PLAGUE AND BIO-TERRORISM

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Abstract
Plague ranks high on the list of biological agents that might be used by terrorists. However, its status as a weapon of mass destruction is based on two assumptions: (a) that it caused the Black Death and (b) that it was responsible for heavy mortality in India in the late 19th and early 20th centuries. As to the former there is no convincing evidence that it was the agent of the medieval pandemic. Plague deaths in India, whilst apparently high when expressed as a total over the 44 years before antibiotics became available, were annually no greater than those of local endemic diseases. Even pneumonic plague, the only form liable to be used by terrorists, has serious limitations as to delivery method and contact rate. Furthermore, evidence from past epidemics of pneumonic plague has shown that one infected person would, on average, infect only 1.3 others, a situation that could be coped with by movement restriction, isolation and antibiotics.

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2 A pandemic occurs when a single disease spreads across a large area or is world-wide.
1. Introduction

Bubonic plague is the latest scare topic to hit the bookshelves with authors, or more likely publishers, vying with one another in their attempts to alarm. The title of the latest offering tells us that plague is the world’s most dangerous disease and the jacket blurb that “the marmots of Central Asia, in particular, have long been hosts to the most virulent and frightening form of the disease, a form that can travel around the world in the blink of an eye” !1 Evidence for this is, not surprisingly, lacking although in the text the author does advance the view that plague originating from marmots presents a greater danger than that from rats 2. Considering that well over 95% of plague deaths in the world since 1894 have been due to rats, this is a curious claim.

For anyone seeking to present an alarmist view it is mandatory to refer to India in the early years of the current pandemic e.g. “in the early twentieth century, plague again swept Asia, taking the lives of 12 million in India alone” 3. As we shall see later, when this figure is viewed in relation to population size, time span and biological conditions a quite different picture emerges.

Another recent book, ‘The Barbary plague: the Black Death in Victorian San Francisco’ 4, while giving a fascinating account of the battle between civic, medical and state authorities, with a hostile press in the middle, nevertheless misses the point that whereas the Black Death, whatever it was, killed perhaps 22 million in 3 years in medieval Europe, the so-called Black Death of San Francisco, which was bubonic plague, killed only 113 people in 5 years, a figure that owed little to rodent control or any other sanitary measures. Surely that obvious difference should have urged some caution in the title: but would have sold fewer books.

Plague has a poor image, the history books telling us repeatedly of the calamitous effect wrought by the disease in the past: in England, for example, mortality estimates for the Black Death vary between 20 and 50% of the population 5. Plague figures high on the list of organisms that could be used in terrorism and as weapons of mass destruction and long before such matters arose the word had entered the language as an object of fear e.g. to avoid something like the plague. We should do well to remember, though, that in past centuries the word ‘plague’ was used in a generic sense. Writing in 1721 the London physician Dr. George Pye declared that plague, “differs from an epidemic disease, but in degree of violence only: and consequently any epidemic sickness that rages with more than ordinary violence and which occasions an extraordinary mortality amongst mankind, may be and is properly termed a pestilence, or the plague” 6.
Plague is perhaps the most intriguing of those bacteria and viruses of interest in bio-terrorism because it has an interesting distant past and a well researched more modern past during which we learned how complicated its biology really is. However, we also learned that modern plague is a much less fearsome proposition than most of the outbreaks of ancient ‘plague’, no matter what those presenting the alarmist scenarios say. This fear has been engendered by, on the one hand, a muddling of ancient and modern data, and on the other an imperfect understanding of the biology of plague and its epidemiology. The biology of the disease is more complex than that of most other serious disease organisms because of the involvement of rodents and fleas in the cycle of infection and in turn on the dependence of those animals upon climatic conditions and the structure and social status of human dwellings. It is important, therefore, to begin with a clear idea of plague biology i.e. the disease of bubonic plague and its septicaemic and pneumonic forms.

2. The Plague Disease

2.1 Bubonic plague

Plague is cause by the bacterium *Yersinia pestis*. It produces an acute disease in rats and man, fatal in both in a high proportion of cases, and is transmitted to man by the rat flea. In man the bacteria become localised in lymph nodes in the neck, armpit and groin forming large swellings, the buboes, from which the disease gets its name. Before antibiotics were available between 60 and 90% of such cases died, usually within three to five days. An important aspect of any epidemic disease is its ability to infect further victims and so ensure its own survival. Consequently, such factors as the length of time that an infected person can infect others in contact is an essential feature of the effectiveness profile of an organism. People ill with bubonic plague pose no threat to others since in this form of plague, even in fatal cases, there are few bacteria in the blood and the human flea would not be effective in inter-human transmission because there would be too few bacteria to form colonies in the gut of the flea.

2.2 Septicaemic plague

This form of plague is relatively rare. It occurs when the bacteria, instead of entering the lymph nodes, spread throughout the blood system to form an overwhelming septicaemia, death usually occurring within 24 hours. Bacteria are only present in the blood for a very short time. During this period the human flea can take in bacteria and transfer them to a new host without the need of rats. However, the human flea is not a good transmitter and the fact that bacteria are only available for a short time militates against this route as an effective means of infecting many victims.
2.3 **Pneumonic plague**

When plague bacteria enter lung tissue the third form, pneumonic plague, results. This form of the disease is very dangerous, with a 100% fatality rate unless treated with antibiotics. It may occur in a small number of cases during a bubonic epidemic and is then termed secondary pneumonic plague, as distinct from primary pneumonic plague, which is usually acquired by aerosol infection from wild rodents during skinning when fur trapping. This is the only form of plague with a theoretical capacity for the infection of many people by person-to-person transmission but pneumonic plague is not a common outcome of infection by *Y. pestis*, usually making up less than 3% of cases and, contrary to what is often said, it is not highly communicable. The latter point is especially noteworthy for *Y. pestis* is poorly adapted to transmission by respiratory aerosols, probably because there are loss-of-function mutations in two genes that limit its ability to cross lung epithelium. Furthermore, although bacteria can be spread in the ejected sputum, most patients are dead within 48 hours (1.8 days in Manchuria in 1920-21) and that already short potentially infective period is reduced still further by the fact that the patient is not coughing out bacteria for the first 24 hours and for the remainder of the illness is prostrate.

3. **Rats, fleas and climate**

Plague is primarily a disease of rodents and most cases of human plague are acquired from rats sharing the same buildings, most usually dwellings or work places such as dockyard warehouses. Temperatures between 20 and 28°C promote breeding of the rat flea and at the same time the plague bacteria in the rat blood stream multiply to form an overwhelming septicaemia. The death of large numbers of rats is termed the epizootic, the equivalent of the human epidemic, and without a food supply the fleas move to a new host which may be another rat or a human. Since early times in the East the epizootic provided an early warning system of impending plague and wherever possible people left their villages until the danger was past.

4. **The historical background of plague**

The word plague has its origin in the Latin *plaga*, a blow or a stroke, and this acquired the additional meaning of pestilence because widespread and sudden death was regarded by pagan Romans as a blow from the gods and by christianised Romans as a stroke of divine wrath. ‘Plague’ occurs throughout English early literature, and elsewhere, to describe the appearance of large numbers of animals, particularly pests, or outbreaks of epidemic disease, no matter what type. It was therefore a catch-all word ready for any eventuality.
The received wisdom tells us that there have been three plague pandemics: the first was the one which spread across the Mediterranean region during the reign of the Byzantine Emperor Justinian (A.D.527-565) and had its origin in central or eastern Africa. It appeared in a port at the eastern end of the Nile delta in 541 and took about two years to spread over most of the Middle East and the Mediterranean. It may have reached Ireland in 544 or 545 and perhaps Wales in 547. The second was the Black Death which between 1347-50 spread from the toe of Italy to the most northerly parts of Scandinavia. The third, and present, pandemic began in southern China in the 19th century, reached Hong Kong in 1894, and spread over most of the world in the following decade.

Most scholars of the periods in which the first two pandemics occurred consider that there is little doubt concerning the diagnosis of bubonic plague for both. Such questions as have been asked come from modern studies of the Black Death and these turn out to be equally relevant to the earlier pandemic. The questioning arises from the nature of the third pandemic and it is in this comparative approach that some confusion has arisen and become part of the accepted history. What has happened is “that early authoritative accounts of the third pandemic have included not only historical backgrounds but at times the historical record of the Black Death to supplement the data available for the third pandemic. These accounts are then treated by historians of the Black Death simply as scientific views of the third pandemic, independent of historical interpretation. Thus the history of the Black Death is to some extent explained using evidence of the modern pandemic that was originally based in part on narratives of the Black Death. Because evidence for the early pandemic is so much sparser and more reticent than that for the Black Death, the latter is called upon to help interpret the former. In a less vicious circle than the one connecting the second and third pandemics, the Plague of Justinian thus becomes part of the historical background for the Black Death” 12.

Modern laboratory work has also been affected by the acceptance of the same disease for all three pandemics. The name \textit{Yersinia pestis} is derived from Alexandre Yersin, who first isolated it and after the ‘Great Pestilence’ - the Black Death. This linking of the second and third pandemics as the same disease is therefore now built into its name. Again, a less than neutral terminology has been used for the three biovars (strains) of \textit{Y. pestis} believed to have been responsible for the three pandemics: these are, respectively, \textit{antiqua, mediaevalis} and \textit{orientalis}.

The nub of the debate centres on those aspects of the modern pandemic which contrast it strongly with the other two: there are at least seven major points of difference and these have been covered in detail elsewhere 13,14. This may be an academic debate but with bio-terrorism in mind the distinction between modern and ancient pandemics assumes major importance. As it stands, the perception of the lethal capacity of modern plague is based on its supposed performance in the first two
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pandemics, largely due to an emphasis on modern case mortality and the use of figures for deaths in India between 1896 and 1917. Since bio-terrorism is all about how many people can be killed in a fairly short period of time let us examine just how effective plague is in this respect.

5 Ancient mortality estimates

5.1 The Plague of Justinian

Procopius, in his narrative of the Persian Wars, had returned to Constantinople in 540 and describes high mortality of 10,000 a day and higher, with deserted villages and abandoned cattle. John of Ephesus put the figure in the capital at 16,000 per day with as many as 300,000 taken off the streets where they had died. Without any precise knowledge of the population size before and after, and with due allowance for some degree of magnification, it is difficult to know the true scale of the outbreak.

5.2 The Black Death

Mortality estimates are almost entirely based on proxy data, some of which, such as the replacements of priests, have many flaws. The size of the population of Britain has been a problem for historians but for England a figure of 4.25 to 4.5 million would be consistent with the agricultural resources of the country.

If we take the estimate of 4.5 million, then on the two generally accepted mortality estimates of 20 and 50%, somewhere between 900,000 and 2,250,000 died. The case mortality in modern bubonic plague lies between 60 and 90% and even taking the lower limit, the total number of people who had actually contracted the disease would have been, respectively, 1,500,000 and 3,750,000. Bearing in mind that 90% of the population lived in hamlets or small villages, that it is fairly certain that the rat in N.W. Europe at the time was not present in rural England and that the outbreak killed people at all seasons, then without the rest of the objections it casts serious doubt on bubonic plague as the organism concerned.

Across Europe, 22 million people died at the lowest estimate and, repeating the previous calculation, the number contracting the disease at the 60% mortality level would have been at least 36.6 million.

6 Modern mortality: the Plague of Canton

6.1 Bubonic and septicaemic plague

As we have already seen, bubonic plague is rarely transmitted person to person and the same is true for septicaemic plague. Any organism that is to be used as a terrorist
weapon would have even greater capability if the original recipient could infect many others before succumbing and if those others could in turn affect yet more. In this respect these two forms of plague have no part to play in spreading the organism. Yet bubonic plague in the late 19th and early 20th centuries is still seen as a scourge that caused heavy mortality more or less world-wide and it is worth examining what really happened.

Bubonic plague had been diffusing throughout south-western China since at least 1772 and eventually reached Hong Kong and other south China ports in the last decade of the 19th century. This was a new disease for the area and, on its supposed performance in earlier centuries, it should have produced a matching mortality since knowledge of its transmission mechanism was still unknown and would remain so for the best part of the next two decades. It did not, however, live up to its record.

In 1894 the population of Hong Kong was approximately 242,365. In that year there were 2679 plague cases, 2552 of which died, a case fatality rate of 92.6%. This was the worst year yet only 1.05% of the population died of plague. Between 1894 and 1923 the only other year in which plague cases exceeded 2000 was 1914, with 2146 cases, of which 2020 died. By that time the population numbered 495,209 so that the percentage dying of plague was only 0.4.

In the period 1893-1907 a total of 95,832 people died in Hong Kong and it is interesting to compare deaths from plague with those from other illnesses:

- Plague: 12,506
- Pulmonary tuberculosis: 9,392
- Tetanus: 7,293
- Malaria: 6,977
- Bronchitis: 6,037

Even more interesting is the situation in Taiwan for the years 1906-15. The total number of deaths was 949,405 and the principal causes of death were:

- Malaria: 94,741
- Childbirth & Pregnancy: 83,043
- Diarrhoea (infant & other): 75,340
- Respiratory ailments: 62,694
- Bronchitis: 39,314
- Pneumonia: 42,110
- Plague: 7,974

Steam was now taking the place of sail and from the South China ports plague was soon transmitted to all the major ports in the world and to all the continents. Yet
again, its impact was not only variable but in places slight and even in the worst affected situations no more serious than local endemic diseases. World plague mortality in the period 1894-1938 is shown as follows:¹⁷

<table>
<thead>
<tr>
<th>Area</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>India</td>
<td>12,500,000</td>
</tr>
<tr>
<td>China &amp; Taiwan</td>
<td>250,000</td>
</tr>
<tr>
<td>Indonesia</td>
<td>214,000</td>
</tr>
<tr>
<td>Africa</td>
<td>120,000</td>
</tr>
<tr>
<td>Madagascar</td>
<td>32,000</td>
</tr>
<tr>
<td>South America</td>
<td>24,000</td>
</tr>
<tr>
<td>North America</td>
<td>1,000</td>
</tr>
<tr>
<td>Europe</td>
<td>1,000</td>
</tr>
<tr>
<td>Rest of world</td>
<td>10,000</td>
</tr>
</tbody>
</table>

India bore the brunt of the mortality but even there the figures have to be put into perspective. If the 12.5 million deaths were equally distributed throughout the 44 years there would be 284,090 deaths per year. However, plague in India was most severe in the years 1896-1917 when 9,841,396 people died of the disease. At that time the total population was around 329 million so that over those early years plague was responsible for an average of 447,336 deaths, or 0.135% of the total population per annum. (In passing it should be noted that this plague pandemic killed just over 13 million people world-wide: the influenza pandemic following World War 1 killed over 40 million whilst at least 300 million died from smallpox in the 20th century. It has been estimated that the annual number of cases of smallpox in the world in the early 1950s may have been as high as 50 million¹⁸).

However, the effects of plague in India were not evenly distributed throughout the twenty years 1896-1917: in 1897 plague deaths per 100,000 of the population were about 20: by 1901 this figure had risen to 100, in 1904 it was 370 and by 1907 it was up to 410. Unlike the Black Death the effect was not an immediate, heavy mortality for it was eleven years before the highest death rate was reached. During the first four or five years of plague in India it failed to alter the annual mortality significantly because other endemic diseases were numerically more important. A good example of this is cholera:
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*Benfield Hazard Research Centre, Disaster Studies Working Paper 10 (September 2004)*

<table>
<thead>
<tr>
<th>Year</th>
<th>Deaths recorded from cholera</th>
<th>Deaths recorded from plague</th>
</tr>
</thead>
<tbody>
<tr>
<td>1896</td>
<td>471,779</td>
<td>2,288</td>
</tr>
<tr>
<td>1897</td>
<td>555,035</td>
<td>55,548</td>
</tr>
<tr>
<td>1898</td>
<td>152,703</td>
<td>117,733</td>
</tr>
<tr>
<td>1899</td>
<td>171,410</td>
<td>135,996</td>
</tr>
<tr>
<td>1900</td>
<td>797,222</td>
<td>92,106</td>
</tr>
</tbody>
</table>

Total: 2,148,149 from cholera, 403,671 from plague.

It is a telling fact that in the third pandemic, 95% of the plague deaths in the world between the onset of the pandemic and World War II occurred in India. The reason for this was that the biological situation in India provided optimal conditions for the maintenance of the rat and flea vectors and for their ability to contact human victims. This was particularly true of the cities. In Bombay for example, the modernisation and development of the city in the late 19th century had produced a serious public health situation even before the arrival of plague. Commercial growth had caused a huge increase in the number of the working class with enormous overcrowding in the already congested slums and widespread environmental contamination. The population per acre in Bombay reached a maximum of 759; by comparison the greatest density in Calcutta was 208 and in London, somewhat more highly built upon, it was 222.19.

If plague-infected fleas are to be used in bio-terrorism then large numbers must be delivered to the target population. One logistical problem, among many, is that even in the flea species *Xenopsylla cheopis*, which is the most effective one known, only 12% of the fleas are ‘blocked’: that is they have a large blockage of plague bacteria in their gullet which enables them to infect a warm-blooded animal such as man.20 During the Sino-Japanese war of the 1930s the Japanese dropped plague-infected fleas on the Chinese and it is reported that many people were killed in these trials.21 Since towns represent the largest concentration of people they would be the obvious target but present one difficulty: that is, when viewed from above, the largest component of a city is rooftops and most of the falling fleas would land there in situations where they would be harmless and easy prey to birds and other predators, depending on the climate and regional fauna. The difficulties in this form of plague bio-terrorism are considerable and probably outweigh the advantages.

### 6.2 Pneumonic plague

This is the only form of plague that is worth consideration by a bio-terrorist. The two most severe epidemics of pneumonic plague have taken place in Manchuria and it is
these that are cited as examples of the way in which this form of plague might produce a pandemic. However, examination of the logistics and evolution of the two outbreaks shows a less alarming picture and in fact suggests that had one designed the physical features of this situation as an experiment to show how a pandemic could be started it would not have been deemed a success. It is, therefore, important to study the details of the two outbreaks.

The first epidemic in 1910-11 was probably due indirectly to an increase in the price of marmot skins which rose from 0.30 rouble in 1907 to 1.20 roubles in 1910. The exports of these skins increased from 700,000 in 1907 to 2.5 million in 1910, a rise that was due to an increase in the number of hunters: in the summer of 1910 there were 11,000 Chinese hunters at Manchouli and by October there were still 4,600. Rodents dying of plague appear sick and drowsy and are easy prey. The native hunters were skilled: they shot animals and had learned to avoid sick marmots. The influx of get-rich-quick hunters were unskilled Chinese who used snares and, since these are unselective, the hunters had to handle sick animals. Pneumonic plague cases may arise in skinning wild animals when the nature of the handling compresses the lungs so that plague bacilli are released as an aerosol which the hunter, working close, is in an ideal position to inhale.

Despite being pneumonic the evolution was slow and cases occurred as early as April 1910 and continued throughout the summer. Although officially recorded at Manchouli, the hunting centre, on 12 October, it was present a month earlier. This was too late to prevent the spread of plague along the railway system, west to Siberia and east into China. From Manchouli to Shantung the disease covered 1700 miles during the period September to April and almost 60,000 people died.

The conditions in which the hunters lived on their way home were ideal for spreading a disease like pneumonic plague. The Chinese inns were mud-built, one-storied, badly lit and ill-ventilated with windows tightly sealed with paper to exclude the cold air. At night the inmates lay close together on a heated brick platform to economise on space and for the sake of warmth. At stations on the way, hunters left the trains and made their way to their home communities, taking plague with them and starting fresh foci. Although this was a potential recipe for a much wider epidemic the fact that in the 1921 outbreak average life expectation was only 1.8 days acted as a restraint on the spread of the disease. Another feature that restricts the diffusion of this form of plague is that it is almost never transmitted more than two or three steps from the initial pneumonic case.

The important epidemiological feature to come out of the two Manchurian epidemics was the realisation that although pneumonic plague had a considerable diffusion potential in cold weather, the infectivity of patients was not so absolute as had been believed: “In Manchuria, as in other plague areas, a short, close contact is often
insufficient to produce infection. Likewise, the number of victims among the persons isolated after having been found in contact with plague patients is, as a rule, very low” 22. The author of this paper, the Chinese plague expert Lien-Teh Wu, concluded that the sick room by itself, and even when occupied by plague patients, was not particularly dangerous except when standing in the direct line of the spit or droplet. Modern work showing the limited ability of \textit{Y. pestis} to cross lung epithelium, 23,24, supports this observation made 80 years earlier.

Even the confined space in railway carriages was shown to be less conducive to infection than was thought and Wu quotes cases where patients with fever and cough travelled in railway cars with 47, 37, 47 and 30 other persons for more than nine hours and yet, despite the close proximity of the pneumonic plague-infected person, none of these other passengers became ill.

Recent work has shown that the average number of secondary cases per primary case (R\textsubscript{o}) for eight outbreaks of primary pneumonic plague between 1907 (Seattle, USA) and 1997 (Madagascar) was 1.3. This figure means that one infected person would, on average, infect 1.3 others and bearing in mind that when R\textsubscript{o} becomes less than 1.0 the epidemic will die out it shows the low infectivity of pneumonic plague. Similar figures for bubonic and septicaemic plague are not available but they will be well below 1.0 and almost certainly close to zero. Despite this low figure the authors maintain that “pneumonic plague poses a potentially increasing risk to humans in plague non-endemic regions either as a consequence of an aerosolized release or through importation of the disease” 25.

Urban populations present the only worthwhile target for bio-terrorism in so far as the aim would be to kill as many as possible in a short time and provide a nucleus of organisms for the infection of others, the material of choice being pneumonic plague. With this in mind the World Health Organisation calculated in 1970 that in the worst-case scenario, “If 50 kg of \textit{Y. pestis} were released as an aerosol over a city of 5 million, pneumonic plague could occur in as many as 150,000 persons, 36,000 of which would be expected to die. The plague bacilli would remain viable as an aerosol for 1 hour for a distance of up to 10km. Significant numbers of city inhabitants might attempt to flee, further spreading the disease” 26. It is not known how the contact calculation of organisms and people was made: was it at rush hour or when most people were in the buildings at work, and so on? Again, though, the area of rooftops would ‘absorb’ a high proportion of the material: was that taken into account in the calculation? It is assumed that in this scenario antibiotic treatment would be available.

Because plague bacilli would not remain viable for long, then this method would have to rely upon a successful contact rate in the first hour: after that point further
contacts would be made by those already infected. In this scenario, a restriction on movement would need to be total and immediate.

7. Discussion

The differences between the two earlier pandemics and the modern one lead us to think that the Black Death almost certainly, and the Plague of Justinian to a great extent, were not due to \textit{Y. pestis}. Furthermore, the performance of modern plague does not justify the position it holds in the array of potential biological warfare agents and one has to conclude that much that is laid at the door of modern plague arises from the belief that it was the agent responsible for the mortality in the two earlier outbreaks, particularly the Black Death.

Recent studies have suggested that some ‘plague’ epidemics in England were due to an organism that was passed by respiratory means person to person and had an incubation period of around 37 days. Such an organism, with a long incubation period during which an infected person could travel and infect several others at markets and such gatherings, would have been singularly effective as well as appropriate for the pattern of diffusion seen in the Black Death. The widely dispersed human population could not have been infected by a rat-borne disease. The authors suggest that a virus caused the high mortality: they call the disease ‘haemorrhagic plague’\textsuperscript{27}. It is interesting that the Italian City States were active in epidemic surveillance and introduced maritime quarantine in 1377 in the Venetian colony of Ragusa. This was at first 30 days and was then extended to 40, a period which was strictly kept in Europe for 300 years. Its purpose in northern Italy was to prevent importation of the disease, rather than to isolate individuals who were already ill\textsuperscript{28}.

In practical terms the production of a suitable plague weapon would pose more than the usual technical difficulties associated with bacterial growth. The only way of getting plague into a person is by either plague-infected fleas or an aerosol of plague bacillus. The former has obvious problems, in addition to which the low proportion of ‘blocked’ fleas reduces the effective strike rate by about 90%. Field experience has shown that pneumonic plague is neither easy to acquire nor transmit further and it can be treated with antibiotics.

However, following World War II, the Soviet Union had biological weapons programmes and carried out research into antibiotic resistance, genetic engineering and methods of producing \textit{Y. pestis} in aerosol form\textsuperscript{29}. It is also thought that they had developed means of producing pneumonic plague bacilli that would be more effective in their means of penetrating lung tissue. That may be so, but others were no doubt pursuing the same ends and it leaves them in a position somewhat akin to that of nuclear weapons in the Cold War whereby use and counter-use may result in a lot of dead people but produce a stalemate. The fear has been expressed that scientists
involved in such work in the former Soviet Union may have gone elsewhere, taking
their expertise and, perhaps, samples with them for sale to the highest bidder, or at
any rate someone who would give them work.

The development of plague as a weapon of mass destruction is only feasible under
the auspices of national laboratories which could produce the considerable quantity
of material needed to produce death on a large scale. Even then, the difficulties of
distributing the organisms to a civilian population present many problems and the use
of aerosol plague on the battlefield could be a double-edged weapon in addition to
such awkward matters as ambient temperature and survival of the plague bacillus, an
organism that does not live for long outside the body.

Modern terrorism is carried out by small groups of people or individuals whose
strength lies in their ability to move about frequently and to communicate
electronically. The production of plague and other organisms would demand a pattern
of behaviour that is not easy to reconcile with a cryptic lifestyle: extensive glassware
and metal flasks would be necessary, plus cages for animals and the considerable
space needed for such laboratory work. All this is too obvious unless carried out in
some deserted corner of the world and even then it is difficult to see it being
conducted in caves in Afghanistan. Even if enough plague material could be
manufactured, there remains the question of transporting it to the target.

Considering the technical difficulties of preparation and distribution, the use of
plague by small groups of clandestine terrorists is unlikely. By far the most potent
weapon in relation to this organism is fear and outbreaks of plague in various parts of
the world in the early 20th century produced a reaction that had overtones of the
events that had taken place in the 14th century. For example, when plague reached
San Francisco in 1900 the New York Journal proclaimed ‘Black Plague Creeps Into
America’ 30, yet in the first ten months there were only 23 recorded plague deaths.
This slow start is typical of bubonic plague, in contrast to the Black Death, which in
the same period of time in 1348 had covered the whole area from the toe of Italy to
the south of England.

Given the particular, but unreasonable, fear of plague that would be presented by the
media and even those who ought to know better, the spreading of innocuous material
thought to be plague might achieve a degree of panic that could be difficult to
control. The experience of an epidemic that included a few cases of pneumonic
plague in India in 1994 31, was an illustration of how things can quickly get out of
hand when plague is believed to be present and the modern response, it seems, is
unlikely to be very different from the medieval one. The most dangerous object today
would be the unrestrained use of the single word Plague.
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