>10.1</td>
<td>-</td>
</tr>
<tr>
<td>Proportion of total production in the USA in 1992 (%)</td>
<td>-</td>
<td>21.5</td>
</tr>
<tr>
<td>Proportion of total production in the USA in 1997 (%)</td>
<td>-</td>
<td>17.8</td>
</tr>
</tbody>
</table>

### Description of the aetiological agents
Several different viruses, bacteria and protozoa have been associated with PEC (Table IV). Those of greatest importance appear to be rotaviruses, TCV, turkey enterovirus, astrovirus, Salmonella spp., E. coli, and Cryptosporidium spp. Recently, unclassified, small, round viruses have been isolated from poult with PEMS (87, 106). Reviews have been published on viruses that cause enteritis in poultry (43) and turkeys (85).

#### Rotavirus
Rotaviruses are classified as a separate genus within the family Reoviridae (33). Rotaviruses are non-enveloped, spherical, and have a diameter of approximately 70 nm. Intact viruses consist of two icosahedral capsid shells (approximately 50 nm and 70 nm in diameter); the particles have a distinctive 'wheel-like' appearance by negative-stain electron microscopy.
Turkey Coronavirus replication occurs in the epithelial lining of the inner capsid that radiate towards the rim. The genome is comprised of eleven linear segments of double-stranded (ds) ribonucleic acid (RNA) with a total molecular weight of approximately $12 \times 10^6$ (33).

Classification of avian rotaviruses has been based primarily on immunofluorescent antibody (FA) procedures and polyacrylamide gel electrophoresis (PAGE) analyses of ds RNA segments. Avian rotaviruses that cross-react by FA with antisera prepared against group A mammalian rotaviruses are classified as group A avian rotaviruses (62). Rotaviruses that lack the group A antigen are referred to as 'atypical' rotaviruses. Two antigenically distinct 'atypical' avian rotaviruses have been identified in turkeys; one of these has been classified as group D, but the other one remains unclassified. Polyacrylamide gel electrophoresis analysis of ds RNA is also useful for classification of avian rotaviruses; electrophoretic migration of RNA segments is an indicator of serogroup classification and RNA profiles may be useful in epidemiological studies (62).

Rotavirus replication occurs primarily in the small intestines, in differentiated, mature epithelium lining the upper portions of intestinal villi. Based on studies with mammalian rotaviruses, diarrhea caused by avian rotaviruses probably occurs due to destruction of mature villous epithelium and replacement by immature epithelium from crypts. Immature, undifferentiated cells which replace cells destroyed by the virus lack disaccharidases and have impaired absorptive ability. Diarrhea is likely to be a result of the effects of both malabsorption and maldigestion (67).

In vitro propagation has been possible with turkey group A rotaviruses, but not atypical rotaviruses. Cell culture propagation of group A rotaviruses has been accomplished in a variety of cell types, including primary chicken kidney cells, primary chicken embryo liver cells and a continuous line of rhesus monkey kidney cells (MA104) (62). Serial propagation in cell culture usually requires trypsin treatment of the inoculum.

**Coronavirus**

Turkey coronavirus is a member of the family Coronaviridae. Members of the genus Coronavirus are RNA-containing viruses that infect a wide variety of avian and mammalian species (103). The viruses are characterised on the basis of their distinctive morphology – pleomorphic, enveloped particles with diameters of 60 nm-220 nm, with long (12 nm-24 nm), widely spaced, petal-shaped surface projections (103). A close genomic relationship between infectious bronchitis virus and TCV has been identified (16).

Turkey coronavirus replication occurs in the epithelial lining of the bursa of Fabricius and upper portions of intestinal villi. Diarrhea in TCV infections, like rotavirus infections, is believed to result from malabsorption and maldigestion.

The turkey was considered to be the only natural host for TCV, but recently chickens have been infected with the virus. Experimental inoculation of young chickens resulted in intestinal infection, as determined by immunohistochemistry and virus isolation, but the chickens did not develop clinical disease (47). In addition to TCV, evidence suggests that pouls may also be susceptible to infection with bovine coronavirus (25, 54), and preliminary characterisation suggests that the virus causing stunting syndrome is probably a member of the family Coronaviridae (5).

Turkey coronavirus may be propagated in embryonated chicken and turkey eggs by inoculation of the amniotic cavity (69). Virus replication in inoculated embryos is restricted to intestinal epithelium and the epithelium of the bursa of Fabricius. Attempts to propagate the virus in cell culture generally have not been successful.

**Enteroavirus**

Since the discovery of entero-virus-like viral particles in the faeces of pouls with diarrhoea in the UK in 1979 (66), several viruses resembling enteroviruses, referred to as entero-virus-like viruses (ELV) or pseudopicornaviruses, have been suggested as causes of PEC (66). Enteroviruses comprise one of four genera within the family Picornaviridae (6). Picornaviridae are non-enveloped, icosahedral viruses which range from 22 nm to 30 nm in diameter. Virions lack an obvious surface structure and no surface projections are visible. These viruses possess a genome comprised of single-stranded RNA of approximately 7.5 kilobases (2.5 x 10^6 molecular weight) (6). Genera within the family Picornaviridae are distinguished by their sensitivity to acid, buoyant density of the virion in CsCl, and clinical manifestations in the affected host (6). Enteroviruses are stable at acid pH, have a density of 1.33 g/ml in CsCl and replicate preferentially in the intestinal tract. Turkey ELVs have been classified on the basis of size, morphology, cytoplasmic replication in enterocytes and resistance to acid pH.

Turkey ELVs have been demonstrated to replicate preferentially in the jejenum and ileum (50). Virus replication occurs principally in villous epithelium located halfway between the tip and base of the villus.

Turkey ELVs may be propagated in embryonated turkey eggs (44). Laboratory propagation is accomplished by amniotic inoculation of embryonated turkey eggs; virus replication occurs in embryo intestines.

**Astrovirus**

Members of the genus Astrovirus are small, roughly spherical viruses, 28 nm to 31 nm in diameter (79). The viruses possess a characteristic morphological feature, namely: a five-
six-pointed star which covers the surface of approximately 10% of virus particles. The biochemical structure of avian astroviruses is largely unknown, as these viruses have not been propagated in vitro.

**Salmonella**

*Salmonella* are Gram negative, non-spore-forming bacteria which are members of the family Enterobacteriaceae. Motile, non-host-adapted serotypes of *Salmonella* are referred to as paratyphoid *Salmonella*. Of the serotypes of *Salmonella* identified in turkeys with PEC, paratyphoid serotypes are most common. Serotypes are identified using the Kaufmann-White method, based on antigenic differences identified in both somatic and flagellar antigens (39).

Paratyphoid salmonella have an extensive host range. The bacteria can be isolated from a wide variety of species including humans and other mammals, avian species and reptiles.

*Salmonella* are readily cultivated in the laboratory as nutritional and growth requirements are simple (102). The bacteria are facultatively anaerobic and grow well under both aerobic and anaerobic conditions. *Salmonella* can be cultured on a variety of simple culture media. Optimal growth is at 37°C and pH 7, but can occur over a wide range of temperatures (5°C-45°C) and pH (pH 4-9).

**Escherichia coli**

*Escherichia coli* are Gram negative, non-spore-forming bacteria which also are members of the family Enterobacteriaceae. Identification of *E. coli* is based on biochemical characteristics, serotyping and virulence properties. Serotyping is determined by differences in O (somatic), H (flagellar), and K (capsular) antigens. Virulence properties which allow categorisation of strains of *E. coli* include elaboration of enterotoxins and shiga toxins, and the presence of certain genes, such as the *E. coli* attaching and effacing (*eae*) gene (70).

*Escherichia coli* have not generally been considered to be intestinal pathogens of poultry; however, enteropathogenic strains of *E. coli* have recently been associated with PEC (48). Enteropathogenic strains of *E. coli* are identified by characteristic attaching and effacing lesions in intestinal tissues (Fig. 15), absence of enterotoxin and shiga toxin production, lack of cell invasiveness, and presence of the *eae* gene (70).

*Escherichia coli* have a wide host range including mammalian, avian, reptilian and amphibian species. Enteropathogenic *E. coli* have been identified in a variety of mammalian and avian species, including turkeys.

*Escherichia coli* are readily cultivated in the laboratory using ordinary culture media. *Escherichia coli*, like *Salmonella* spp., are facultatively anaerobic and grow well under both aerobic and anaerobic conditions. *Escherichia coli* have simple nutritional and growth requirements.

**Cryptosporidium**

Cryptosporidia are small coccidian parasites that replicate within the microvilli of the intestinal tracts of vertebrates. Two species have been identified as causes of intestinal infection in turkeys, namely: *Cryptosporidium meleagridis* and *C. baileyi*. However, *C. meleagridis* is probably the predominant species associated with PEC, as *C. baileyi* infection is restricted in turkeys to the cloaca, bursa of Fabricius and respiratory system (22).

Replication of *C. meleagridis* in vivo occurs primarily in the middle and lower small intestine of turkeys. Parasites are found within the brush border of intestinal epithelium (Fig. 16). Attempts to propagate cryptosporidia in vivo have not been successful.

**Epidemiology**

The occurrence of PEC is influenced by environmental conditions and farm location. In general, PEC tends to be more evident during the warmer seasons of the year. This is
Fig. 16
Ileum from a poult experimentally infected with *Cryptosporidium meleagridis* in faeces from turkeys with poult enteritis mortality syndrome, three days post-exposure
Villous atrophy, fusion, crypt hyperplasia and interstitial infiltration of inflammatory cells are visible. The numerous small spherical organisms along the surface of the villus are cryptosporidia. x159; stain: haematoxylin and eosin

particularly true for the severe forms such as PEMS; most cases in the south-eastern USA are reported between May and September, with only sporadic outbreaks at other times of the year. Possible explanations for this pattern include more rapid pathogen multiplication at higher temperatures, leading to increased contamination of the environment, greater numbers and activity of possible vectors (rodents, birds, insects, etc.), and decreased resistance of the turkeys because of heat stress.

Recent outbreaks of coronaviral enteritis in North Carolina have occurred from October through to December. The last quarter of 1999 witnessed a dramatic increase in TCV infections following hurricanes Floyd and Irene in eastern North Carolina. Severe flooding probably disrupted normal biosecurity and movement of vehicles among farms. In addition, spread of micro-organisms was facilitated by the adverse environmental conditions. Milder forms of PEC appear to occur throughout the year, although this has not been investigated adequately.

The highest incidence of PEC is reported in areas of dense turkey production where farms are located less than a mile from each other, especially if more than one production company is involved. The reasons for this are unknown, but possible explanations include the following:

- a high level of microbial contamination in the environment
- the ease by which infectious agents can spread from one property to another
- the existence of multiple types of enteric pathogens and opportunists
- d) the density and movement of vectors
e) the ease of access by infectious agents to susceptible populations.

In an investigation of PEMS involving fifty-two farms in North Carolina, hatchery of origin, removal of used litter by a contractor, and ineffective rodent control measures were determined to be associated with affected flocks. However, no strong evidence was available to support vertical transmission, and in contrast to experimental studies (26), field research on pests (flies, darkling beetles and rodents) failed to demonstrate a clear relationship between the disease and these potential vectors/carriers. Factors not associated with the disease included the following: breed, integrated company with whom the growers had a contract, proximity to cattle or hogs, distance of turkey houses from roads or trees, and method of dead bird disposal. Mortality associated with PEMS only occurred during the brooding period. Excess mortality after brooding associated with enteric diseases was found to be caused by coronavirus infection. Although anecdotal reports suggest that PEMS is more severe in females than in males, this observation is probably attributable to differences in the way hen and tom flocks are managed, as experimental studies have not demonstrated a sex difference in susceptibility.

Diagnostic methods

Diagnosis of PEC requires monitoring, analysis of growth and performance during brooding, clinical evaluation, collection of diagnostic samples, necropsy of affected birds, and isolation and identification of enteric pathogens.

Often the most common, mild forms of PEC can only be recognised by comparing flock records with appropriate standards for growth, uniformity and feed utilisation. Unfortunately, 'gold standards' do not exist. As the milder forms of PEC occur so frequently, standards or averages calculated by breeder organisations, or within an integrated company, typically include flocks that have experienced some degree of PEC. Recently published industry averages fall well below the genetic potential of the turkey (35). Unless a growth standard can be developed in a facility where enteric disease can be prevented, the most appropriate standard to use is the top performing flock within the company. This represents the maximum production that can be expected given the same management programme, nutrition and genetic stock. As a minimum, flocks should be weighed and evaluated for uniformity, and estimated feed use should be determined either at the end or near the end of the brooding period. Preferably, flocks should be weighed weekly, in which case affected flocks can be readily identified by a decreased growth rate during weeks two to six (Fig. 17).
Clinicopathological findings

In monitoring flocks for clinical evidence of enteric disease, indicators other than mortality need to be established as this is rarely elevated in most cases of PEC. When a flock with clinical signs is identified, a visit to the farm is necessary to assess the environment, feed and management, and to collect samples. The most accurate information will be obtained early in the course of the disease. Six to ten representative affected birds should be selected for necropsy. If mortality is occurring, dead birds should be examined in addition to the live ones selected for necropsy. Six to ten serum samples, a composite sample of droppings, a water sample, and a feed sample from the feeders need to be collected. Samples of serum and droppings can be taken from any birds in the flock or from those selected for necropsy. The flock should be revisited approximately three weeks later to collect a set of convalescent serum samples. If evaluation of the birds is not possible when clinical signs are present, or if the onset of the disease is uncertain, normal two- to three-week-old turkeys can be placed into the flock as sentinels and sampled six to eight days later.

The following is a necropsy and sampling protocol which has been developed by the authors for investigating outbreaks of PEC. Birds must be killed individually and necroscopy performed immediately. Critical microscopic evaluation of the intestinal mucosal surface is only possible when autolysis has been minimised. Similarly, artifact resulting from exposure of the tissues to water, scraping, or rough handling must be avoided. After the bird has been killed, the investigator should open the abdomen, isolate the duodenum, and remove a cross-section of descending and ascending duodenum including pancreas. The duodenal segments should be opened and placed into fixative (10% buffered neutral formalin is adequate for most evaluations).

Any ingesta present in a 2-cm segment of jejunum at Meckel’s diverticulum should be gently expressed, the segment should then be removed, and placed into fixative. Fixation will be adequate without the intestine being opened if the intestine is empty or contains fluid only. An adjacent area of jejunum is then opened, the mucosa blotted on absorbent towelling to remove any adhering ingesta, and touch impressions are made on one half of two microscopic slides. The slides can be briefly air-dried, and blotted on towelling to remove any thick areas, and then air drying of the slides completed. An area of the mucosa should be scraped and placed into the centre of a few drops of warm physiological saline. A coverslip is placed on the mucosal scraping and gently pressed to spread the sample. The saline and the sample should not be mixed together before placing the coverslip.

For immunofluorescent procedures and virus isolation, two 2-cm segments of jejunum should be removed from the opposite, unsampled, cut end and snap frozen in liquid nitrogen or on dry ice. Alternatively, a 0.5-cm ring can be frozen directly in cryostatic mounting medium for immunofluorescence.
A 2-cm segment of ileum and adjacent proximal caeca should be placed into fixative. Adjacent pieces of ileum can be frozen for virus isolation and immunofluorescence. Another segment of the ileum is opened and mucosal touch impressions are made on the other half of the microscopic slides. A wet smear from the ileum or caecum is not necessary. The remaining intestinal segments should be opened to examine for lesions, and then those from Meckel's diverticulum to the cloaca pooled, frozen and stored at -70°C in case experimental studies are required. Samples collected in the field should be kept refrigerated, but not frozen, and submitted to a diagnostic laboratory as rapidly as possible.

Samples of lymphoid organs, especially the bursa of Fabricius, liver, and any gross lesions, should also be collected and placed into fixative. If turkey coronavirus is suspected, one half of the bursa should be frozen for immunofluorescence. Weighing of the birds and lymphoid organs may be desirable to determine their relative weights. Location and removal of all of the thymus is not necessary. The authors have found that the left lobes proximal to the thyroid gland are readily accessible and constitute approximately 58% of total thymic tissue.

Jejunal wet smears should be kept warm and evaluated as soon as possible. As the saline cools, motility of protozoa will diminish or cease, rendering detection more difficult. The interface between the mucosal scraping and saline should be examined for motile protozoa under the low power lens of a microscope. By not mixing the saline and mucosal scraping together, the organisms can be easily observed as they swim from the sample into the surrounding fluid. If motile protozoa are seen, their identity can be confirmed by using the high power lens. Cochlosoma moves at a moderate pace with seemingly random spinning, twirling, rotational movements. The large ventral sucker is clearly evident giving the organism the appearance of a snail shell. Spiromonas has a rapid, darting, directed movement. The appearance is suggestive of a mouse with a pointed nose and trailing tail. Trichomonads display a jerking motion because of their undulating membranes, but show little movement, often appearing to have the distal ends fixed on pieces of debris. The undulating membrane suggests fingers waving within a sock or thin mitten. Detection of protozoa in the mid-jejunum, especially if numerous, is considered to be more significant than detection in the lower gut or in very low numbers. Some protozoa are normally found in the caecum. In addition, small, rapidly darting spiral bacteria, typical of Campylobacter, may be identified. In a well-prepared wet smear, the mucosal sample will consist of a confluent layer of the sheared tips of villi. Identification of coccidia, cryptosporidia and long-segmented filamentous bacteria in the mucosal sample may be possible.

One of the mucosal impression smears should be stained with Gram's stain and the other with a cellular stain such as Wright's, Giemsa, or a combination of the two. High numbers of large Gram positive bacilli are indicative of clostridial proliferation and possible necrotic enteritis. Protozoa may be seen in the Gram-stained smear but the resolution is not as good as in the cytological smear. Any motile protozoa observed on wet smears, coccidia and cryptosporidia, together with the types and numbers of bacteria can be determined by examining the cytological smears. The number of inflammatory cells, especially heterophils, will provide an indication of the degree of enteritis. Haemolysis of erythrocytes is usually seen when high numbers of clostridia-like bacteria are present. Additional ileal smears can be stained with auramine-O or a modified acid-fast stain to demonstrate cryptosporidia, which are most numerous in the ileum. If indicated, caecal smears can be prepared in the same way as jejunal and ileal smears to examine for coccidia.

Feed should be analysed for ingredients that may cause diarrhoea, such as sodium and mycotoxins. Similarly, water should be checked for levels of sodium, sulphates, nitrates and magnesium. The composite sample of droppings is examined for viruses and bacteria as described below, and for protozoan cysts using a faecal flotation. Faecal samples can be archived for possible future use by addition of an equal amount of 20% dimethyl sulphoxide in physiological saline, mixing thoroughly, dispensing in 1 ml amounts, snap freezing, and storing at -70°C. Levels of digestive enzymes, especially the disaccharidases found in the enterocyte brush border, can be determined using frozen intestinal segments. The levels of these enzymes are decreased in birds affected by PEC (4). The ability to absorb D-xylose is another physiological parameter that can be measured to determine the status of intestinal function (40). Paired sera are examined for antibodies against known enteric pathogens. For discovery of new pathogens, convalescent serum can be used against tissues collected during the acute stage of the disease in an immunofluorescent assay (64).

Isolation and identification of infectious agents
Methods used for detecting enteric viruses involved in PEC vary considerably depending on the agent of interest (81). Diagnosis of infection with Rotavirus is generally based on detection of viruses in faeces using EM, detection of viral antigens using FA procedures, or demonstration of RNA of Rotavirus in faeces using PAGE (62). Detection of Rotavirus in faeces by direct EM is a sensitive diagnostic approach and this method will detect rotaviruses of all serogroups (94). Immune EM and FA require specific antisera; however, these procedures may be used to identify specific serogroups. Detection of RNA of Rotavirus in faeces using PAGE has been demonstrated to be almost as sensitive as EM (94).
Diagnosis of TCV infection requires detection of virus, viral antigen, or virus-specific antibodies using virus isolation, FA, EM, or serology (69). Virus isolation may be accomplished by amnion inoculation of embryonated turkey eggs, with subsequent identification of viral antigen in embryo intestines using FA procedures. Electron microscopy may be used to detect TCV in intestinal tissues and bursa of Fabricius of infected turkeys; however, identification can be confused by the presence of cell membrane fragments ('fringed particles') that may resemble coronavirus particles (42). Immune EM is a preferable procedure in that TCV may be specifically identified with TCV-specific antisera. Serological detection of infection may be accomplished by an indirect FA procedure using frozen sections of infected turkey embryo intestines as a source of antigen. In areas where serology for TCV infection is unavailable, the commercial enzyme-linked immunosorbent assay (ELISA) for infectious bronchitis of chickens may be useful. A low level of cross-reaction occurs in the test, thereby detecting high titres to TCV.

Diagnosis of turkey enterovirus infections is most commonly accomplished by EM examination of droppings or intestinal contents (66). Both direct and immune EM procedures for detection of turkey enteroviruses have been described. In addition, diagnosis may be accomplished by detection of viral antigens in tissues or faeces using FA or antigen-capture ELISA.

Infections with Astrovirus may be diagnosed either by direct EM or immune EM of droppings or intestinal contents (79). Using direct EM, Astrovirus spp. may be confused with other small round viruses, such as Enterovirus; thus, diagnosis depends on identification of particles with characteristic size and surface structure. Immune EM is therefore the preferred diagnostic procedure.

Diagnosis of infection with Salmonella is dependent on laboratory culture and identification. Clinical samples that are most commonly recommended for laboratory culture are the caudal ileum, caeca, caecal tonsils, and caecal contents. However, samples of droppings or intestinal contents and extra-intestinal tissues (liver, spleen, heart or kidney) may also be cultured; many paratyphoid Salmonella species may become systemically disseminated in infected turkeys (39).

Standard methods for isolation and identification of Salmonella generally follow the sequence shown below:

a) enrichment to encourage growth of small numbers of Salmonella in clinical samples
b) plating on selective agar media to obtain isolated colonies
c) identification based on biochemical and serological tests.

Several enrichment media are commonly used including tetraionate broth and selenite-cystine broth. Selective media used include brilliant green agar, xylose-lysine-desoxycholate (XLD) agar, bismuth sulphite agar and Hektoen enteric agar. Culture media are generally incubated for 24 h at 37°C, but incubation of enrichment media at elevated temperatures (42°C) is often used to suppress growth of other bacteria in samples containing faecal material (intestinal tissues and droppings/intestinal contents).

Biochemical and serological tests are used to confirm the genus identity and serotype of bacteria isolated on selective agar plates. Biochemical identification is based on the fermentation pattern of the isolate using a variety of carbohydrates. Serological identification is usually determined using slide and/or tube agglutination tests (102).

Diagnosis of infection with E. coli is dependent on laboratory culture and identification coupled with identification of virulence factors (70). Cultivation of E. coli is usually performed using selective media such as eosin-methylene blue agar, MacConkey agar, or tergitol-7 agar. Presumptive diagnosis may be made from the character of colonies produced on these plates; definitive diagnosis is based on biochemical reactions. Enteropathogenic strains may be identified by occurrence of characteristic attaching and effacing lesions in intestines (Fig. 15), detection of the eae gene of E. coli using polymerase chain reaction procedures, and absence of enterotoxin and shiga toxin production (70). Serotyping is rarely sufficient for identifying enteropathogenic E. coli. In addition, this method is expensive and only a small number of reference laboratories can perform the test reliably.

Cryptosporidium can be diagnosed by identifying oocysts in intestinal tissues, intestinal contents, or samples of droppings (22). In histological sections stained with haematoxylin and eosin, parasites appear as basophilic bodies of 2 µm-6 µm, along the surface of intestinal epithelial cells (Fig. 16). Diagnosis may also be accomplished by identification of oocysts in intestinal contents and droppings using concentration procedures followed by microscopic detection. Various microscopic procedures are available for identifying oocysts of Cryptosporidium. These include bright-field or phase contrast microscopy, acid-fast staining, negative staining, or staining with auramine-O and examination with a fluorescence microscope.

Public health significance

Other than the relationship between bacteria, such as Campylobacter, Salmonella, and certain strains of E. coli, which are associated with PEC and can cause food-borne illnesses in people, and concerns regarding the transfer of antibiotic resistance between those micro-organisms infecting animals and those infecting humans, no public health risk is known to arise from PEC. No significant illness has been reported in farm workers, growers, service personnel, researchers, or others who have been in contact with affected flocks or birds, and no evidence exists to suggest that pathogens associated with the various forms of PEC can infect...
humans. However, this is an area of limited knowledge and the lack of information should not be interpreted as evidence that infections do not occur. For example, cryptosporidiosis caused by *C. baileyi*, a pathogen that can occur in turkeys, although more frequently found in chickens, was identified in an immune compromised individual (27). Any potential pathogen, especially those that occur in the digestive tract of food-producing animals, should be considered as a possible public health risk and the necessary precautions should be taken during processing to prevent carcass and product contamination.

### Prevention and control methods

Management of PEC is primarily supportive. An integrated approach is required because no single factor can control this enteric problem. Therefore, current intervention strategies incorporate both drug and management components, and actions are required before and after an outbreak has occurred. Clinical evidence shows that eggs from new breeder hens (less than seven weeks in production) should not be used for at-risk farms because smaller poults are more susceptible to PEC.

Given the viral nature of PEC, no simple solution exists. Supportive care is needed at the onset of clinical signs. This includes water-soluble multiple vitamin preparations with vitamin E at twice the recommended level (because of the antioxidant properties of vitamin E, which help stabilise enterocytes), and water-soluble antibiotic treatment. Response to antibiotics can be variable. Generally those that possess activity against Gram positive bacteria, such as tylosin, lincomycin, or penicillin, are most effective. Once the disease is present, antibiotic treatment may help speed improvement (2, 99) and limit mortality, but typically has little impact on stunting. Broad-spectrum antibacterials are not recommended during the first ten days of age because of a possible negative impact on normal intestinal microflora. Probiotics have been used without much success. If coccidia are present, the on-going anticoccidial programme must be evaluated.

Palliative care is not complete without sustained efforts to optimise the environment. If birds show huddling, a slight increase of ambient temperature (1°C-2°C) is often required. For example, higher mortality due to PEMS has been observed at lower temperatures during brooding, even under dry conditions. When litter moisture increased, mortality also increased (31). Every effort should be made to keep the litter as dry as possible (using ventilation, tilling, or top dressing with fresh litter). Various lighting programmes did not affect the mortality or stunting that occurs in PEMS (93).

Anorexia accounts for much of the stunting and growth depression, and any action that will increase feed intake should have a positive effect on PEC. Remixing feed, frequently walking through the flock to encourage movement, manually turning feed lines on and off, top dressing feed with rolled oats, whole grain, confectionery sprinkles, calf milk replacer, etc., and even using flashing strobe lights have all been used to encourage sick poults to eat. Alimentation through water is often more successful, as the birds continue to drink even after ceasing to eat. Electrolytes such as the World Health Organization rehydration solution (3.5 g sodium chloride, 2.5 g sodium bicarbonate, 1.5 g potassium chloride and 20 g dextrose per litre) in the drinking water may reduce the effect of PEC on a flock. However, efforts to reduce the impact of PEMS by adding sucrose and potassium phosphate to the drinking water only delayed mortality (28). Care is needed when adding carbohydrates to drinking water because of bacterial multiplication. Milk replacer has been used to treat coronaviral enteritis but can cause problems in automatic water lines (69). Supplementation with betaine, an osmoregulator, may help to control diarrhoea (34).

The feed used must be of top quality. A starter feed with a low percentage of protein (24%-26%) is recommended. This protein level contributes to maintaining the pH of the upper small intestine, which may help to preserve intestinal integrity. The response of the poult to PEC is influenced by nutrition. To be effective, diet modifications should be instigated early in the outbreak. Poults fed complex diets containing several protein sources seem to perform better. Highly digestible, nutritious ingredients (e.g. fish meal [if well stabilised with antioxidants] or dried whole egg powder) can alleviate some of the impact of the disease (13, 34). However, this may not be economically or technically practical. Such diets are costly and most feed mills are not able to produce small special batches of feed for isolated cases. However, changes in pellet size and texture of crumbles may be possible and beneficial. Special rations with concentrated essential nutrients may be useful to partially counteract the lowered feed intake of affected poults. Another approach is to segregate the birds from an affected flock into groups and feed each group according to size and stage of development rather than age. Runted birds (body weights of less than 50% of average body weight is a useful measure) should be rigorously culled. This will improve flock uniformity and may even permit the remaining birds to perform better.

A good relationship between ‘service people’ (personnel employed by poultry production companies who supervise production and move between farms), veterinarians and nutritionists is paramount to shorten delays in improving the environment, the management and the nutrition or feed presentation for birds affected by PEC.

### Prevention

Although research is being conducted on potential vaccines, and maternally-acquired antibodies have been shown to aid in the protection of poults against infections with Rotavirus during the first week of life (92), the problem of PEC is not likely to be solved in the near future. Therefore, the best
method to control PEC is to prevent transmission. Given the infectious nature of the disease, significant efforts have been targeted at improving cleaning, disinfection and biosecurity (in particular, limiting movement of people from farm to farm) (38).

The resistance of the viruses associated with PEC is incompletely known. Of six different treatments, only formaldehyde inactivated agents that cause PEMS (18). McLoughlin et al. were able to eliminate running and stunning syndrome from contaminated premises by depopulation followed by thorough cleaning and disinfection (60). Performance is generally better when only one age group of turkeys is kept on a farm, such as all-in/all-out operations or separate brooding and finishing farms. In addition, better flocks tend to be produced in farms which allow at least two weeks before restocking, including at least a full week after cleaning and disinfection. However, immediate removal of litter from a contaminated house is not recommended. The relatively high number of organisms present in the litter immediately after the flock is removed increases the chance of dispersal and spread to other nearby flocks during litter removal. Litter should not be taken out of the house for at least one week after the flock has been removed, to allow a reduction in numbers of pathogens through natural mortality. When used litter from a quarantined farm is removed, great care needs to be taken to spread this litter away from at-risk farms (those with young birds would be at highest risk). Vehicles used to move the litter (which should be covered) must be thoroughly washed and disinfected afterwards. Serious attention needs to be devoted to developing cost-effective technology for rearing poults on raised slats or wire floors. In limited trials, birds raised in this way have show reduction in numbers of pathogens through natural mortality.

Efforts to control disease in regions of high farm concentration cannot rely solely on single farm level prevention methods. A regional perspective is essential to co-ordinate the movement of service personnel (e.g. contractors involved in live haul or disposal of dead birds). In high-density areas, all-in/all-out production or off-site brooding will probably become the only viable option. An added key ingredient will be stringent, non-compromising, biosecurity. Finally, the frequent emergence or re-emergence of infectious diseases in poultry in the context of global trading will require enhanced communications among poultry companies in the same region. Regional depopulation/repopulation programmes may be required to effectively control infectious diseases, particularly enteric disorders. Zone raising (a regional all-in/all-out system) may eventually be required.

Regional level management

Efforts to control disease in regions of high farm concentration cannot rely solely on single farm level prevention methods. A regional perspective is essential to co-ordinate the movement of service personnel (e.g. contractors involved in live haul or disposal of dead birds). In high-density areas, all-in/all-out production or off-site brooding will probably become the only viable option. An added key ingredient will be stringent, non-compromising, biosecurity. Finally, the frequent emergence or re-emergence of infectious diseases in poultry in the context of global trading will require enhanced communications among poultry companies in the same region. Regional depopulation/repopulation programmes may be required to effectively control infectious diseases, particularly enteric disorders. Zone raising (a regional all-in/all-out system) may eventually be required.

International trade considerations

Day-old turkey poults

At least some of the infectious agents associated with PEC occur wherever turkeys are produced, and the complex has a world-wide distribution. However, information is limited and additional study is needed on the specific organisms, including distribution, and role in intestinal diseases of young turkeys. Transmission is predominantly faecal-oral. At present, Salmonella is the only organism associated with PEC that is known to be vertically transmitted. Breeder flocks, hatching eggs and poult should be tested to ensure freedom from Salmonella, particularly S. enterica subsp. arizonae, and the serovars S. Typhimurium and S. Enteriditis. None of the viruses involved in the complex have been shown to be vertically transmitted, but several have only recently been
identified and their biology is unknown. No epidemiological evidence exists to suggest that PEC is associated with specific breeder flocks.

**Finished product**

Turkey meat or other products destined for consumption are not a source of the infectious agents that cause PEC in young turkeys. However, these products should be wholesome and not contaminated with any bacteria associated with PEC, which could potentially cause food-borne illness in humans.

**Acknowledgements**

Drs D.P. Wages and E. Gonder provided invaluable insights on control, prevention and treatment of PEC. Dr D. Carver shared her findings on the comparative epidemiology of PEMS and turkey coronavirus. Dr M. Stringham contributed information on pests as vectors of PEC and their control. The authors express their appreciation to Dr S. Clark and Roche Animal Nutrition and Health for supporting a conference on coronaviral enteritis and related disorders, the proceedings of which provide the poultry industry with the most comprehensive, up-to-date source of information on enteric diseases of turkeys (20).

---

**Complexes entéritiques du dindonneau**

H.J. Barnes, J.S. Guy & J.-P. Vaillancourt

**Résumé**

Le « complexe entéritique du dindonneau » est un terme générique qui englobe diverses maladies intestinales infectieuses des jeunes dindons. Certaines maladies, telles que l’entérite à Coronavirus et le syndrome de malabsorption sont relativement bien décrites tandis que d’autres, comme l’entérite virale transmissible, le retard de croissance du dindonneau et le syndrome de mortalité par entérite des dindonneaux, restent mal connues. Toutes les formes du complexe entéritique du dindonneau sont multifactorielles, transmissibles et infectieuses. L’arrêt de croissance et les troubles de l’assimilation dus à l’entérite sont les signes cliniques les plus marquants. Dans les formes plus graves, des cas de malabsorption, une inhibition de l’immunité et des cas de mortalité sont signalés. On observe des lésions macroscopiques et microscopiques dans toutes les formes d’entérite, mais elles ne semblent guère spécifiques. D’autres lésions peuvent être observées, selon l’agent responsable. La pathogénie implique essentiellement :

a) une altération de la muqueuse intestinale, généralement par un ou plusieurs virus infectant les entérocytes ;

b) une inflammation ;

c) une prolifération d’agents secondaires, habituellement des bactéries.

Des facteurs non infectieux interagissent avec les agents infectieux, modulant ainsi l’évolution et la gravité de la maladie. La diarrhée résulte essentiellement des troubles osmotiques liés à une mauvaise digestion et à une malabsorption, mais elle peut également avoir une composante sécrétoire. La transmission se fait essentiellement par voie fécale-orale. Aucune incidence grave sur la santé publique n’a été reconnue ou suspectée. La prophylaxie consiste à éliminer les agents infectieux des locaux contaminés et à empêcher leur introduction dans les élevages. Pour ce faire, il faut mettre en place des programmes efficaces de nettoyage, de désinfection et de bio-sécurité. La conduite en bande unique et la séparation des unités de couvaison et de finissage sont également utiles. La lutte contre la maladie peut nécessiter une coordination régionale entre tous les éleveurs de dindons, surtout lorsque la production est très concentrée, ainsi qu’un programme de quarantaine pour les formes les plus graves du complexe.
entéritique du dindonneau. Il n’existe aucun vaccin ou mesure spécifiques pour lutter contre les agents responsables de ce complexe. Il existe, en revanche, un traitement pour la composante virale, tandis que les antibiotiques, notamment ceux qui sont efficaces contre les bactéries qui prennent la coloration de Gram, peuvent contribuer à réduire l’impact des infections bactériennes. Il semble que le complexe entéritique du dindonneau soit présent dans tous les élevages commerciaux de dindes, mais les informations à ce sujet sont incomplètes et la répartition des divers agents associés à ce complexe reste en grande partie méconnue. La maladie occasionne de lourdes pertes économiques, essentiellement dues au fait que les dindes ne peuvent exprimer tout leur potentiel génétique.

Mots-clés

Complejo de enteritis del pavipollo

H.J. Barnes, J.S. Guy & J.-P. Vaillancourt

Resumen
"Complejo de enteritis del pavipollo" (CEP) es un término general que designa las enfermedades intestinales infecciosas de los pavos de corta edad. Algunas de estas enfermedades están bien caracterizadas (enteritis coronaviral o síndrome de mala absorción, por ejemplo), mientras que otras conservan aún su parte de misterio (como la enteritis viral transmisible, la inhibición del crecimiento del polluelo o el síndrome de mortalidad de pavipollos por enteritis). Todas las formas del CEP son multifactoriales, transmisibles e infecciosas. Entre sus manifestaciones clínicas destacan el retraso del crecimiento y el escaso aprovechamiento de los alimentos provocados por la enteritis. En los casos más graves se han descrito enanismo, disfunciones del sistema inmunológico y mortalidad. Aunque se observen todo tipo de lesiones macro y microscópicas propias de la enteritis, éstas tienden a ser inespecíficas, y en función del agente infeccioso habrá también otras lesiones. La patogénesis más común trae aparejada la siguiente sintomatología:

a) alteraciones de la mucosa intestinal, debidas en general a la infección de enterocitos por uno o más virus;
b) inflamación;
c) proliferación de agentes infecciosos secundarios, generalmente bacterias.

Hay factores de tipo no infeccioso que interaccionan con los agentes infecciosos, modulando así el curso y la gravedad de la enfermedad. La diarrea, a la que se atribuye un carácter principalmente osmótico asociado a la mala digestión y la mala absorción, puede tener además un componente secretor. La enfermedad, que de momento parece irrelevante en términos de salud pública, se transmite básicamente por vía fecal/oral. Las medidas profilácticas se basan en el principio de eliminar el agente infeccioso de las instalaciones contaminadas y prevenir su introducción en las bandadas. Para ser eficaces, esas medidas deben traducirse en un programa adecuado de limpieza, desinfección y seguridad biológica. También resultan de ayuda la aplicación de un sistema de rotación completa de incubadora (all-in/all-out) a la separación de las unidades de incubación y de engorde. Pero luchar eficazmente contra la enfermedad exige la coordinación a escala regional de todas las granjas productoras de pavos (especialmente...
cómo un programa de cuarentena para las formas más graves del CEP, considerando sobre todo que no existen vacunas ni medidas específicas para combati r a los microorganismos involucrados. En caso de infección vírica, el tratamiento constituirá una simple medida de apoyo, mientras que el uso de antibióticos, sobre todo cuando son eficaces contra bacterias grampositivas, puede ser útil para reducir la incidencia de infecciones bacterianas. Aunque falten pruebas concluyentes al respecto, y además se ignora en buena medida la distribución de los diversos microorganismos relacionados con el CEP, de la información existente puede deducirse que esta patología está presente en todas las instalaciones industriales de cría de pavo s. El CEP provoca pérdidas económicas muy cuantiosas, debidas principalmente a la incapacidad de los pavo s para realizar plenamente su potencial genético.

Palabras clave

References


