BLACK DEATH:
The Causes and Effects of a Pandemic
by
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Abstract
Medical historians, bacteriologists, epidemiologists, immunologists, and entomologists alike have argued for almost a century about the cause of one of the worst pandemics in human history: “The Black Death.” This phrase was not used until the 17th century by British historians who are thought to have been connecting death and darkness with an especially mournful period in the late medieval literature from 1347-1352. It has been estimated that half the populace of Europe were robbed of their lives by a disease demonstrating several different forms during five long and horrifying years. Most microbiologists today believe the phrase “The Black Death” comes from the dark patchy appearance of the skin that is manifest in modern cases of bubonic plague. Using a plethora of translated descriptions among writers and medical practitioners during the time of The Black Death correlations will be drawn with modern medical descriptions of infectious disease that have direct bearing on specific causality. There are several alternative views among historians who differ with the generally held position that bubonic plague be held responsible for the Black Death. This debate will be reconstructed along with the addition of new evidence highlighting key descriptions of skin manifestations and symptomology that reassures the status of bubonic plague as the cause of The Black Death. The purpose of this study not only focuses the cause of Black Death in order to perhaps prevent reoccurrence, it helps us to understand how medical practitioners, natural philosophers, and the public in general struggled to cope with accepted knowledge about a disease that supremely challenged the reality of their experience

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Introduction
According to F.A. Gasquet, the seventeenth century English origin of the name “Black Death” had something to do with “a collective state of mourning,” or the “special characteristic symptoms” of an unparalleled epidemic[1]. It requires an enormous burden of proof for any microscopic organism to be held responsible for killing roughly 30-40 percent of the population of Europe, or an estimated 17 to 28 million people from 1347-1352[2]. Since the isolation and description of *Yersinia pestis* at the end of the “golden age” of microbiology in 1894, by the Swiss-French bacteriologist Alexandre Yersin, it is widely held that the small bacterium was responsible for the Black Death and several more pandemics that followed in Europe and Asia. There are several alternative views among historians who differ with the generally held opinion that bubonic plague caused the Black Death. The alternative views of Graham Twigg and Samuel Cohn will be analyzed by comparing modern and medieval observations about skin manifestations. Evidence concerning skin pathology will be enjoined with the epidemiology, immunity, nutrition, and the entomology during the Black Death. The purpose of this study not only focuses the cause of Black Death in order to perhaps prevent reoccurrence, it helps to understand how medical practitioners, natural philosophers, and others tried to cope with accepted knowledge about disease that supremely challenged the reality of their experience.

Comparative Skin Manifestations
Yersinia pestis infection causes a bewildering variety of symptoms, which have been classified into three types of plague called bubonic plague, septicemic plague and pneumonic plague. In the United States from 1970 to 1994, 78 percent of all cases were bubonic plague, 13.2 percent septicemic plague, and 4.4 percent were pneumonic plague[3]. Untreated plague mortality is 40 to 60 percent for bubonic plague, and 100 percent for septicemic or pneumonic plague[4]. Based on the extraordinarily high population mortality rates during the Black Death, it is likely that septicemic and pneumonic plague were common forms of infection. The perplexity of differing symptoms for three types of plague can be resolved by comparing medical microbiology textbooks. Twigg and Cohn exclusively refer to *Manson’s Tropical Diseases*, a highly regarded medical text with a different specialist authoring each disease.

Analysis of skin manifestations offers a method for considering the variety of opinions present in modern medical descriptions and in chronicles and plague tractates. There are three aspects to consider in the skin

pathology of bubonic plague. The first is the extent of skin reaction to flea inoculation. There are surprisingly few comments in the medical literature about skin manifestations related to flea inoculation. According to Davis and Dolbecco, "a small pustule may be present at the portal of entry in the skin but more often there is no discernable lesion[5]." An inflammatory response on the skin is dependent upon the number of infecting microbes being regurgitated into the wound by the flea. Possibilities range from no reaction, to a small red area at the bite, to instances where the bite itself could abscess with *Yersinia pestis* forming lesions of varying size, and potentially becoming infected with other bacteria.

There are few descriptions of small pustules on different sites of the body in the chronicles. According to one possible account taken from a chronicle at the monastery of Neuberg in southern Austria in 1351:
The signs that generally preceded the pestilence were red apostumes dotted around the genitals or under the armpits, and those victims with no hope of recovery voided blood.[6]

Another possible instance is accorded to Geoffrey le Baker, a clerk of Swinbrook in Oxfordshire:
People who one day had been full of happiness, on the next were found dead. Some were tormented by boils which broke out suddenly in various part of the body, and were so hard and dry that when they were lanced hardly any liquid blowed out. Many of these people escaped, by lancing the boils or by long suffering. Other victims had little black pustules scattered over the skin of the whole body. Of these people very few, indeed hardly any, recovered life and health.[7]

Geoffrey le Baker has possibly suggested multiple flea inoculations containing large numbers of highly infectious *Yersinia pestis*. As few as three to fifty *Yersinia pestis* bacterium can be infective with flea inoculation.[8]

The second aspect of skin appearance is the formation of buboes. Buboes are caused by swollen lymph nodes draining infection nearest to where flea inoculation with *Yersinia pestis* has occurred. The buboes are commonly described in connection with the Black Death by chroniclers and in the plague tractates. White blood cells present at the site of the bite are designed to ingest and digest rapidly multiplying bacteria and generally are capable of removing them from blood to the lymph nodes. However, phagocytosis is unsuccessful during bubonic plague due to powerful *Yersinia pestis* virulence factors that interfere with phagocytic mechanisms. *Yersinia pestis* is thereby permitted exponential proliferation in the regional lymph nodes. An unknown author of the plague in Padua describes the buboes as tumors. The chronicler informs us:
Some were infected very badly by this plague and died suddenly from blood poisoning, others from malignant tumour, or from worms. A certain sign of death, found on almost everyone, were incurable tumours near the genitals, or under the armpits, or in some other part of the body, accompanied by deadly fevers.[9]

According to M.D. Smith in *Manson's Tropical Diseases*, bubonic plague consists of buboes that cause severe pain, swelling, and tenderness. Buboes often attain the size of a hen's egg, and the overlying skin is red and may erupt or abscess in later disease stages. M.D. Smith describes these under the heading of bubonic plague:
In some patients small skin lesions such as vesicle and pustules may be seen in the region drained by the affected nodes. These may ulcerate or form an eschar or rarely a carbuncle. Y. *pestis* can be isolated from these lesions. Although uncommon, perhaps the best-known skin manifestation is a patchy purpuric dermal necrosis, which gave rise to the popular name Black Death.[10]

It is significant to note "patchy purpuric dermal necrosis" as a distinctively different objective subcutaneous skin symptom occurring near death.

The following medical microbiology texts list these dark subcutaneous skin manifestations as less frequent with bubonic plague, but common with septicemic plague. Nearly all record the dark subcutaneous skin manifestations as the probable source for the name "Black Death." In contrast, M.D. Smith posits these patchy purpuric skin manifestations as uncommon during bubonic plague, and omits observations about

subcutaneous skin manifestations during septicemic plague. This omission implies that patchy pupuric skin manifestations were uncommon during the Black Death of 1348, and conflicts with other authorities. In Nester's *Microbiology*, "The dark hemorrhages in the skin from disseminated intravascular coagulation and the dusky color of skin and mucous membranes probably inspired the name black death for the plague."[11] In Madigan's *Brock Biology of Microorganisms*, "Multiple hemorrhages produce dark splotches on the skin giving plague its historical nickname, the Black Death."[12] In Talaro's *Foundations in Microbiology*, "The release of virulence factors causes disseminated intravascular coagulation, subcutaneous hemorrhage, and purpura that may degenerate into necrosis and gangrene."[13] In Ingraham's *Introduction to Microbiology*, "blood vessels are destroyed, and subcutaneous bleeding causes black spots to appear on the skin."[14] In Tortora's *An Introduction to Microbiology*, "This term [Black Death] comes from one of its characteristics, the dark blue areas of skin caused by hemorrhages.[15]

An analysis of objective skin symptoms in medical microbiology texts offers three distinct skin colors most often described as black, purple or blue splotches. Black splotches are brought about near death from endotoxic leakage throughout the capillary network of vital organs including the skin. With millions of microscopic lesions occurring in the blood vascular system, there is a desperate attempt on the part of the body clotting system to "plug all leaks." This process is known as disseminated intravascular coagulation, or D.I.C. As the body runs out of clotting factors, blood hemorrhages out into the dermis and epidermis. These dark areas may be diffuse underlying a large area of the skin. The subcutaneous tissues may become necrotic in a short number of hours setting up conditions for gangrene. Septicemic plague is often dismissed in connection with the Black Death because it is relatively rare in modern cases. However, septicemic plague symptoms are commonly described in the fourteenth century plague tractates as would especially be expected with a new virulent strain of *Yersinia pestis*. With the advent of a new strain of bacteria, it must be argued that a more significant role for the cosmopolitan *Pulex irritans*, as opposed to the rat flea Pulex irritans, is to be expected if septicemic plague was more common during the Black Death.[16]

The third disease manifestation of *Yersinia pestis* is pneumonic plague. Symptoms of pneumonic plague are evident in plague tractates translated by Anna Campbell. For example, Ibn al-Khatib, a western Islamic poet and philosopher from Granada, offers a brief description of pneumonic plague during the Black Death: An acute disease, accompanied by fever in its origin, poisonous in its material, which primarily reaches the vital principle by means of the air, spreads in the veins and corrupts the blood, and changes certain humors into a poisonous character, whence follow fever and bloodspitting and breaking out in exanthem of a pestilential sort.[17]

The complex aspect of pneumonic plague is that it occurs in two forms. Primary pneumonic plague has no skin manifestation and is transmitted by direct-contact, respiratory-droplet route. Although pneumonic plague does not occur frequently in modern cases, it was very likely highly contagious and fatal during the Black Death in light of a more virulent strain of *Yersinia pestis*. Secondary pneumonic plague is the result of septicemic metastases into the spleen, liver, meninges, and lungs after flea inoculation. In these cases, dark hemorrhagic skin manifestations are common, but there are no buboes. Ibn al-Khatib appears to be describing secondary pneumonic plague in the context of exanthem skin manifestations, and with absence of buboes.

Secondary pneumonic plague also may arise from bubonic plague. In this case there are buboes with potentially eruptive lesions and dark hemorrhagic skin manifestations. Once again, with the exception of *Manson's Tropical Diseases*, medical texts describe the dark skin hemorrhages near death as the likely cause of the name Black Death. If the majority of medical texts are correct, then the Black Death was often due to septicemic plague, thereby strongly implicating a more virulent strain of *Yersinia pestis*. Samuel K. Cohn rejects the notion that Black Death was caused by bubonic plague. Cohn tells us in his book, "This book reexamines over 400 narrative chronicles, 250 plague tracts, 50 saints' lives, merchant letters and more."[18] Cohn is interested in detailing the symptoms of the late-medieval plague in order to provide clues about the etiology and epidemiology of the Black Death. However, he underestimates the variety of skin manifestations based on the different classifications of bubonic plague when he informs us: These sources show that the signs and symptoms of the late-medieval and modern plagues do not match one another nearly so closely as present-day doctors and historians continue to proclaim.[19]

With the many classifications of bubonic plague and their concomitant skin manifestations, it is expected that signs and symptoms of late medieval chroniclers would not always match one another. The variety of skin descriptions in connection with bubonic plague lends support to the etiology of *Yersinia pestis* during the Black Death. In fact, it would more easily rule out *Yersinia* as the cause of the Black Death if all skin manifestations were the same in every case.

Giovanni Boccaccio's *Decameron*, offers the most well known literary treatment of the symptoms of Black Death. His description of symptoms approximates what other contemporaneous chroniclers in Florence had to say about the Black Death:

On the contrary, its earliest symptom, in men and women alike, was the appearance of certain swellings in the groin or the armpit, some of which were egg-shaped whilst others were roughly the size of the common apple. Sometimes the swellings were large, sometimes not so large, and they were referred to by the populace as *gavoccioli*. From the two areas already mentioned, this deadly *gavocciolo* would begin to spread, and within a short time it would appear at random all over the body. Later on, the symptoms of the disease changed, and many people began to find dark blotches and bruises on their arms, thighs, and other parts of the body, sometimes large and few in number, at other times tiny and closely spaced.[20]

After examining Boccaccio's description of the Black Death in Florence, Cohn informs us how this description fails to square with modern case evidence. Cohn writes:

As we shall see, neither the plague boils nor such spots were to spread over the body in more than 3,000 clinical cases reported in Bombay in 1896-97 or in 36 cases in Glasgow in 1900. In 95% of cases only one plague boil formed and with no modern plague have black pustules spreading randomly all over the body been reported. At most, a few points might form around the fleabite, but such marks are rare and usually occur with pneumonic or [septicaemic] plague, when buboes do not have time to form at all.[21]

It is likely that less severe symptoms in Bombay or Glasgow were due to changes in herd immunity over a five hundred year period since the Black Death. Most significant to medical historians is Boccaccio's description of egg-shaped swellings on the skin, which perfectly match the description of buboes in bubonic plague. His description of disease symptoms changing to "dark blotches" aligns with "dark patches" in modern medical microbiology texts. It is highly probable that *Yersinia pestis* became more virulent in timely accordance with the Black Death, and thereby became a severe new threat in an immunologically naive human population.[23]

Louis Heyligen of Beeringen, was a musician performing with a group for Cardinal Giovanni Colonna at the papal court in Avignon. His extraordinarily descriptive letter includes an exceptionally accurate rendering of symptoms affecting bubonic plague victims. A copy of it was made at the time and placed into the chronicle of an unknown Flemish cleric as part of his account of the plague in 1347. Heyligen wrote:

It is said that the disease takes three forms. In the first people suffer an infection of the lung, which leads to breathing difficulties. Whoever has this corruption or contamination to any extent cannot escape, but will die within two days. Anatomical examinations, in which many corpses were opened, were carried out in many Italian cities, and also, on the pope's orders, in Avignon, to discover the origins of this disease, and it was found that all those who died suddenly had infected lungs, and had been coughing up blood. And this form is the most dangerous of all these terrible things, which is to say that it is the most contagious, for when one infected person dies everyone who saw him during his illness, visited him, had any dealing with him, or carried him to burial, immediately follows him without any remedy. There is another form of the disease, which exists alongside the first one, in which boils erupt suddenly in the armpits, and men are killed by these without delay. And there is also a third form, which again coexists with the other two, but take its own course, and in this people of both sexes are attacked in the groin and killed suddenly. Because of the growing strength of this disease it has come to pass that, for fear of infections, no doctor will visit the sick (not if he were to be given everything the sick man owns), nor will the father visit the son, the friend the friend, the acquaintance the acquaintance, nor anyone a blood relation – unless, that is, they wished to die suddenly along with them, or to follow them at once.[24]

Heyligen's letter offers a detailed medical analysis from a person without any medical training. Further, it is the only description of bubonic plague that describes all three manifestations of the disease beginning with pneumonic plague, bubonic plague and septicemic plague. Scott, Duncan, and Cohn fail to recognize

this significant description by chroniclers in the biological role of *Yersinia pestis* in the Black Death. Rosemary Horrox, on the other hand, conveys her regard for the chroniclers when writing:

Even the words plague or pestilence, which became the standard terms for the disease, were originally non-specific, and have remained so: not all plagues are *the plague*. This is not to say that contemporaries had failed to recognize they were dealing with a specific disease, or why they were hazy about its manifestations. On the contrary, writers across Europe not only present a consistent picture of the symptoms of the disease, but had realized that the same disease was taking two distinct forms.[25]

But Horrox, like many others, falls into the trap of minimizing septicemic plague as the third distinct form of plague during the Black Death.[26] Presumably, this is because modern medical plague authorities have diminished the possibility of its involvement with Black Death. Horrox does provide excerpts from chroniclers detailing at least a dozen explanations about the symptoms of bubonic plague. Many of these excerpts arrive at the term "boil" which in a modern sense refers to a *Staphylococcus* skin infection. In bubonic plague, boils were probably analogous to literal eruptions in the skin from swollen buboes, or actual *Staphylococcus* infections that occurred after buboes were lanced to balance the humours.

Graham Twigg makes a thorough accounting of fourteenth century symptoms described by chroniclers from many different areas of Europe. He offers a description by a Montpellier doctor named Simon De Covino who refers to the disease "pestis inguinaria" or "the bubonic plague of the East."[27] He describes "a burning pain, beginning under the arms or in the groin and extending to a region of the heart."[28] Covino adds, "A mortal fever then spreads to the vital parts; the heart, lungs and breathing passages being chiefly affected."[29] Twigg then dismisses Covino:

This account seems at variance with bubonic plague for once the buboes have appeared there is little likelihood of the disease then involving the respiratory system as he indicates and as it would in pneumonic plague alone, which would be unlikely to show buboes.[30]

Twigg misses the diagnosis in this case. In light of the many plague classifications and associated skin manifestations, it seems plausible that Covino is describing bubonic plague and secondary pneumonia from bacteremia and metastasis to the lungs. It is doubtful that such an event was rare during the Black Death.

In an analysis of French medical descriptions, Twigg offers commentary from 1348 by Gui de Chauliac, one of the doctors for Pope Clement VI. As quoted by Twigg, Gui de Chauliac had written:

The plague began in January. During the first two months the disease consisted mainly of constant fever and blood spitting of which the sick died in three days. For the next five months, in addition to constant fever, there were external carbuncles, or buboes, under the arm or in the groin and this ran its course in five days. He observed that the contagion was so great, especially when there was blood spitting, that not only by remaining with the sick but even by looking at them people seemed to take it.[31]

Twigg informs us that this account has "some" likeness to "true plague," but this disease behaved differently further north in Burgundy and Normandy.[32] Here it appears plausible that Gui de Chauliac is describing primary pneumonic plague in January and February of 1348, and in the next five months arrival of septicemic and bubonic plague with occasional secondary pneumonic plague.

Twigg provides analysis of Michael of Piazza in Sicily from 1347, where the disease was believed to have first spread to the mainland of Europe. According to Piazza:

He said that the arrivals bore in their bones a disease so virulent that even those who only spoke to them contract the infection which was invariably fatal, the disease spreading to all who had any intercourse with the infected. Those who became infected had severe pain throughout their body and then there developed on their thighs or upper arms a boil about the size of a lentil which the people called "burn boil" (antrachi). This so infected the body that the patient violently vomited blood, and vomiting lasting without pause for three days at the end of which time there being no means of healing it, the patient died.[33]

Here it is evident that Cohn followed Twigg in believing that variety, read as inconsistency in chronicled skin manifestations, provides unmitigated evidence against bubonic plague during the Black Death. Twigg went on to convince himself that the Black Death was caused by Bacillus anthracis, the etiological agent for anthrax. He provides minimal evidence to support his view that symptoms and pathology of anthrax coincide with the chronicles. Norman Cantor apparently reached the same conclusion in a highly idiosyncratic book devoid of formal citations.[34]

Immunity and Nutrition

The basis for Samuel Cohn's judgment that *Yersinia pestis* did not cause the Black Death stems from his belief that bubonic plague fails to produce natural immunity in human hosts.[35] According to leading contemporary authorities in the field of *Yersinia pestis* research, natural immunity is functional, if not always effective, in persons infected with *Yersinia pestis*. Natural immunity with bubonic plague infection is confirmed by a present day survival rate of between 20-50%.[36] Admittedly the term "natural immunity" is confusing, but it is used only to distinguish between artificial immunity such as vaccines, or inoculation with antiserum.[37] Natural immunity exists in humans to any foreign chemical or cell entering the body in the form of antibody or cell mediated immunity. The body always reacts immunologically to foreign cells like *Yersinia pestis* bacteria, but there is no promise that antibody and cellular reactions will be swift and voluminous enough to prevent death. Interestingly, the bacterial genus Salmonella causes common gastrointestinal infections from fecal contaminated foods or water, and provides some measure of protection from bubonic plague as a result of cross reacting antibodies.[38] It has been challenging for immunologists to develop a vaccine conferring immunity to bubonic plague that offers protection for more than two years. Cohn confuses this fact with lack of natural immunity to *Yersinia pestis*.[39] Vaccine development and manufacturing are a matter of technical immunochemical complexity well beyond the issue of etiology, treatment, and prevention of *Yersinia pestis* infection. Clearly, there is no current substitute for having survived *Yersinia pestis* infection where lifelong immunity is conferred.

Perhaps some of the confusion of Cohn stems from his use of Robert Gottfried who explained immunity in "*The Black Death; Natural and Human Disaster in Medieval Europe*." Gottfried offers his version of immunology:

Disease for which there was immunity had less of an impact on medieval Europe than did more complex, multiple infections such as dysentery, influenza, and plague, for which immunity is quite limited, if it exists at all.[40]

This sentence stipulates the human body generally has no immunity to dysentery, influenza, or plague, which is patently false. Cohn also states that humans have no natural immunity to *Helicobacter pylori*.[41] This organism is a minor resident of the small intestine and is sometimes pathogenic in the stomach. Once again, the transient or pathogenic existence of *H. pylori* in humans does not infer absence of natural immunity.

Specialized white blood cells, like the antigen driven clonally selected T and B cells, are produced in significant enough quantities to confer life-long immunity for plague survivors. Cohn explains how natural immunity develops during plague epidemics that followed the Black Death and then contradicts by saying there was no natural immunity in humans to *Yersinia pestis* after modern epidemics in India. Cohn misconstrues late 19th and early 20th century medical texts, reports and commissions like *Manson's Tropical Diseases* from 1898. Cohn falters with his understanding of current immunological and epidemiological principles. Natural immunity existed after the Black Death and still exists in modern cases of bubonic plague.

There are several parameters to consider in terms of immunity to *Yersinia* infection. Cohn correctly enunciates these in context of modulation of *Yersinia* virulence, long-term survival issues dependent upon enzootic hosts, and human genetic predisposition. However, the lower population mortality rates with modern bubonic plague should not be confused with individual mortality rates. Population mortality rates include people not infected with *Yersinia pestis*. In contrast, individual mortality rates involve only those infected with *Yersinia pestis*. Cohn and others provide significant evidence that Black Death population mortality rates may have reached 60 percent in some cities in Europe. Cohn fails to estimate unique individual mortality rates during the Black Death that may have averaged 85 percent due to prevalence of septicemic, primary and secondary pneumonic plague.

The cyclical nature of infectious disease is associated with extent of malnutrition, whether by starvation, hunger, or poor nutrient intake. The biological war of pathogenesis versus human immunity often boils down to a fight over possession of iron. Nearly all organisms need iron used to power cellular energy motors sustained within mitochondria. Stephen Ell examines whether higher levels of blood iron in men possibly made them more susceptible to infections with *Yersinia pestis* during the Black Death.[42] While there is solid research evidence showing that high iron levels make people more susceptible to infections,

high levels only occur in people taking iron supplements, with red wine alcoholics, or in cases of genetic disorder called hemochromatosis. High iron levels, therefore, are generally irrelevant when considering medieval immunity. Conversely, Ell considers whether pre-menopausal women with very low serum iron values, might be less susceptible to plague infections than men. Research with *Yersinia pestis* yielded one of the first instances where iron availability influenced the severity of infection and disease.[43] Few microbiologists working with *Yersinia pestis* would concur that this exceptional vascular pathogen would fail to kill the malnourished.[44] Lowered immune status would simply override any consideration for decreased saturated transferrin levels. Perhaps it should be emphasized that iron is an important cofactor in certain immunochemical reactions. Therefore, in all likelihood, septicemic plague and secondary pneumonic plague would be more common in malnourished people, where bone marrow is easily accessible for "mining" iron. In any case, Horrox has noted the poor probably died in greater number due to increased contacts with rats and their fleas than because they were malnourished.[45]

Contagion

The Black Death generated 281 treatises offering abundant information about the cause of pestilence during the fourteenth century.[46] Many plague tractates suggest a "heavenly" cause in connection with a triple conjunction of Mars, Jupiter and Saturn that took place on March 24, 1345.[47] The mid-fourteenth century Florentine chronicler, Matteo Villani, was one of the first to offer how the Black Death had been transmitted in an "earthly" manner. Villani suggested infection was spread by the healthy mixing or possibly when conversing with the sick.[48] Tommaso del Garbo, a famous Florentine medical practitioner, tried to explain the speed of disease spread by informing us, "The pestiferous infection is caught by the sight and by the touch."[49] In an account of the dangers of corrupted air based on the teachings of Avicenna, the following excerpt is from Jacobus, a royal and papal physician and Chancellor of Montpellier translated by Bengt Knutsson:

I say that pestilence sores are contagious because of infectious humours, and the reek or smoke of such sores is venomous and corrupts the air. And therefore one should flee such persons as are infected. In pestilence time nobody should stand in a great press of people because some man among them may be infected. Therefore wise physicians visiting sick folk stand far from the patient, holding their face towards the door or window, and so should the servants of sick folk stand. Also it is good for a patient to change his chamber every day and often to have the windows open against the north and east and to spar the windows against the south. For so thy wind has two causes of putrefaction. The first is that it makes a man, whether whole or sick, feeble in his body. The second cause is written in Aphorism chapter 3, the south wind grieves the body and hurts the heart because it opens man's pores and enters into the heart.[50]

It is evident with Villani, Jacobus, and del Garbo, the concept of contagion was incubating in the minds of mid-fourteenth century natural philosophers. Ibn al Khatib had defied Muslim beliefs by arguing that this disease was spread directly from person to person during the Black Death.[51]

Based on population mortality rates, *Yersinia pestis* was transmitted to humans more frequently during the Black Death. It has been determined that bubonic plague is a zoonotic disease primarily affecting rodents where humans play an unfortunate role in the long-term survival of *Yersinia pestis*. However, bubonic plague has long been enigmatic due to a quiescent period in one species, only suddenly to create an epizootic in another mammal. *Yersinia pestis* is significantly "transmission adaptable" as evidenced by its presence in many different mammals.

The frequency of transmission is dependent on the concentration of bacteria in bodily fluids of the host. As mentioned previously, *Yersinia pestis* is strongly suspected of having mutated and thereby becoming more virulent. Therefore, the hallmark of bubonic plague during the Black Death was unprecedented levels of *Yersinia pestis* in the body fluids of those infected. Microbiologists have found a unique strain of Yersinia that is believed to have evolved during medieval times called mediaevalis ribotype O.[52] Differing strains of *Yersinia pestis* are thought to have caused ancient and modern epidemics. Although more research needs to be performed in demonstrating the precise virulence factors, it is not a stretch to presuppose increased virulence from a mutant strain during the Black Death.

The current category for mode of transmission with *Yersinia pestis* is referred to as vector-mechanical.[53] It takes place when an infected oriental rat flea named Xenopsylla cheopis bites a human host when the normal Rattus rattus host dies. Relative incidence of transmission rate from 1970 to 1994 in the United States reveals 66.9 percent transmission from flea inoculation, 16.1 percent from direct contact with infected animals, 2.2 percent from the respiratory droplet route, and 14 percent undetermined.[54] From this modern data, where animals are supposed to be safely skinned while wearing protective gloves, it is likely that minor abrasions or cuts on the hands created an important route for contamination from animals with unapparent infections during medieval times. This may have been a trigger point for sudden increased rates of infection during the Black Death.

The standard modern explanation for the sudden outbreak and rapid wavelike spread during the Black Death begins with large numbers of Yersinia carrier rats leaving ships at ports throughout Europe, and scattering throughout the continent.[55] These rats had arrived from extended Asian-European and North African maritime trade networks. Another view is that these rats gradually started arriving beginning in the late thirteenth century and set up endemic conditions in rural rats which then suddenly ran into all the cities after earthquakes or floods. With either approach, bubonic plague is not limited by climate. Since 1979, ten countries located on every continent except Australia and Europe reported over 200 plague cases, and all localities were tropical with exception of the United States.[56] Plague infected animals have been detected as far north as Canada and as far south as Mexico in North America.[57]

It is generally accepted there was ample time for *Yersinia pestis* to move from murine phase to sylvatic phase prior to the Black Death. However, there is rarely emphasis on the role of a wide range of hunted mammals. The principle candidate mammals in Europe include squirrels, rabbits, hares, gerbils, voles, chipmunks, and marmots.[58] There are two hundred different mammals that can transmit *Yersinia pestis* to humans from 31 identified species of fleas.[59] The rapid dissemination of Yersinia in animal populations during a sylvatic phase probably involved gerbils and squirrels.[60] It is surprising that no one has developed ideas about the possibility of a widespread urban endemic phase involving domestic animals in port cities. Cohn offers two reports during the Black Death of suspected transmission from clothing and linens, but never seriously considers the extent of how indirect contact with dried biological fluids containing *Yersinia pestis* may have contributed to the rate, frequency and incidence of the disease.[61] The mode of transmission during medieval times likely included unprecedented levels of indirect contact with dried infected biological fluids found in linens, clothing, cooking, eating, and drinking utensils, and in human or animal corpses. Dried biological fluids, contained high concentrations of *Yersinia pestis*, can remain viable for weeks in dried sputum, flea feces, and for months in the soil of rodent burrows.[62] It is unfathomable that Twigg dismissed the idea that a human corpse could deliver bubonic plague.[63] Another indirect contact source would have been iatrogenic, from contaminated medical cauterizing tools used in blood-letting prior to the development of sterilization and aseptic technique.

Twigg dismisses the argument that natural catastrophic happenings such as floods and earthquakes may have contributed to the Black Death.[64] Besides celestial causes, earthquakes are described as contributors to the spread of disease in the plague tractates although "usually regarded as inferior to the first [celestial] and probably dependent upon them."[65] It seems plausible that earthquakes and floods were factors in spreading the epidemic. In India in 1994, floods in Surat disrupted municipal services and led to higher rodent populations and bubonic plague due to sanitation collapse, while in Latur earthquakes crumbled houses storing grains that may have caused an influx of enzootic sylvatic plague animals into urban animal areas.[66] Cohn seems estranged from the mechanical insect phenomenon of incremental jumping and climbing. Grahm Twigg describes how oriental fleas travel over fifty feet from rats, and can leap four inches and walk eight inches vertically. Although normally unable to carry as much *Yersinia pestis* as the oriental flea, the cosmopolitan Pulex irritans can jump 13 inches and is capable of taking its meal from virtually any mammal.[67] With a new virulent strain of *Yersinia pestis*, bacterial concentrations were high enough in the blood stream from increased incidence of bacteremic bubonic and septicemic plague, to significantly increase the rate of infectivity with Pulex. Twigg suggests that Nesophyllus faciatus prefers cold temperatures and was responsible for transmission in northern Europe.[68] Pulex irritans primarily existed indoors where there were domestic pets that remain less affected by temperature. Domestic cats maintain an existence relatively nearer to rats and mice, and could have been a factor in transmitting *Yersinia pestis* to humans often enough to perpetuate a sustained epidemic among humans. The role of Pulex transmission

would surely have been facilitated by war, which forced peasants and animals into cities causing overcrowded and unhygienic conditions.

Graham Twigg was perhaps the first to consider that disease spread through Europe too fast to be bubonic plague by comparison to the slow rate in modern epidemics.[69] He and others have underscored the absence of rat epizootics in writings by fourteenth century chroniclers. The point perhaps overlooked is that a rat epizootic may be unnecessary where there is a high concentration of diverse enzootic animals in urban areas coupled with a virulent new strain of *Yersinia pestis* transmitted by Pulex irritans. Epizootics in domestic pets or urban animals would have been largely undocumented if sufficiently in advance of 1347. Scott and Duncan's sophisticated demographic and epidemiological analysis of Black Death provides key evidence favoring airborne spread of disease. They posit a minimum 22-day incubation period for an Ebola-like virus with an exceptional mortality rate they call "hemorrhagic plague."[70] Scott and Duncan believe the 40-day plague quarantine legislated over the next four hundred years in Italian city-states support their view of a long-term incubation period. Interestingly, they also refer to the skin manifestations as "tokens" in purple and blue, but not black.[71] The authors believe the disease was endogenous and oscillating around in France where it gave way to exogenous expression every eleven years on average from 1350 to 1400.[72] They point to the improbability of rat transmission in Greenland and Norway where there was substantial mortality rates. In their conclusion, Scott and Duncan yield to the evidence for bubonic plague in southern Europe, but not in northern Europe where they maintain the notion of an Ebola-like hemorrhagic plague.[73]

While Cohn fails to acknowledge the variety of symptoms due to the different types of plague involving *Yersinia pestis*, and the variety of ways the disease may be transmitted, Scott and Duncan underplay the possible enzootic or epizootic status of mammals other than rats and focus only on the oriental flea. There is much to agree with regarding their emphasis on a pathogenic coupling dependent on high rates of transmission with virulence.[74] It is difficult to understand their thesis that influenza can spread sporadically across Europe with the same rate of speed as bubonic plague during the Black Death, but it cannot be bubonic plague because primary pneumonic plague fails rapid infectivity in modern times. Pneumonic plague was 4 percent of all cases in the Manchurian bubonic plague epidemic from 1910-1911 while only 5 percent of all cases were septicemic plague.[75] However, septicemic plague has been as high as 25 percent of all cases in New Mexico from 1980 to 1984.[76] The argument here is that an especially virulent strain of *Yersinia pestis* infected immunologically niave populations. Under these conditions, it is very likely that septicemic plague progressed to secondary pneumonic plague frequently during the Black Death and was easily transmitted within the "one meter respiratory droplet route" during the Black Death. The chroniclers often describe the exodus of people from cities an easy way to spread respiratory droplet pneumonic plague beyond the initial locale. Especially during the summer primary pneumonic plague may have been more prevalent because of the increased activity of shipping, more fairs, and open markets, while in winter, crowded housing conditions perpetuated infection.

Another point being underestimated regarding the rapid spread across the continents is that domestic cats suffer from bubonic plague and 92 percent have been reported with high levels of Yersinia in the oral cavity.[77] Infected cat saliva has been used to explain the high rate of pneumonic plague transmission in some modern epidemics.

The work of historians like Cohn and Horrox show much difference of interpretation. Cohn largely misconstrues the chronicles and plague tractates with incomplete understanding of modern clinical symptoms, immunology and nutrition. Rosemary Horrox used much of the same information to develop an appreciable understanding of the cause of Black Death. Biologists Twigg, Scott and Duncan, provide valuable insight into the cause of Black Death. Twigg reaches a conclusion that anthrax caused the Black Death, but his detailed explanations about animals and fleas paradoxically support the idea of bubonic plague. For example, Twigg refutes the idea of bubonic plague in England, but offers Nesophyllus faciatus as a transmitter in cold weather. Twigg accepts that bubonic plague cannot be transmitted by the respiratory route in freezing weather, but then accepts pneumonic plague in Manchurian winter.

Scott and Duncan's work is sophisticated in the analytic sense. With the Black Death, they emphasize the 40-day quarantine established in Italian city-states supporting their calculations for a 22-day incubation

period in England.[78] This is considered strong evidence against the possibility of bubonic plague as it has an average 3-day incubation period. However, moderate cases of bubonic plague have incubation periods up to fifteen days with symptoms lasting up to six days before mortality.[79] Twigg also reported that fleas can be infective for 14 days after the rat or other mammal dies if temperature and humidity are optimal, and remain infective for up to six weeks. Scott and Duncan do not address these and other irregularities surrounding bubonic plague epidemics.

Alternative explanations for the cause of Black Death have been evaluated starting with Twigg in 1984. J.F.D. Shrewsbury was the first microbiologist in 1970 to find weakness in the theory of bubonic plague causality during winter, proposing that endemic typhus was responsible.[80] Shrewsbury could not understand how bubonic plague was perpetuated in the winter months, positing an important role for endemic typhus. The problem with his thesis from the standpoint of clinical symptoms is that endemic typhus produces small red spots all over the skin and no buboes.

In 1979, astrophysicist Sir Fred Hoyle and his colleagues in South Wales, speculated that viruses were randomly scattered from the tails of abundant comets roaming the galaxy, and that these are wafted onto the Earth in an electromagnetic stream known as the solar wind.[81] They offer that life on Earth was started by these viruses and epidemics perpetuated by the same process. Hoyle and his colleagues fail to explain how such biological material would avoid destruction when exposed to ultraviolet light found in solar radiation. In 1992, epidemiologist R. Edgar Hope-Simpson organized an old idea into a new model for how epidemic influenza spreads so rapidly.[82] Hope-Simpson finds that influenza can exist in a latent incomplete particle form. If latency exists with influenza virus it might have implications for an enhanced way of spreading primary pneumonic plague during the Black Death.

Conclusion

In light of comparisons in modern clinical descriptions of bubonic plague, it is evident that there are differences among experts describing symptoms of plague. A comparative study of modern clinical symptoms helps to clarify which of three variations on bubonic plague people had during the Black Death. The types of skin manifestations described in chronicles and plague tractates supports bubonic plague as the cause of Black Death. Dark subcutaneous hemorrhages and gangrene variously described at the time of the Black Death support the idea held by F.A.Gasquet and others for the origin of the name Black Death. Only a few of 281 plague tractates have been examined here, but classic descriptions of symptoms available favor septicemic, secondary, or primary pneumonic plague consistent with an extremely virulent and perhaps new strain of *Yersinia pestis*.

In the plague dialectics of Twigg, Cohn, Scott and Duncan, there is lack of emphasis for: 1.) The impact of a new virulent mutant strain of *Yersinia pestis* in a naive immune population; 2.) The cosmopolitan role of Pulex irritans in conjunction with unusually high blood bacterial counts; 3.) Descriptions of skin manifestations in plague tractates and chronicles consistent with frequent cases of septicemic, primary, and secondary pneumonic plague in humans; 4.) Emphasis on the possibility of an endemic urban mammalian carrier population; 5.) The rate of transmission in hunters who handled infected animals; and 6.) The rate of transmission during contact with dried fluids from deceased animals and humans. The remarkable rate of speed that bubonic plague spread across Europe during the Black Death and in subsequent pandemics is more understandable by combining these factors.

For modern day historians, the cause of Black Death is an exceptional interdisciplinary challenge. The magnitude of this mid-thirteenth century pandemic called for a multitude of rationalizations about the cause and treatment for a disease that took three different forms. Only by including information gathered by various medical historians is it possible to fully appreciate the nature of the Black Death as bubonic plague. Medical practitioners and people who lived and frequently died during the Black Death were armed with no practical knowledge about the causes of disease and modes of transmission. Their unremitting response involved scores of frustrated approaches against the continuous barrage of infection with *Yersinia pestis*.

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