BORDETELLOSIS

(Turkey Coryza; Bordetella avium)

DEFINITION

Bordetellosis is an acute, persistent, contagious upper respiratory disease of turkeys characterized by ocular exudation and rhinitis in young turkeys and tracheitis in older turkeys caused by Bordetella avium.

OCCURRENCE

1. Bordetellosis occurs most commonly in turkeys 1-6 weeks of age. All ages of turkeys are susceptible, including breeders.

2. Outbreaks of the disease occur in most turkey-producing areas of the United States. Similar diseases have been reported from Canada, Germany, France, England, Italy, Israel, and South Africa.

3. Farms with continuous confinement production and multiage flocks have the greatest problems with bordetellosis. Bordetellosis occurs most commonly in the summer and fall.

4. B. avium has been recovered from chickens and occasionally other avian species. Presence of B. avium has been associated with increased severity of respiratory disease in broilers, especially when flocks are concurrently infected with infectious bronchitis virus, but its role as a primary pathogen in chickens is less obvious than in turkeys.

HISTORICAL INFORMATION

1. The term turkey coryza (TC) was first used in Canada in 1967 to describe a clinically distinct, acute respiratory disease of turkeys. TC was recognized in Iowa in 1971. Following greater awareness of TC, others recalled similar disease outbreaks that occurred in turkey-producing areas for at least the last three decades. As the number of turkeys being reared in confinement has increased, TC has been identified as an increasingly important respiratory disease and cause of economic loss.

2. The terms alcaligenes rhinotracheitis and turkey bordetellosis were introduced following preliminary identification of the causative agent as Alcaligenes faecalis or Bordetella bronchiseptica-like, respectively.

3. In Europe, a virus has been shown to cause a clinically similar disease, which has been named turkey rhinotracheitis. This disease is caused by a pneumovirus.

ETIOLOGY

1. B. avium has been identified as the cause of bordetellosis. B. avium can be distinguished from other species of Bordetella and nonfermenting, Gram-negative bacteria. Hemagglutination of guinea pig erythrocytes is associated with pathogenicity and is useful in distinguishing B. avium from B. hinzii (formally B. avium-like).

2. Strains vary greatly in virulence but virulence does not appear to be related to the presence or absence of plasmids.

3. B. avium produces hemagglutinin, and heat-stable and heat-labile toxins that can be neutralized by antiserum.

4. Presence of other infectious agents, notably Newcastle virus, other paramyxoviruses, Mycoplasma gallisepticum, Pasteurella and Escherichia coli increase the severity of bordetellosis.
EPIDEMIOLOGY

1. *B. avium* is susceptible to most disinfectants and environmental conditions, especially drying.

2. Older flocks serve as recovered carriers and are thought to be the most important source of infection for younger susceptible flocks on multiage farms. Transmission between flocks occurs as a result of human activity. There is no evidence of egg transmission.

3. Litter and contaminated water have been shown to be sources of infection. The organism has been found to persist for at least 6 months in moist litter but not dry litter. Contaminated water can remain in water lines and be a source of infection for new flocks.

4. Infection of flocks less than 10 days of age strongly suggests the environment as the source of the organism. Infection between 2 and 4 weeks may result either from the environment if poult has substantial maternal immunity or introduction from an outside source. Outbreaks in flocks over 4 weeks of age result from introduction of *B. avium*.

CLINICAL SIGNS

1. Onset is abrupt 4-7 days after exposure, with high morbidity and low mortality. Growth rate is decreased.

2. In young turkeys, initial clinical signs are clear, mucoid, nasal discharge and frothy ocular exudate accompanied by sneezing, "snicking", and flicking of the head. Activity is reduced and heat sources are sought out.

3. Exudates become progressively thicker with pasting of nostrils and matting of eyelids. The palpebral opening often assumes an almond shape. There are voice changes or loss in more severely affected birds, accompanied by tracheal rales. Birds show mouth breathing. The intermandibular tissue tends to balloon giving the profile a baggy appearance [Fig. 1; Bordetellosis; NCSU]. Poult may scratch at matted eyes causing trauma to eyelids. Dried exudate is commonly found on the upper wings and lower neck where the bird wipes off nasal-ocular exudates. Swollen infraorbital nasal sinuses are not typical of bordetellosis but are occasionally seen in a few birds.

4. Tracheal rales persist for several weeks after apparent recovery. Turkeys have been found to be culturally positive for at least 4 months after infection.

5. In uncomplicated outbreaks, mortality remains low. In bordetellosis outbreaks complicated by other respiratory disease agents mortality usually begins 10-14 days after onset of clinical signs and may be high (10-60%). *E. coli* is the most common cause of mortality. Flocks in poor environments, especially if ammonia levels are high, have higher mortality and greater production losses.

6. In older turkeys, nasal and ocular exudation does not occur. Typically the only sign observed in these birds is tracheal rales.

LESIONS

1. Catarrhal rhinitis, sinusitis, and tracheitis with hyperemia of the trachea are the only consistent lesions. In severely affected birds, there is distortion of tracheal rings in proximal segments of the trachea, which leads to narrowing of the tracheal lumen and retraction of the larynx. Cross sections through an affected segment will reveal the characteristic flattening or dorsal infolding of the trachea [Fig. 2; Bordetellosis; UC Davis]. Death occurs by suffocation from an obstructed trachea.

2. A variety of other lesions can be found in complicated outbreaks, depending upon the etiologic agents present.
3. *B. avium* attaches readily to ciliated epithelial cells of the upper respiratory tract [Fig. 3; Bordetellosis; UC Davis]. This leads to deciliation, altered mucus production, impairment of mucociliary clearance, and mucus accumulation. Inflammatory changes are not pronounced but are chronic, which leads to distortion of tracheal rings and hyperplastic bronchial-associated lymphoid tissue.

4. Infection with *B. avium* has been shown to interfere with vaccination for fowl cholera but the mechanism is unknown.

**DIAGNOSIS**

1. The bacterium is readily isolated from the trachea. Typical nonfermenter colonies occur on MacConkey agar in 48-72 hours.

2. If high populations of fermenting organisms are present on the plate, *B. avium* may be inhibited. This situation often occurs when the disease has been going on for several weeks. Early in the outbreak, almost pure, dense growths of *B. avium* are readily obtained.

3. *B. avium* should be looked for in any respiratory disease of turkeys even if another cause is identified because it is a significant predisposing factor to severe respiratory disease outbreaks.

4. A variety of serological tests including rapid plate agglutination, microagglutination, and enzyme-linked immunosorbent assay (ELISA) tests have been developed to detect antibodies to *B. avium*. The microagglutination and ELISA tests are commonly used for diagnostic purposes.

**CONTROL**

1. Clean out and disinfect the brooder house and all equipment between flocks. Make sure house and equipment are thoroughly dry. Depopulate problem farms.

2. Flush water lines with disinfectant between flocks.

3. Control traffic patterns. Traffic should always move from younger to older flocks without backtracking. Ideally only one person who has no other contact with poultry should care for a single brooder house (isolation brooding).

4. Prevent contact between wild birds and young turkeys.

5. An oil-emulsion bacterin is available for use in breeder hens. This will provide poultis with maternal immunity for up to 4 weeks, the interval when infection generally results in a more severe disease.

6. A live vaccine prepared from a temperature-sensitive mutant of *B. avium* is available for use in poultis. Two doses are recommended, the first given via spray cabinet in the hatchery with a booster administered through the drinking water at 2-3 weeks of age.

**TREATMENT**

Although *B. avium* is susceptible to most antibiotics on sensitivity tests, treatment with antibiotics is generally ineffective. This is thought to be due to failure of the antibiotic to reach effective levels in the respiratory tract where the organism is located. Aerosol administration of oxytetracycline is effective in reducing clinical signs during the treatment period but has little long-term benefit. The best management for an infected flock is to move the birds to range if possible. If not, increase ventilation, increase house temperature, and frequently stimulate the flock to move around encouraging them to eat and drink. Higher density "stress" rations and use of vitamins and electrolytes in water are useful adjuncts to general support of sick birds.