

The plague: not just an historical curiosity

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The Gram-negative bacterium *Yersinia pestis* is the causative agent of the infectious disease classically referred to as the plague. Wild rodents, especially ground squirrels and prairie dogs, are the natural reservoir of the organism. The organism can occasionally be transmitted to people who are bitten by an infected animal or infected fleas that feed on those animals. Currently, *Y. pestis*-infected rodents are present on every continent except Australia and Antarctica. Plague is endemic in animals in many countries in Africa, the Americas and Asia and sporadic infections occur, at varying rates, in humans. In 2003, nine countries reported 2118 cases and 182 deaths from plague: 99% of those cases and deaths were reported from Africa.¹

The distribution of human plague coincides with the geographical distribution of infection in animals. Genome studies show that *Y. pestis* was a redundant pathogen of the intestine that acquired virulence genes from other bacteria and viruses approximately 1500 years ago.² The ability of *Y. pestis* to change its genes to suit the environment suggests that it may provide insight into ways in which highly virulent pathogens evolve.

Clinical features

Plague can present in three ways. Bubonic plague occurs following a bite by an infected flea allowing entry of the bacillus to the lymphatic system causing a bubo. The usual clinical presentation of the bubonic plague is inflammation of the inguinal lymph nodes. The bubonic disease can progress to the septicemic form of the plague where it enters the bloodstream causing bacteraemia. The third form of the plague is pneumonic plague which results from exposure to a patient with pneumonic plague or a deliberate aerosolisation of the pathogen. The symptoms of pneumonic plague include fever and a productive cough which may produce blood-stained sputum. The mortality rate ranges from 50 to 60% for untreated bubonic plague and nearly 100% for untreated pneumonic plague. However, early diagnosis and commencement of antibiotic treatment within 24 hours can reduce the mortality rate to less than 5%.

CCR5 mutation

The human genome contains remnants of our past battles with pathogens. One of these is a gene mutation called chemokine receptor (CCR)-5. In 1998, scientists tested

samples of British descendants of the plague survivors and found a high frequency of a gene mutation called CCR5-Δ32. In some parts of Europe today, up to 20% of the population can carry at least one copy of the protective gene.³

Public health response

Urgent priority must be given to a suspected case of *Y. pestis* infection. Hospitals and laboratories are required to notify immediately when the diagnosis is suspected and the New South Wales (NSW) Department of Health is to be notified on the day of the case detection. However, the presenting features of plague are often nonspecific and the diagnosis is unlikely to be suspected until an unusual Gram-negative bacillus is isolated from a blood culture or sputum. Identification of *Y. pestis* requires specialised testing in a reference laboratory accredited to handle security-sensitive biological agents.

Household and other face-to-face contacts of patients with pneumonic plague should be given chemoprophylaxis and placed under surveillance. Strict isolation is only required for patients with pneumonic plague; for patients with bubonic plague with no cough and a negative chest X-ray, effective antibiotic treatment is sufficient.⁴ In endemic areas, control of rats is the primary means for managing plague outbreaks followed by basic environmental sanitation.

The plague in Sydney, 1900–1907

John Ashburton Thompson was the first Chief Health Officer in NSW, architect of the first *Public Health Act* in NSW and an accomplished epidemiologist. His epidemiological investigations in both rats and humans provided the first real evidence for the role of the rat flea in the transmission of plague. He was instrumental in identifying rat control as the foundation of the public health response to plague outbreaks and the success of that response, first in Sydney and then internationally.

References

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