

B. pertusis is a highly communicable & important pathogen of human causing whooping cough or pertusis. *B. parapertusis* causing a similar disease. *B. pertusis* & *B. parapertusis* are closely related with 72-94% DNA homology.

Morphology & identification:

The organism is G negative coccobacilli. Bipolar metachromatic granules can be seen. A capsule is present.

Culture:

The primary isolation of *B. pertusis* requires enrichment media. **Bordet-Gengou medium or Potato-blood-glycerol agar.** The plates are incubated at 37 C for 3-7 days in moist environment (Sealed plastic bag). Small, faintly G negative rods are identified by immunofluorescent staining. *B. pertusis* is non-motile. The organism is aerobic. On blood agar it produces hemolysis.

Bordetellae (Bordetella) *B. pertussis*

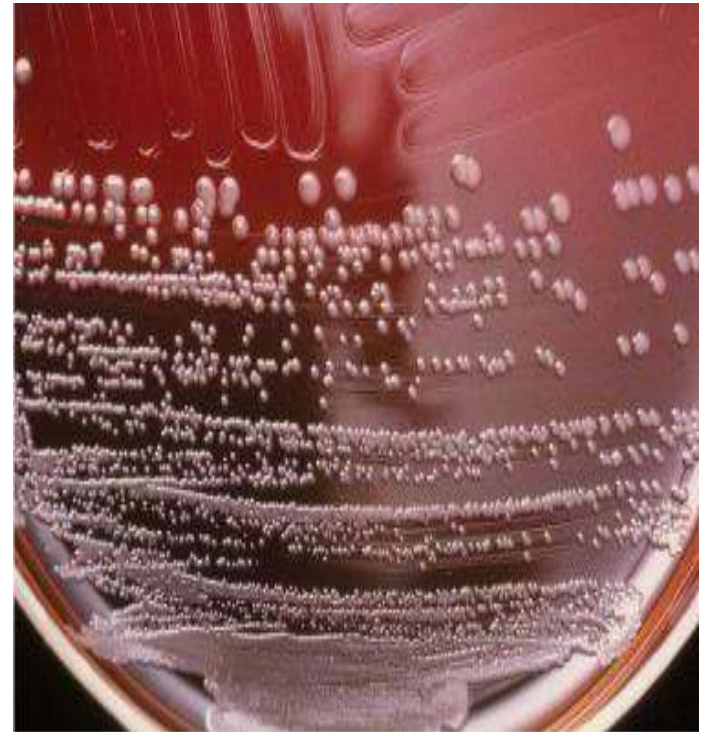


Pathogenesis & pathology:

B. pertusis produce a number of factors that are involved in the pathogenesis of disease. **Pili** & filamentous **hem agglutinin** play a role in adherence of bacteria to ciliated epithelial cells of URT. **Pertussis toxin** promote lymphocytosis, sensitization of histamine. **Adenyl cyclase toxin, dermonecrotic toxin & hemolysin** are also produced by *B. pertusis*. The tracheal cytotoxin inhibits DNA synthesis in ciliated epithelial cells. The LPS in the cell wall may also be important causing damage to the epithelial cells of the URT.

The organism adhere & multiply rapidly on the epithelial surface of trachea & bronchi & interferes with ciliary action. The blood is not involved. The organism liberate the toxins & substances that irritate surface cells causing coughing & marked lymphocytosis. These followed by necrosis of epithelium,

B. pertussis



Secondary bacterial invaders e.g. staphylococci & *H. influenzae* may lead to bacterial pneumonia. Obstruction of small bronchioles by mucous plugs result in diminished oxygenation of the blood, that may contribute to frequency of convulsions in infants with whooping cough.

Clinical findings:

After an incubation period of 2 weeks, the **catarrhal stage** develops with mild coughing & sneezing. During this stage the patient is highly infectious. During the **paroxysmal stage** explosive cough develop with characteristic “**whooping**” upon inhalation. This may lead to rapid exhaustion & may be associated with vomiting, cyanosis & convulsion. The WBCs count is high ($16-30 \times 10^3$)/ cu.mm. with an absolute lymphocytosis.

B. pertussis



Laboratory diagnosis:

Specimens: Saline nasal swab, nasopharyngeal swab or cough plate method during the paroxysmal stage.

Direct fluorescent Ab test: It can be used to examine the nasopharyngeal swab specimens. It is most useful to identifying *B. pertussis* after culture on solid media.

Culture: The saline nasal swab cultured on solid media. Subsequent identification by immunofluorescent staining or agglutination test with specific antisera.

Polymerase chain reaction (PCR).

Serology: Rise in serum agglutinin occur after the 2nd. Week of illness.

Immunity:

Recovery from whooping cough or vaccination followed by immunity. The first defense against *B.pertussis* is the Ab that prevents attachment of the bacteria to the cilia of respiratory epithelium.

Every infant should receive 3 injections of pertussis vaccine during the first year of life, followed by booster doses for a total of 5 doses. Pertussis vaccine is usually administered with toxoid of diphtheria & tetanus (**DPT**).

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Epidemiology:

Whooping cough is endemic in most densely populated areas, occur sometimes in epidemics. The source of infection is usually patients in the catarrhal stage of the disease. Communicability is high (30-90%). Most cases occur in children under 5 years of age & most deaths occur in the 1st. year of life.

Epidemiology:

