

Website: University of South Carolina, School of Medicine,
Microbiology and Immunology
pathmicro.med.sc.edu/ghaffar/zoonoses.htm
<http://pathmicro.med.sc.edu/ghaffar/plagwest.jpg>

PLAGUE

Plague is caused by *Yersinia pestis* and is the disease known in the middle ages as the black death. This is because it frequently leads to gangrene and blackening of various parts of the body. Capillary fragility results in hemorrhages in the skin which also result in black patches.

Morphology and physiology

Yersinia pestis is a pleomorphic, Gram-negative, bipolar staining, facultatively aerobic, nonmotile, bacillus. Optimal temperature for growth is 28 degrees C. It is a facultative intracellular parasite.

Epidemiology, transmission and symptoms

The three documented pandemics of plague (Black Death) have been responsible for the death of hundreds of millions of people. Today, sporadic infections still occur. In the U.S., animal (sylvatic) plague occurs in a number of western states, usually in small rodents and in carnivores which feed on these rodents.

Humans are infected by carrier rodent fleas or by contact with infected animals. The flea acquires the *Y. pestis* organisms during a blood meal from infected rodents. These organisms lose their capsule, multiply in the intestinal tract and partially block the proventriculus. During the feed on a human host the flea regurgitates some of the organisms into the wound. The bulk of non-capsular organisms are phagocytized and destroyed by neutrophils. However, few organisms are taken up by histiocytes which are unable to kill them and allow them to resynthesize their capsule and multiply. The encapsulated organisms, when they are released from histiocytes are resistant to phagocytosis and killing by neutrophils. The resulting infection spreads to the draining lymph nodes which become hot, swollen, tender and hemorrhagic giving rise to the characteristic black buboes whence the name of the disease, **bubonic** plague is derived. Within hours the organism spreads into the spleen, liver and lungs resulting in pneumonia. While in circulation, the organism causes diffuse intra vascular coagulation resulting in intra vascular thrombi and purpuric lesion all over the body. If untreated, the infection has a very high (unto 90%) mortality rate. The organism is exhaled in cough droplets, infect other humans in close proximity and cause **pneumonic** plague, which more difficult to control and has 100% mortality.

Pathogenesis

Many factors play direct and indirect roles in *Y. pestis* pathogenesis.

Low calcium response (*lcr*) : This is a plasmid-coded gene that enables the organism to grow in a low Ca^{++} (intracellular) environment. It also coordinates the production of several other virulence factors, such as V, W and *yops* (*Yersinia* outer proteins).

V and W proteins These plasmid-coded proteins are associated rapid proliferation and septicemia.

Yops: A group of 11 proteins, which are coded by plasmids, are essential for rodent pathogenesis and are responsible for cytotoxicity, inhibition of phagocyte migration and engulfment and platelet aggregation.

Envelope (F-1) antigen: This is a protein-polysaccharide complex which is highly expressed at 37 degrees in the mammalian host but not in the flea and is anti-phagocytic.

Coagulase and Plasminogen activator. Both of these are plasmid-coded proteins. [Coagulase](#) is responsible for micro thrombi formation and [plasminogen activator](#) promotes the dissemination of the organism. It also destroys C3b on the bacterial surface, thus attenuating phagocytosis.

<http://www.cdc.gov/epo/dphsi/annsum/1997/97graphs/9733.htm>

See: www.cdc.gov/epo/dphsi/images/plaggraf.gif

<http://chip.med.nyu.edu/datafiles/13/usaplague.jpg>

