Plague: A Decade Since the 1994 Outbreaks in India

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Abstract
The severe 1994 plague outbreaks in Surat and Beed drew attention to plague as a continuing source of both natural and potentially manmade disease. This article written a decade later reviews various aspects of Plague not only as a disease but also as an infectious disaster. ©

INTRODUCTION

Plague has plagued the world over thousands of years. Currently it enjoys the status of being a category one biological weapon. India was gripped by a mass epidemic of Plague at twin locations in 1994. It is a decade since such a large epidemic in India occurred. A review of the status of Plague is being presented a decade after its unique bi-regional mass casualty outbreak in India.

HISTORY OF PLAGUE

Approximately two thousand years ago, the plague bacterium, Yersinia pestis, came into existence with the development of genetic changes in the genome of Yersinia pseudotuberculosis. Yersinia pseudotuberculosis and its progenitor, Yersinia enterocolitica, are gastrointestinal pathogens; however, with the acquirement of two plasmids that allowed the plague bacteria to grow in fleas and to cause systemic illness, Yersinia pestis became a much more virulent pathogen than its predecessors.3

There have been three acknowledged pandemics due to plague. The first pandemic, known as the Justinian plague, was caused by the plague strain, Antiqua. This pandemic arose in Egypt in AD 541 and spread throughout Europe. By the conclusion of the pandemic, 50 to 60% of the populations in North Africa, Europe, and central and southern Asia had been decimated.

The second pandemic, widely known as the Black Death or Great Pestilence, began in 1346. It is believed that this pandemic was caused by the Medievalis strain of plague and began with the siege of Kaffa (now Feodossia, Ukraine) in 1346. In one of the earliest instances of the use of biological agents in warfare, the invading Tatars catapulted bodies of dead plague victims over the walls of Kaffa. The refugees that fled Kaffa then unknowingly carried the plague bacterium to the rest of Europe. This pandemic resulted in the deaths of 20 to 30 million people in Europe, or nearly one-third of the population. In addition to the widespread mortality, this pandemic produced extensive changes in the political, religious, and cultural fabric of Europe.2

Further, according to Cole (2001), the Black Plague helped eliminate leprosy from Europe. Prior to the outbreak of the Black Plague, lepers were housed in leprosaria run by religious orders; however, with the outbreak of plague, both the lepers and their caregivers were equally decimated thus eliminating much of the leprosy reservoir in Europe.

Interestingly, there have been recent studies that have indicated that the Black Plague may have also produced selective pressures in the remaining European population that gave a survival advantage against bubonic plague to those individuals with mutations in the chemokine receptor CCR5. In modern times, these CCR5 mutations have provided some individuals with “immunity” to versions of HIV-1 that require the normal CCR5 receptor as a coreceptor to gain entrance into immune cells. However, there is still much debate as to whether this increased rate of CCR5 mutation in the European population is due to past plague outbreaks or possibly to smallpox outbreaks.4-6

The tactics used at Kaffa were again repeated at the battle of Carolstein in 1422 and in the Russian war against Sweden in 1710;1 however, these battles did not result in major pandemics.

The third pandemic began in Yunnan, China in 1855 and was caused by the plague strain, Orientalis, which is the dominant modern strain. The outbreak had spread to Hong Kong by 1894 and Bombay by 1896. Eventually, this pandemic reached every continent except Australia and caused an estimated 26 million plague cases worldwide and killed more than 12 million in China.

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and India. 

During this pandemic, much was learned in Hong Kong and Bombay about plague. The plague bacterium was first cultured by Andre Yersin in 1894 in Hong Kong. Paul-Louis Simond detected the bacterium in the remains of dead rats and proposed the transmissibility of plague via rat fleas (Xenopsylla cheopis). Also, at this time, Waldemar Haffkine was developing a crude plague vaccine. 

Plague was introduced to the United States in approximately 1902 through marine shipping. This single clone then spread through both North and South America and there is likely been the only source of all the endemic cases in North and South America. Through the 1910s to 1920s, there were further smaller outbreaks of plague. From 1910 to 1911, approximately 60,000 cases of pneumonic plague occurred in Manchuria. Concurrently, northern India was also having an outbreak of pneumonic plague. From 1898 to 1918, India had over 25 million people die from plague.

After 1920, international regulations mandating rat control on ships and in harbors greatly decreased the spread of plague. In addition, the advent of antibiotics greatly reduced the death rates from plague.

During the 1930s, Japan was developing a biological weapons program under the direction of Shiro Ishii. The program was known as Unit 731 and employed 3000 scientists and technicians. During its 13-year existence, numerous Chinese civilians and prisoners of war from China, Manchuria, America, Britain, Australia, and the Soviet Union were infected with a variety of infectious agents including plague. It is estimated that 3,000 prisoners died at the center of this program, Ping Fan. In addition, Japan aerial sprayed and released plague-infected fleas over at 11 Chinese cities. Further, the biological weapons testing by Japan resulted in an estimated 1,700 deaths and 10,000 cases among the Japanese troops stationed at Changteh.

Other countries including Germany, Britain, and the United States also began experimenting with biological weapons. Knowledge of the experimentation in Germany caused the United States to start offensive biological weapons research at Camp (Fort) Detrick Frederick, Maryland in 1943. During the 1950s and 1960s, the United States experimented with plague as a potential biological weapon until the Nixon administration disbanded the program in 1969. The United States never created sufficient plague for use as a biological weapon; however, other countries including the Soviet Union have been suspected of weaponizing plague.

In 1972, the Biological Weapons Convention was ratified by the United States, the United Kingdom, and the Soviet Union. Since that date, over 140 countries have ratified the treaty. This treaty prohibits the stockpiling of and research into bioweapons for offensive purposes. However, there is still covert research and stockpiling of biological agents.

In the more recent era, endemic outbreaks of plague still occur. By 1967, the number of cases of plague in India had been reduced to zero (National Institute of Communicable Diseases); however, the severe cases of bubonic plague cases in Beed and pneumonic cases in Surat in 1994 attest to this statement. There were 596 presumptive cases of bubonic plague and 146 presumptive cases of pneumonic plague. Of these cases of pneumonic plague, fifty-four deaths resulted. With the reports of the cases, hundreds of thousands of residents from Surat fled to other cities and brought with them the potential to spread plague organisms.

Tremendous fear rapidly spread beyond Surat and Beed with the possibility that these severe plague cases could occur in neighboring cities or possibly even other countries. With the increase in global travel, it was exceedingly possible that plague could swiftly spread to other countries. In response to this threat, several countries closed their borders to travelers and cargo from India and stopped all air flights to and from India. Although there was widespread panic over the possibility of plague spreading to other countries, the purported plague cases in Surat were never definitely confirmed and remain an issue of debate.

Several factors appear to have contributed to these outbreaks. Crowded living conditions and refuse-contaminated flood waters around the shanty towns of Surat provided a breeding ground for rats and infected fleas. The storage of grain in damaged homes left after the 1993 earthquake in Maharashtra also likely provided an attractive food supply for the rats. Further, limited health care for the poor and an insufficiently funded public health system prevented rapid identification and treatment of the outbreaks. Finally, the religious practice of rat worship likely increased the potential for exposure between humans and infected rat fleas. Unlike the purported plague cases in Surat, the cases of plague were definitely confirmed in Maharashtra.

Also, in the late 1990s, the first known cases of multiple-drug resistant plague were becoming known in Madagascar. Cases of plague began to reoccur in India with 16 cases of pneumonic plague and four deaths, Malawi, and Zambia in 2002; however, none to the severity of those outbreaks in 1994. Even at late as 2003, there were ten laboratory confirmed and one probable case of plague in Algeria.

Interestingly, in many areas of Asia, there are still numerous reservoirs of clonally diverse plague. These reservoirs provide potential sources for new outbreaks of plague that may exhibit characteristics not seen in the three known biovars of plague.

Finally, even in the United States, there are still occasional cases of plague diagnosed in the western
Plague contains several plasmids that contribute to its virulence. First, it contains a plasmid, pCD1, which contains a type III secretion system (TTSS). This system secretes several anti-inflammatory and antiphagocytic toxins (Yersinia outer-membrane proteins or Yops) that promote extracellular bacterial growth by disrupting phagocytosis and cellular communication thus leading to phagocytic apoptosis.

One of these Yops, YopM, appears to be necessary for virulence in pneumonic plague at least in a mouse model. Kerschen, Cohen, Kaplan, and Straley (2004) found that YopM interfered with innate immunity by depleting natural killer cells in the body. They proposed that YopM affected the expression of interleukin-15 and the interleukin-15 receptor alpha.

In addition to the Yops, it appears that the V antigen (LcrV) appears to have a role in plague virulence. The V antigen is currently under study as part of potential plague vaccines. The V antigen appears to increase the amounts of interleukin-10 in the infected host. Normally, interleukin-10 reduces inflammation and aids against septic shock; however, plague appears to co-opt this system via the V antigen (Brubaker).

Further, unlike the TTSS in Yersinia pseudotuberculosis or the TTSS encoded on pCD1, plague also contain a chromosomal TTSS (TTSS-2) that appears to be closely related to the TTSS encoded by Salmonella pathogenicity island 2 (SPI-2) found in Salmonella enterica serovar Typhimurium. SPI-2 promotes the replication of Salmonella within macrophages.

In addition, plague contains the plasmids, pMT1 and pPCP1. Both pMT1 and pPCP1 are associated with the increased virulence of plague. pPCP1 encodes for a potent plasminogen activator that aids bacterial spread in the tissues; and pMT1 has been associated with vector-borne transmissibility. Further, the response regulator PhoP may also regulate several genes involved in the intracellular replication of plague and this intracellular replication may be very important when the disease has begun at a peripheral location (bubonic plague).

Finally, studies by Garcia et al (2002) have found approximately 37 plague genes that have increased activity at pH 5.5 and approximately 38 plague genes that have increased activity at pH 4.4. They also found that the catalase gene (KatA) and the catalase-peroxidase gene (KatY), which are both involved in the detoxification of damaging oxygen species and free radicals normally produced by macrophages, have increased activity at pH 5.5. It is been theorized that several of these genes may be involved in the virulence process of plague.

**Epidemiology**

Plague is an enzootic disease throughout the world except Australia. The primary reservoir is rodents such
as rats, mice, ground squirrels, and prairie dogs.

Plague is usually transmitted by the bites of infected rodent fleas. The plague bacilli block the gastrointestinal tract of the flea. It appears that this blockage is actually a biofilm of plague organisms and that it is a bacterial defense measure against predation by invertebrates. In the meanwhile, the starved flea ravenously bites mammals in vain attempts to feed. These bites introduce the plague bacteria into the mammalian tissues thus producing bubonic plague. Then, uninfected fleas then may become infected by biting the infected mammal.

In order for a flea to become colonized, it must ingest approximately 10,000 plague bacteria; and to ensure infectivity, the flea must ingest approximately 100,000,000 plague bacteria per milliliter of blood ingested; although, the typical blood meal of a flea is approximately 0.1 microliters.

However, plague can also be transmitted by the handling of infected rodents, rabbits, wild carnivores, and house pets. House cats appear to be especially susceptible to both the bubonic and pneumonic forms of plague, and can be implicated in the spread of plague to humans. Of 15 human cases of plague following exposure to infected domestic cats, 4 of these cases were pneumonic plague.

Environmental and social factors may also assist the spread of plague. The pneumonic form of plague appears to have enhanced transmission in humid climates. In addition, the El Nino weather pattern can also produce cyclical changes in the occurrence of plague outbreaks. Further from studies of the outbreaks of plague in Manchuria in the 1910s and 1920s, it has been suggested that indoor contacts of infected patients are at higher risk for contracting plague than outdoor contacts and that overcrowding additionally contributes to the spread of the plague organism.

Further, Keeling and Gilligan (2000) mathematically examined the effects that the culling of rats could have on the potential for bubonic disease to occur in a human population. They concluded that if the rat population was continually kept at a minimum even with infected rats, the risk of a large plague outbreak in the rat and human populations could be minimized. However, if the culling of the rats started after the development of human cases of bubonic plague (due to an infected rat population), there would be an increased likelihood of a significant plague outbreak in the human population because the infected fleas would have to seek an alternative food source (i.e. humans).

Plague remains endemic in much of the world and because it has been reappearing in areas plague-free for decades. For this reason, the World Health Organization has listed plague as a reemerging disease. Endemic plague foci remain in Africa, Asia, North America, and South America. According to the World Health Organization, 14 countries reported 2,603 cases and 212 deaths from plague in 1999. These figures are comparable to the rates over the prior decade. The majority of the cases and deaths were from Africa (WHO).

While in the United States, at least 390 plague cases have reported over the past 50 years primarily in the western United States. Approximately eighty-four percent of the cases were the bubonic form (with a fatality rate of 14%). Thirteen percent were septicemic plague (with a fatality rate of 22%). In contrast, only 2% were pneumonic plague yet these cases had a fatality rate of 57%.

**CLINICAL SIGNS AND SYMPTOMS OF PLAGUE**

Although, *Yersinia pestis* is the sole cause of plague, the disease may present clinically in several different forms including the bubonic, septicemic, pharyngeal, and pneumonic versions. The mortality rate for untreated bubonic plague is from 50% to 60%, and 100% for untreated pneumonic and septicemic plague. However, the fatality rate for bubonic plague drops to less than 5% with prompt treatment; although, even with proper treatment within 24 hours, the prognosis remains poor for both pneumonic and septicemic plague. Plague may further spread to the brain as plague meningitis, which can occur in about 6% of the septicemic and pneumonic cases. However, if there is a successful recovery from plague, there may be short-term immunity to the organism.

With the bubonic form, the incubation period is usually from 2 to 10 days. The usual cause of bubonic plague is the bite from an infected flea. Less than 25% of bubonic cases may be a skin lesion at the site of the fleabite. Buboes, which are extremely tender, erythematic, and nonfluctuant lymph nodes, usually develop with bubonic plague. The buboes may appear in the axillary and cervical areas; but, they are more commonly seen in the inguinal lymph nodes since the legs are the most common site of flea bites.

Buboes are typically accompanied by malaise and an acute high fever. The liver and spleen may also tend and palpable. Bubonic plague may progress to metastatic infection, septic shock, and blood clots in the small arteries. The blood clots in the arterioles may produce gangrene in the fingers and toes. These gangrenous areas provide explanation for the term, the Black Death. In addition, secondary pneumonic plague may occur in 25% of bubonic plague cases.

Next, the septicemic form of plague may develop from any of other forms or from direct plague inoculation into the bloodstream. Septicemic plague can present with an acute onset of bacteremia, septic shock, thromboses with and without preceding lymphadenitis, disseminated intravascular coagulation, necrosis of
small blood vessels, and purpuric skin lesions.

With pharyngeal plague, pharyngitis and cervical lymphadenitis may develop from exposure to larger infectious droplets and/or ingestion of infected tissues. These droplets are too large to reach the lungs.

Finally for pneumonic plague, it can be divided into two types. The first type is primary pneumonic plague, which may develop from the inhalation of infectious droplets, as might present from a biological attack or from close exposure (usually six feet) to a person infected with pneumonic plague. While, secondary pneumonic plague spreads via the bloodstream from bubonic or septicemic plague.

In general, the incubation period for pneumonic plague ranges from 1 to 6 days. Symptoms may include a high fever, headache, muscular weakness, severe malaise, muscle pains, and rigors (severe chills). These symptoms may progress further as dyspnea, stridor, cough with bloody or purulent sputum, and may present as a bronchopneumonia. This pneumonia may appear as bilateral infiltrates or consolidation on chest radiology.

Further, gastrointestinal symptoms such as nausea, vomiting, abdominal pain, and diarrhea may occur. The tip of the nose and the fingers and toes may also exhibit acrocyanosis as a sign of thromboses and digital gangrene and a low-grade disseminated intravascular coagulation may develop. And, in rare instances, the individual may develop cervical buboes. The individual may also infrequently develop a low-grade disseminated intravascular coagulation. Finally, in the later stages, signs of septic syndrome may develop. These symptoms may then progress to respiratory failure, circulatory collapse, and severe bleeding that lead to death.\(^2\)

### Diagnostic Measures and Laboratory Analysis

Diagnosis may be made by one of several methods; however, there are currently no rapid methods of detection. Although in the past few years, there have been a variety of studies examining more rapid and accurate methods of detection of the plague organism. First, a study by Loiez, Herwegh, Wallet, Armand, Guinet, and Courcol (2003) used real-time polymerase chain reaction (PCR) to test sputum samples for the plasminogen activator gene from *Yersinia pestis*. They found that their test was 100% specific for the plague plasminogen activator gene and may useful for the rapid diagnosis of pneumonic plague. Another study used PCR methods to better differentiate *Yersinia pestis* and *Yersinia pseudotuberculosis* by analyzing the O-antigen gene cluster to reduce the potential for misidentification.\(^{20}\)

For plague diagnosis, Wright, Giemsa, Wayson, or Gram staining can be used. The staining will show Gram-negative bacilli with a bipolar safety-pin appearance. However, it must be noted that bipolar staining of cells is not limited to *Yersinia pestis*. Other *Yersinia* species, enterics, and Gram negative microorganisms (in particular the *Pasteurella* species) may exhibit a bipolar safety-pin appearance.

Diagnostic samples may come from blood, sputum, cerebrospinal fluid, or lymph node aspirates. However, sputum may be contaminated by normal throat flora; therefore, bronchial and transtracheal aspirates are preferred over sputum.

Chest radiography may show patchy peribronchial infiltrates, cavitation, consolidation, hilar adenopathy, and pleural effusions. However, these results are of limited usefulness.

With bubonic plague, greater than 80% of the blood cultures will be positive for plague organisms. In addition, blood from all plague cases may indicate leukocytosis with a total white blood count of 20,000 cells. Hematological tests will also show an increase in the number of bands; and, greater than 80% of cells will be polymorphonuclear cells. Further, fibrin split products, the liver enzymes ALT and AST, bilirubin, blood urea nitrogen, and creatinine may also be elevated.

Plague typically grows slowly at normal incubation temperatures. It grows optimally at temperatures of 25°C to 28°C. The bacillus can be grown on blood agar, MacConkey agar, or in an infusion broth. Its colonies are initially smaller than other *Enterobacteriaceae*; therefore, it may require 48 hours for growth, which can produce misidentification with automated systems.

For presumptive identification of plague on SBA agar, the colonies will be gray-white to slightly yellow opaque and will be 1 to 2 mm in diameter after 48 hours of incubation on SBA agar plates at 35°C. After 48 to 72 hours, the colonies will have a fried-egg appearance and may have a hammered-copper shiny appearance with minimal hemolysis. In contrast, on MacConkey agar plate, tiny lactose-negative colonies will appear after 24 hours growth at 35°C. Finally, in brain heart infusion broths, the plaque bacterial growth will be clumped or flocculent after 24 to 48 hours at 28°C to 35°C. In addition to these currently available media, a potentially more effective media for the isolation of *Yersinia pestis* has also been developed. This new media is based on the brain heart infusion agar but additionally includes the selective agents, irgasan, cholate salts, crystal violet, and nystatin.\(^{19}\)

The literature recommends that if plague is suspected the culture should be split into two samples. The first portion should be incubated at 28°C for rapid growth; and the second culture should be grown at 37°C for identification of the diagnostic fraction 1 (F1) antigen. This may require up to 72 hours from specimen collection to proper identification.

While working with suspected plague organisms, simple clinical activities and cultures should be processed in biosafety level 2 conditions. And, activities
involving high potential for aerosol or droplet production activities including centrifuging, grinding, vigorous shaking, and animal studies should done under biosafety level 3 conditions.

However, presumptive diagnosis may also be made on the basis of serum antibody titers and on the basis of the detection of antigens. Increased serum antibody titers to *Yersinia pestis* F1 antigen in patients without a history of plague vaccination are a presumptive diagnosis. In addition, the detection of F1 antigen in a clinical specimen by fluorescent assay is also presumptive. The F1 antigen is normally present on naturally occurring plague bacteria.

Finally, confirmatory diagnoses may be made on the basis of the isolation of *Yersinia pestis* from a clinical specimen or from a fourfold or greater increase in the serum antibody titer to the *Yersinia pestis* F1 antigen.

The Centers for Disease Control (CDC) provides cases classifications based on clinical symptoms and laboratory results. A suspected case is defined as a clinically compatible case that is lacking presumptive or confirmatory laboratory results. A probable case is defined as a clinically compatible case with available presumptive laboratory results. And, a confirmed case is defined as a clinically compatible case with available confirmatory laboratory results.

**Treatment and Prophylaxis**

According to the Centers for Disease Control and Prevention Lesson 5 Medical Management (of plague), the preferred treatment for adults with plague is streptomycin 1 gram intramuscularly twice a day or gentamicin 5 mg/kg intramuscularly or intravenously daily or 2 mg/kg loading dose with 1.7 mg/kg intramuscularly or intravenously three times a day. Alternatively, adults may be given doxycycline 100 mg intravenously twice a day or 200 mg intravenously, ciprofloxacin 400 mg intravenously twice a day, or chloramphenicol 25 mg/kg intravenously four times a day. However, in instances of mass casualty, doxycycline 100 mg orally twice a day or ciprofloxacin 500 mg orally twice a day are preferred treatments; and alternatively, chloramphenicol 25 mg/kg orally four times a day may be given.

As for children, the CDC recommends streptomycin 15 mg/kg intramuscularly twice a day (maximum of 2 grams daily) or gentamicin 2.5 mg/kg intramuscularly or intravenously threes times a day. Alternatively, children may be given ciprofloxacin 15 mg/kg twice a day or chloramphenicol 25 mg/kg intravenously four times a day. However, in instances of mass casualty, doxycycline 2.2 mg/kg orally twice a day (for children under 45 pounds) or 100 mg orally twice daily or ciprofloxacin 20 mg/kg orally twice a day are preferred treatments; alternatively, chloramphenicol 25 mg/kg intravenously four times daily may be administered.

**Vaccines**

As for plague vaccines, there have been several studies testing different methods for the prophylaxis and potential treatment of plague in recent years. Prior to its discontinuation in 1999, the plague vaccine that had previously been used in the United States had been effective against bubonic plague but had only variable effectiveness against pneumonic plague.25 Currently, the potential vaccines that are being tested for potential use against plague include vaccines that use portions of the F1 capsular antigen of the plague bacterium,24 vaccines that use monoclonal antibodies specific to the F1 and V antigens of the plague bacterium,25 and a nasal Proteosome-based vaccine that can be used for pneumonic plague.26 Each of these potential vaccines has shown positive results.

**Microbial Resistance**

Since the 1990s, there have been increasing reports of plague organisms resistant to multiple antibiotics normally used to treat plague. The first reported case multiple-drug resistant plague was in Madagascar in 1997. It is likely with the continuing use and misuse of antibiotics that further cases of antibiotic-resistant plague will become known. In addition, there have been previous reports of the intentional development of antibiotic-resistant plague organisms prior to the end of the Soviet Union.

**Prevention and Surveillance**

For endemic plague, effective rodent control and proper medical treatment have greatly reduced the morbidity and mortality from the disease. Rapid diagnosis and treatment of the disease are essential in preventing the spread of plague.

Potentially, plague may also be used as a form of bioterrorism; however, the amount of plague, the particular strain, the dispersal method, and environmental conditions would greatly affect the size of an intentional outbreak. The first indication of a covert plague attack would likely be a sudden outbreak of pneumonia-like illness with sepsis. However, the symptoms may vary depending on the dispersal method.

A study by Gani and Leach (2004) examined the potential for secondary cases of pneumonic plague that might potentially develop from primary pneumonic cases due to either natural or manmade sources. They estimated that the average number of secondary cases infected by a primary case would be 1.3; however, they also indicated that the actual eventual number of cases would depend greatly on the rapidity of detection and treatment of the plague outbreak.

**Infection Control**

For individuals presenting with symptoms of pneumonic plague, they should be maintained under respiratory droplet isolation for the first 48 hours after the start of treatment. If the diagnosis of plague is
confirmed, the person should remain in respiratory droplet isolation until the sputum samples are negative for plague bacilli. If there are insufficient beds for plague patients, they may be cohorted together; however, they will need to wear surgical masks during transportation.

For individuals in close contact with suspected or confirmed plague cases, they should begin seven days of prophylactic therapy with the appropriate antibiotics and should respiratory droplet precautions. If, however, these individuals refuse prophylaxis, they do not need to be isolated but should be observed for the development of a fever or cough during the seven days following the exposure. If they develop symptoms, they should have immediate treatment.

Cleaning of hospital beds after the discharge of a pneumatic plague patient only require standard precautions. Clothing and linens can be cleaned per hospital policy. And, bodies of plague victims should be handled with the routine strict precautions.

Heat, disinfectants (such as 2 to 5% bleach), and sunlight will normally destroy the plague bacterium. However, there are studies that have shown that the plague bacterium can persist on materials commonly used in hospitals for several days. Plague does not have a spore form; therefore, there is little need for environmental decontamination after a plague exposure.2

**Responsibilities of the Public Health System in Plague Control**

Even before a plague outbreak, there should be planning for a plague outbreak. Written plans should exist that indicate the steps that will be taken in the advent of an outbreak. Each person that will be involved in the process should know the activities that will perform in the event of an outbreak. Further, there should be contingency plans in case the original plan is ineffective.

In addition, there needs to be surveillance for potential signs of a plague outbreak whether natural or manmade. This surveillance should be ongoing and should be communicated to all involved parties. Both health care workers and the general public should be notified of the surveillance as needed. In addition, the media should be kept informed so that accurate information is reported. The media can be useful in disseminating necessary information and preventing panic in the public.

Further, prevention, early detection and then prompt treatment with isolation are essential to halting a plague outbreak. Finally, after the cessation of a plague outbreak, the complete outbreak should be reviewed to determine areas of improvement in order to decrease the likelihood of future outbreaks.

**CONCLUSION**

Plague remains both a source of endemic disease in the world and a potential source of bioterrorism. However, with prompt diagnosis and treatment, the morbidity and mortality due to plague can be greatly reduced; and, future outbreaks like those that occurred in Surat and Beed in 1994 can be prevented.

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