Yersinia pestis **System:** Terrestrial

Kingdom	Phylum	Class	Order	Family
Bacteria	Proteobacteria	Gammaproteobacteria	Enterobacteriales	Enterobacteriaceae

Common name pest (German), bubonic plague (English), chuma (Russian), plague (English), peste (French)

Synonym
Bacterium pestis , Lehmann and Neumann 1896
Bacillus pestis , (Lehmann and Neumann 1896) Migula 1900
Pasteurella pestis , (Lehmann and Neumann 1896) Bergey *et al.* 1923
Pestisella pestis , (Lehmann and Neumann 1896) Dorofeev 1947
Bacillus pestis
Bacterium pestis ,
Pasteurella pestis
Pestisella pestis ,

Similar species *Alcaligenes spp., Pseudomonas spp., Yersinia pseudotuberculosis*

Summary *Yersinia pestis* is a gram-negative bacterium that causes plague, a highly contagious and lethal disease and the cause of three disease pandemics throughout human history. It is a zoonotic disease and exists in natural cycles involving transmission between rodent hosts and flea vectors. Humans are usually infected through bites from rodent fleas that carry the disease. Modern antibiotics are effective against *Y. pestis*, but if treatment is delayed or inadequate then the disease can cause severe illness or death.

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Species Description
Among the Enterobacteriaceae, *Yersinia pestis* is unique in both its choice of host habitat (blood, lymphoid system, reticuloendothelial system) and primary mode of transmission (flea vectors). *Y. pestis* has two main habitats—in the stomach of proventriculus of various flea species at ambient temperature or in the blood or tissues of a rodent host at body temperature (Perry 19997 in Prentice and Rahalison 2007). *Y. pestis* has been recorded to naturally infect over 203 rodent species and 14 lagomorph species. However only a small proportion are actually significant hosts, with rodents being far more important host taxa than lagomorphs (Gage and Kosoy 2005).

Notes
Curson *et al*. (1989) report that bubonic plague exhibits marked seasonality, closely related to seasonal changes in flea and rat populations. It tends to be a disease of late summer and early autumn, although it is possible that the artificially maintained indoor temperatures of many houses in the winter may allow fleas to survive. It is also possible that *Y. pestis* can be transmitted to humans via lice and other insects.
Fleas require fairly specific climatic conditions and do best in moderately warm (15-20 degrees Celsius) and moist climates (90-95% humidity). In species of fleas such as *Xenopsylla cheopis* , *Y. pestis* reproduces most rapidly at warm temperatures of up to 27 degrees Celsius. Above this temperature, changes occur in blood coagulation, resulting in digestion of the blocking plug and the rapid elimination of *Y. pestis*. *Y. pestis* produces two antiphagocytic components (that impede or prevent the function of defensive white blood cells), called F1 capsule antigen and the V antigen. Both are required for the disease to be harmful and are only produced when the host's body temperature is not lower than 37 degrees Celsius (Fix 1997).

Lifecycle Stages
Yersinia pestis is maintained in nature through transmission between hematophagous [blood feeding] adult fleas and certain rodent hosts, with occasional involvement of some lagomorphs (Gage 1998; Pollitzer 1961)" (Gage and Kosoy 2005). Evidence of *Y. pestis* infection in a wide range of other mammal groups suggests that virtually all mammals are susceptible to this bacterium (Gage 1998; Gage 1999; Pollitzer 1961 in Gage and Kosoy 2005).
"Typically, plague is thought to exist indefinitely in so-called enzootic (maintenance) cycles that cause little obvious host mortality and involve transmission between partially resistant rodents (enzootic or maintenance hosts) and their fleas (Gage *et al* 1995; Poland and Barnes 1979; Poland *et al*. 1994). Occasionally, the disease spreads from enzootic hosts to more highly susceptible animals, termed epizootic or amplifying hosts, often causing rapidly spreading die-offs (epizootics)" (Gage and Kosoy 2005). Humans and other highly susceptible mammals also experience their greatest exposure risks during epizootics."
However, there is some debate over whether epizootic and enzootic cycles actually exist. In North America plague causes die-offs of colonies of prairie dogs (*Cynomys ludovicianus*). "It has been argued that other small rodents are reservoirs for plague, spreading disease during epizootics and maintaining the pathogen in the absence of prairie dogs; yet there is little empirical support for distinct enzootic and epizootic cycles." Stapp *et al*. (2008) investigated a number of small rodent species in northern Colorado, and found no evidence that any small rodent acts as a long-term, enzootic host for *Y. pestis* in prairie dog colonies.
The question of whether *Y. pestis* can survive outside its normal host or vector has been a controversial issue. A recent study by Ayyadurai *et al*. (2008) confirmed that *Y. pestis* remains viable and virulent after 40 weeks incubation in sterilized humidified sand. Survival in soil is clearly an important mechanism for plague persistence during inter-epizootic periods and plays an important role in the epidemiology of the plague (Ayyadurai *et al*. 2008).
The main vectors responsible for transmission of *Y. pestis* to humans are usually rodent fleas, *Xenopsylla cheopis* and *Nosopsylla fasciatus*, or in some cases the human flea, *Pulex irritans* (Curson *et al*. 1989). In North America the primary vector of *Y. pestis* to humans is *Oropsylla Montana* (Eisen *et al*. 2007-C). When a flea bites its host it ingests *Y. pestis* and becomes infected. *Y. pestis* may reproduce so rapidly that it blocks the flea's proventriculus, a small organ located between the esophageus and stomach. This block prevents any ingested blood from reaching the midgut, causing the flea to starve. Regurgitation of ingested blood and infectious material from the blockage are forced back into the wound, infecting the host. This combined with increased feeding attempts from starvation make blocked fleas dangerous vectors of *Y. pestis* (Eisen *et al*. 2007-D). Spread by blocked fleas has been the accepted paradigm for plague transmission for many years.
However Eisen *et al*. (2007-D) point out "that this mechanism, which requires a lengthy extrinsic incubation period before a short infectious window often followed by death of the flea, cannot sufficiently explain the rapid rate of spread that typifies plague epidemics and epizootics" and explain the importance of unblocked fleas in *Y. pestis* epizootics. Unblocked fleas are immediately infectious, transmit the bacterium for at least 4 days, and remain infectious for long periods as they do not suffer block-induced mortality.

Uses
Yersinia pestis has the potential to be used as a weapon in bioterrorism. The United States Centers for Disease Control classified *Y. pestis* a Category A Select Agent due to its potential to pose a severe threat to public health and safety. Concerns stem from the fact that during the Cold War, both American and Soviet scientists devised means to effectively aerosolize *Y. pestis*, thereby removing the need for the flea vector (Inglesby 2000 in Smiley 2008). Although antibiotics are effective against plague, antibiotic resistant strains are known to exist. \"Covertly aerosolized, antibioticresistant *Y. pestis* would be a formidable weapon of terror\" (Smiley 2008).

Habitat Description
Among the Enterobacteriaceae, *Yersinia pestis* is unique in both its choice of host habitat (blood, lymphoid system, reticuloendothelial system) and primary mode of transmission (flea vectors). *Y. pestis* has two main habitats—in the stomach of proventriculus of various flea species at ambient temperature or in the blood or tissues of a rodent host at body temperature (Perry 19997 in Prentice and Rahalison 2007). *Y. pestis* has been recorded to naturally infect over 203 rodent species and 14 lagomorph species. However only a small proportion are actually significant hosts, with rodents being far more important host taxa than lagomorphs (Gage and Kosoy 2005).

Reproduction
According to Campbell *et al*. (1999), bacteria reproduce asexually using binary fission. Binary fission is a type of cellular division in which each dividing daughter cell receives a copy of the single parent chromosome. Growth of bacteria can be extremely fast if the resources needed are not limited and the colonies do not poison themselves with the accumulation of their own wastes (Campbell *et al.* 1999). The generation time (the time it takes for the colony to double in size) is 1.25 hours (Chu 2001).

Nutrition
Campbell *et al*. (1999) write that in order to grow in nature or in the laboratory, a bacterium must have an energy source, a source of carbon and other required nutrients, and a permissive range of physical conditions such as oxygen concentration, temperature, and pH. *Y. pestis* is a chemoheterotroph, meaning that it must consume organic molecules for energy and carbon.

General Impacts
Yersinia pestis is the causal agent of plague in humans and other mammals, although the overwhelming proportion of attention and research has focused on its impacts on humans. *Y. pestis* is recognized as causing three major disease pandemics in the 1st, 14th-17th and 19th centuries, resulting in around 200 million deaths. The second pandemic known as the Black Death caused the deaths of over 30% of the population of Europe. While *Y. pestis* no longer causes problems of such magnitude, it is still a public health concern in Africa, Asia and South America (Titball and Williamson 2001). There are at least 2000 cases of plague reported annually. In the United States it is a rare disease of humans, with only 112 cases reported between 1988-2002, although fatality rates remain high (MNWR 2002 in Eisen *et al*. 2007-B).
Biologists are increasingly realizing that wild mammal species are highly susceptible to *Y. pestis*. In North America more than half of rodent species of conservation concern occur within the range of *Y. pestis*. The impacts of plague on these populations are not well understood, but certain features increase the vulnerability of rodent species to plague. These include low natural resistance, high population densities, coloniality and sociality, abundant flea vectors, and lack of ability to cope with high demographic or environmental stochasticity.
Please follow this link for more details on the impacts of *Yersinia pestis*.

Management Info
Please follow this link for more details on the prevention, management and control of the spread of *Yersinia pestis*

Pathway
The bacteria live in fleas, which are carried by rats, rabbits, humans and other mammals. These animals can be transported around the world with human cargo. Humans can carry the fleas and the disease unknowingly during the 1 to 6 day incubation period.

Principal source: Gage, K. L., D. T. Dennis, and T. F. Tsai. 2001. *Prevention of Plague: Recommendations of the Advisory Committee on Immunization Practices (ACIP)*. Center for Disease Control, Morbidity and Mortality Weekly Report.

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ALIEN RANGE

[1] ALGERIA
[1] ARMENIA
[1] BOTSWANA
[3] CHINA
[1] ECUADOR
[1] INDONESIA
[1] IRAQ
[1] KENYA
[1] LIBYAN ARAB JAMAHIRIYA
[1] MALAWI
[1] MOZAMBIQUE
[1] PERU
[1] SAUDI ARABIA
[1] SYRIAN ARAB REPUBLIC
[1] TURKEY
[17] UNITED STATES
[1] YEMEN
[1] ZIMBABWE
[1] ANGOLA
[1] BOLIVIA
[1] BRAZIL
[1] CONGO, THE DEMOCRATIC REPUBLIC OF THE
[1] INDIA
[1] IRAN, ISLAMIC REPUBLIC OF
[1] KAZAKHSTAN
[1] LAO PEOPLE'S DEMOCRATIC REPUBLIC
[1] MADAGASCAR
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[1] UGANDA
[1] VIET NAM
[1] ZAMBIA

Red List assessed species 13: EN = 3; VU = 1; NT = 1; DD = 1; LC = 7;

Cynomys gunnisoni **LC**
Cynomys ludovicianus **LC**
Eliurus ellermani **DD**
Eliurus minor **LC**
Eliurus petteri **VU**
Mustela nigripes **EN**
Spermophilus washingtoni **NT**
Cynomys leucurus **LC**
Cynomys parvidens **EN**
Eliurus grandidieri **LC**
Eliurus penicillatus **EN**
Gymnuromys roberti **LC**
Spermophilus elegans **LC**

BIBLIOGRAPHY
63 references found for **Yersinia pestis**