Bordetella bronchiseptica

Host species

- laboratory animals (Goodnow 1980)
- wide range of domestic and wild mammalian animals, birds, humans

Organotropism

- respiratory tract

Clinical disease

- many infected animals remain asymptomatic (Bemis 1992)
- clinical disease is most commonly associated with respiratory symptoms such as sneezing, ocular, nasal discharge, coughing, and dyspnoe; signs of systemic disease include pyrexia, anorexia, chorioretinitis, vomiting, and diarrhea (Goodnow 1980, Harkness & Wagner 1995, Keil & Fenwick 1998, Speakman et al. 1999)
- pig: atrophic rhinitis with resultant twisting or shortening of the snout; most severe disease in combination with toxigenic Pasteurella multocida infection (Kamp & Kimman 1988, Sakano et al. 1992)
- rabbit: snuffles; most infections become problematic only in association with Pasteurella multocida infection (Deeb et al. 1990)

Pathology

- pig: rhinitis, atrophy of nasal turbinate bones, pneumonia (Goodnow 1980, Kamp & Kimman 1988, Sakano et al. 1992); ultrastructural changes in the turbinates are characterized by progressive degenerative changes in osteoblasts and osteocytes (Fetter et al. 1975, Silveira et al. 1982)
- dog: rhinitis, sinusitis, tracheobronchitis, pneumonia (Goodnow 1980, Bemis 1992)
- cat: tracheitis, suppurative bronchopneumonia, lymphadenitis (Speakman et al. 1999)
• **guinea pig**: serous to purulent otitis media, necrotizing tracheitis, suppurative necrotizing bronchopneumonia (Boot & Walfoort 1986, Trahan et al. 1987, Harkness & Wagner 1995)

• **rat**: acute to subacute bronchopneumonia, atrophic rhinitis (Burek et al. 1972, Kimman & Kamp 1986)

**Morbidity and mortality**

• differential host susceptibility: pigs, dogs, and guinea pigs are most susceptible; rats, rabbits, and horses have moderate susceptibility; chickens, mice, and humans are least susceptible (Goodnow 1980, Bemis 1992, Harkness & Wagner 1995)

• B. bronchiseptica infection prevalence may vary from 0 to 100% depending on the population being tested; infections are generally higher in young animals, debilitated animals, and animals kept in close confinement

• variation in the pathogenicity of B. bronchiseptica isolates

• disease associated with B. bronchiseptica is frequently accompanied by infection with other agents

• in general low mortality

**Zoonotic relevance**

• transmissible between species

• airborne and contact transmission

• infections in humans are often associated with an immunocompromised host (Woolfrey & Moody 1991)

**Interference with research**

**Physiology**

• lactic dehydrogenase activity and lactic acid and total protein concentrations were higher, and alkaline phosphatase activity was lower in blood plasma of severely affected pigs early in B. bronchiseptica infection compared with controls (Baetz et al. 1974)

• neutral mucins are decreased in nasal mucosa of pigs infected with B. bronchiseptica (Perfumo et al. 1998)

• in dogs, B. bronchiseptica infection leads to bronchial hyperresponsiveness to histamine (Dixon et al. 1979, Richards 1983) and methacholine (Nishikata et al. 1989); in guinea pigs, infection leads to hyperresponsiveness to histamine in the nasal mucosa with increased vascular permeability and recruitment of nociceptive nerve-parasympathetic reflexes (Gawin et al. 1998)

• B. bronchiseptica dermonecrotizing toxin (DNT) impairs bone formation (Horiguchi et al. 1995a)

**Cell biology**

• adherence of B. bronchiseptica to ciliated respiratory epithelial cells (Yokomizo & Shimizu 1979, Cotter et al. 1998) and induction of ciliostasis (Bemis & Wilson 1985)
- evidence for binding of B. bronchiseptica to sialyl glycoconjugates on swine nasal mucosa (Ishikawa & Isayama 1987) and to glycosylated receptors on dendritic cells (Guzman et al. 1994a)
- internalization and persistence of B. bronchiseptica in dendritic, epithelial, and phagocytic cells (Guzman et al. 1994b, Schipper et al. 1994, Forde et al. 1998)
- B. bronchiseptica exerts a cytotoxic effect on various human cell lines (van den Akker 1997)
- in osteoblast-like MC3T3-E1 cells, B. bronchiseptica DNT induces a morphological change, inhibits elevation of alkaline phosphatase activity, reduces accumulation of type I collagen (Horiguchi et al. 1991), stimulates DNA synthesis (Horiguchi et al. 1993) and protein synthesis (Horiguchi et al. 1994), induces membrane organelle proliferation and caveolae formation (Senda et al. 1997), and causes actin stress fiber formation and focal adhesions through the activation of the GTP-binding protein Rho (Horiguchi et al. 1995, Horiguchi et al. 1997)
- in Swiss 3T3 fibroblasts, Bordetella bronchiseptica DNT induces p21rho-dependent tyrosine phosphorylation of focal adhesion kinase and paxillin (Lacerda et al. 1997)

Immunology

- alveolar macrophages from rabbits colonized with B. bronchiseptica exhibit ultrastructural and functional changes (alteration of metabolic activities upon stimulation, decreases in cell adherence, phagocytic uptake, and bactericidal activity) (Hoidal et al. 1978, Zeligs et al. 1986)
- neutrophils are critical to the early defense against B. bronchiseptica infection (Harvill et al. 1999)
- B. bronchiseptica induces primarily a Th1-type T-cell response (Gueirard et al. 1996)
- serum concentrations of C-reactive protein are increased in dogs and monkeys infected with B. bronchiseptica (Yamamoto et al. 1994, Jinbo et al. 1999)
- B. bronchiseptica DNT suppresses antibody responses in mice (Horiguchi et al. 1992)

Interactions with other infectious agents

- B. bronchiseptica colonization may increase the severity of canine parainfluenza-2 virus in dogs (Wagener et al. 1984)
- B. bronchiseptica infection predisposes the nasal mucosa to colonization with Pasteurella multocida in pigs (Chanter et al. 1989, Elias et al. 1992) and rabbits (Deeb et al. 1990, Harkness & Wagner 1995) enhanced adherence of Pasteurella multocida to porcine tracheal rings preinfected with Bordetella bronchiseptica (Dugal et al. 1992)

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