

The History of the Twentieth Century

Episode 171

“Pale Horse, Pale Rider I”

Transcript

[music: Fanfare]

During the winter of 412-11 BC, the Greek city of Perinthos, a port on the Sea of Marmara, experienced a wave of illness. Hundreds of Perinthians fell ill with high fevers, coughing, sore throats, aches, and in some cases more severe symptoms.

We know this because a physician recorded these facts and his record of the incident survives to our time. The so-called “Cough of Perinthus” is historically significant for three reasons. First, the physician who recorded the event was Hippocrates, one of the greatest names in the history of medicine. Second, Hippocrates used the word “epidemic” to describe the event, a term that literally means “on the people.” It was already in use in the Greek language, applied to various phenomena, but Hippocrates was the first to use it to describe the outbreak of a disease, this one, and we primarily use the word in this Hippocratic sense in our time.

Third, although we cannot say with certainty, this “epidemic” just might have been, might have been, the first recorded outbreak of the disease we know today as “influenza.”

Welcome to *The History of the Twentieth Century*.

[music: Opening Theme]

Episode 171. Pale Horse, Pale Rider. Part one.

A virus is a peculiar thing.

It is nothing more than a very large and complex molecule. Your body and mine are structures built from an assortment of very large and complex molecules, some of which are larger and more complex than a virus. But a virus is a particular kind of molecule with some very strange properties.

For example, it can collect material from its environment and use this material to build a protective coating of proteins around itself, called a “capsid.” Now, I have to be careful about my

language here. Verbs like “collect” and “build” imply action, will, intention. A virus has no will. It does not think. It does not take action. The physical structure of the virus itself is what causes the capsid to assemble.

But the strangest property of a virus is that it reproduces. You might think this means it is alive, although in fact it isn't, at least not in the way we usually mean. A virus doesn't eat. It doesn't grow. It doesn't expend energy. What it does do is penetrate a living cell and co-opt the cell's own reproductive mechanism, which it then uses to make copies of itself. This process typically kills the host cell.

Again, I have to be careful with my language here. Words like “penetrate,” “co-opt,” and “use” imply a will, an action, but there is no will and no action. These things happen because they are built into the structure of the molecule. I struggle here because the English language lacks a proper vocabulary for describing a virus. Viruses are just too far outside our everyday experience.

Viruses also evolve. As they copy themselves, small changes may appear in the structure of the molecule. Sometimes these changes impair the ability of the virus to reproduce further. Occasionally, they increase the chances of successful reproduction. One way this can happen is if the changed structure of the virus helps it elude the immune system of the host organism.

Why are there viruses? No one knows the answer to that question, though there are speculations. A virus is dependent on its host to reproduce. It also depends on its host to move about and spread to other individuals. The implication of this is that the most “successful” virus—if you can pass judgment on a molecule using human terminology—the most successful virus is one that reproduces and spreads without incapacitating its host. That virus will reproduce in greater numbers and spread itself over a larger area.

The influenza virus likely appeared first in birds. Water birds, in particular. This conjecture is based on the fact that the virus doesn't make birds very sick, suggesting that the birds and the virus have had a long time to adapt to one another.

About 10,000 BC, human beings began to practice agriculture, including the domestication and cultivation of livestock. Agriculture allowed the development of larger human communities, into the thousands or tens of thousands. With this development came a number of new and terrible diseases, illnesses that can make a human being very sick or even kill, and because humans now live close together, the diseases spread rapidly in a short time. The advantage that a virus or other pathogen used to enjoy by not making its host too sick disappears. New diseases emerged, many of them specific to human beings, diseases that were serious, even lethal, but could spread from person to person fast enough to stay ahead of the sickness and death they left in their wake. Smallpox. Tuberculosis. Measles. And influenza.

Influenza probably made the jump from birds to people when early humans began raising ducks. As it happens, pigs are a useful intermediary. The cells in their respiratory linings resemble bird cells in some respects and human cells in other respects, making them a convenient pathway for the influenza virus to travel as it mutates from an avian pathogen to a human pathogen.

Places where birds, especially ducks, and hogs and humans all live together in close contact are therefore an ideal environment to generate new strains of human influenza. Such environments have been common since 10,000 BC, and they continue to exist even in our time. We call them “farms.” New forms of influenza are constantly appearing, which is why getting sick from influenza once does not confer lifetime immunity in the same way that, say, getting sick from measles does.

Hippocrates believed that illnesses were caused by an imbalance of humors in the body. This was an idea that persisted into the 18th century. Other ancient physicians in the Greco-Roman world and in China speculated that miasma, bad air, could cause sickness. Fog, mist, smoke, or dirty or bad-smelling air. These were all implicated as sources of disease. Consider for example the disease malaria. Its name is derived from the Italian words for “bad air,” illustrating what was once believed to be the cause of the illness. This miasma theory would persist into the 19th century.

In medieval Europe, disease was often attributed to supernatural forces, such as demons, witchcraft, or magic. Epidemics were sometimes thought to be divine punishment for sinful behavior. In the 14th century, some Italians began to believe that influenza was caused by malign astrological influences, hence the word *influenza*, which is simply the Italian word for “influence.” It worked its way into the English language as the name for the disease in the 18th century, during that time when the English thought everything Italian was oh, so cool. The name is often shortened to “flu.” It is known as *la grippe* in French and *die Grippe* in German. My Pennsylvania Dutch father always called it “the grippe.” I think I was a teenager before I first heard the word “influenza.”

In the years following the discovery of the New World, the native population of the Americas suffered multiple epidemics of new diseases brought by Europeans that the natives had no prior immunity to or experience with, including influenza, which led to a great loss of life and a precipitous decline in the population. No one knows for sure what the numbers were, but in our day there is speculation that the rapid population loss led to reforestation in large areas of the New World which in turn led to a drop in atmospheric carbon dioxide which may have caused the phenomenon known as the Little Ice Age that began at this time and lasted until the numbers of humans living in the Americas recovered in the 18th and 19th centuries.

In 1676, the Dutch draper and part-time lensmaker Antonie van Leeuwenhoek first observed what he called “animalcules” through a microscope of his own manufacture. “Animalcule” is simply a word that means “tiny animals.” Today we more often call them “micro-organisms.”

Van Leeuwenhoek and those who followed him soon realized that the world around us is teeming with these tiny animals, but micro-organisms were little more than a scientific curiosity for the next 150 years. By the early 19th century, though, biologists began to come around to the view that micro-organisms might be the cause of disease. This insight is called the germ theory of disease and it is usually ascribed to the French biologist Louis Pasteur, although to be fair, he didn't think it up on his own; he built on the work of his predecessors in the field.

But the germ theory was one of the greatest scientific breakthroughs in a century replete with great scientific breakthroughs. Germ theory revolutionized food safety as in, ahem, pasteurization. It also revolutionized medicine. Doctors didn't have many tools for curing or treating infectious diseases in the 19th century, but the germ theory explained how diseases were transmitted, and by disrupting that process by which diseases spread, doctors could hope to nip an outbreak of disease in the bud, before it could become an epidemic, and thereby save many lives.

A classic example is the work of the English physician John Snow—no, not *that* Jon Snow. In the 1850s, London was experiencing repeated outbreaks of cholera. Now, the population of London was growing rapidly at that time, owing to the Industrial Revolution, and the Thames River was little more than an open sewer, so noxious that its smell plagued the city. In the summer of 1858, the weather became so hot and the stench became so thick that the event went down in history as “The Great Stink.”

Many physicians at the time believed the bad smell coming off the river was the cause of cholera in the city. But in 1854, during a particular outbreak of cholera in the Soho district, John Snow visited the neighborhood, walked around, talked to some residents, and discovered that the victims were all drawing water from the same public water pump. Snow tipped off the local authorities, who disabled the pump by removing its handle. The outbreak ended, and Snow had proved that cholera was caused by contaminated water, and not by anything in the air. Snow suspected a micro-organism, although microscopic examination of the pump water at the time failed to come up with the culprit. Still, this incident became a classic demonstration of how germ theory saves lives.

Similarly, in 1900, the US Army doctor Walter Reed and his team confirmed the theory that yellow fever was transmitted by mosquito bite. This insight made possible, among other things, the completion of the Panama Canal project.

Although Louis Pasteur's work was immensely valuable and saved many lives, his greatest frustration was his inability to identify the microbe that caused rabies. Pasteur suggested that perhaps it was something too small to be visible in a microscope. In 1884, a colleague of Pasteur's named Charles Chamberland developed a porcelain filter with holes in it so small that they could filter out bacteria. Eight years later, in 1892, a Russian biologist named Dmitri Ivanovsky was studying an infection known as “tobacco mosaic disease.” It affected tobacco

plants. It was known to be a disease, as it had already been demonstrated that juice from an infected tobacco plant could transmit the infection to a healthy tobacco plant, but Ivanovsky was the first to try filtering tobacco plant juice through one of these porcelain filters. He discovered that the juice was still infectious even after the filtering, which implied that the infectious agent, whatever it was, was much smaller than a bacterium. In 1898, a Dutch biologist was the first to use the word “virus,” from the Latin word for “poison” or “venom,” as the name for these unknown, super-tiny infectious agents.

And that was the state of medical science at the time of the Great War. The germ theory was now accepted as fact and had proved valuable in curbing the transmission of diseases, but viruses were merely a laboratory curiosity, a scientific oddity that might or might not have any real world significance.

You probably already know that in the age of large-scale armies that began in the late 18th century, it was common for these armies to suffer more casualties from disease than from enemy action. So one of the first places where germ theory was employed was in military medicine. Casualties from disease declined steadily in the late 19th and early twentieth centuries. During this same period, weapons technology advanced considerably, as we have already examined at length in this podcast, and thus casualties from enemy action were steadily increasing. By the Great War, the two trend lines have crossed, and casualties from disease are now lower than casualties from enemy action. So, yay, science! I...guess...?

On the other hand, the Great War also brought together greater numbers of human beings, packed tighter together and shipped around to more places all over the planet, than had ever been seen before. These were the perfect conditions for a new epidemic.

[music: Bach, *Brandenburg Concerto No. 4 in G*]

In early March 1918, a dust storm kicked up in eastern Kansas. It obscured the sun and stung the noses and throats of the US Army recruits undergoing basic training at Camp Funston, part of Fort Riley. Shortly after the dust storm ended, a company cook named Albert Gitchell spent an uncomfortable night. The following Monday morning, March 4, he didn't feel up to preparing breakfast for the soldiers in his company. He went to the infirmary instead, complaining of a sore throat, fever, and aches and pains.

None of the workers in the infirmary saw anything unusual in this, until a second soldier appeared just minutes later, complaining of the same symptoms. By lunchtime, the infirmary had 107 cases on its hands. By Friday, they were up to 522, and hospital tents had to be put up to accommodate all these patients. The following weeks saw hundreds more cases and 46 deaths. The post surgeon diagnosed the disease as influenza. Soon thousands of American soldiers who were at Camp Funston during this outbreak were in France. It was the beginning of an epidemic that would sweep the world.

Albert Gitchell was probably not “patient zero,” to use the modern term. He is merely the first recorded case known to us today, but it seems unlikely he was the first human being on the planet to become infected. The US Army post at Camp Funston does not seem a likely place for the emergence of a new influenza virus.

No one can say for sure where the virus did in fact first emerge, but in our time there are three competing hypotheses that seem at least plausible. Since there’s no way of knowing which of them, if any, is correct, I’m going to go ahead and give you all three.

The first hypothesis places the origin in Kansas, in the United States, and before I go on with this, I have a confession to make. I am an American myself, and when I first encountered a theory that the United States was the proposed point of origin for the virus, I reacted to this very negatively. That can’t be right, I thought to myself. Epidemics don’t begin in my country; they come to my country from somewhere else.

Funny thing about that, I learned as I got further into my research that this is a very common reaction to an epidemic, and you will soon see this as we proceed with the narrative. There’s something in human nature that rebels at the thought that a disease might originate among your own people, in your own community. It is far more comfortable to believe that diseases come from somewhere else, that they are born among sketchy foreigners with questionable hygiene practices and they enter one’s own country through the borders, like an invading army.

This first hypothesis points to rural Haskell County, Kansas, which lies in the opposite corner of the state from Camp Funston. It is an extremely rural county, with the distinction of being the flattest county in Kansas, and that’s saying something. There’s a difference in elevation of less than one hundred feet from one end to the other. In 1918, Haskell County was an impoverished place. It was home to about 1,700 people spread across 578 square miles. Most of them still lived in houses made of sod that lacked modern amenities. They were farmers. They grew corn. And they raised pigs and chickens.

You know what that means.

A couple of weeks before the outbreak at Camp Funston, on February 21, the local newspaper in Haskell County reported that “[m]ost everybody over the country is having lagrippe or pneumonia.” That same issue of the paper also reported that “Dean Nilson surprised his friends by arriving at home from Camp Funston on five days furlough. Dean looks like soldier life agrees with him.” Twelve days later, the outbreak at Camp Funston began.

There was a doctor in Haskell County named Loring Miner. He was the only doctor in Haskell County. Dr. Miner was an educated man who besides medicine had an interest in ancient Greece and could and did read classical literature in the original Greek. His son followed in his footsteps and also became a physician. At this moment, he is serving in the US Navy.

There was at this time no official requirement that doctors report outbreaks of influenza, but Dr. Miner was sufficiently concerned about this particular outbreak—it was sudden and virulent, and it had killed a number of his patients. We don't know exactly how many, but even a few would have been an alarming number in a community as small as Haskell County. Anyway, Dr. Miner was sufficiently concerned to write up a report and submit it to *Public Health Reports*, a weekly publication of the US Public Health Service devoted to alerting doctors and health care professionals to the outbreak of new diseases.

So we know about the outbreak in tiny Haskell County because of the diligence of Dr. Miner. It occurred before the first reports out of Camp Funston, although by the time Dr. Miner's report was printed up and circulated, the outbreak at Camp Funston had already begun. In fact, it took until the early 21st century, almost a hundred years after the fact, before an investigator first noticed Dr. Miner's report and picked up on its implications.

So it may be that patient zero was some poor but honest, hardworking Methodist farmer in Haskell County, Kansas, who caught a new strain of influenza from one of his hogs in January 1918. Within weeks, some earnest and patriotic young farm boy would leave Haskell County, perhaps for the first time in his life, and carry the virus with him to basic training at Camp Funston.

On the other hand, was Haskell County really the origin of the virus, or was Dr. Miner merely more thorough than most of his colleagues in rural Kansas? Maybe the disease had already become established at Camp Funston but had not yet attracted official attention. Maybe soldiers on furlough brought the disease home to Haskell County. Maybe it reached Kansas by some other route.

Which brings us to the second hypothesis, which looks to China as the source of the new strain. China is a nation where, for a variety of cultural and culinary reasons, large numbers of birds and pigs and humans regularly come in contact with each other. In our day, China is usually the birthplace of new strains of influenza, but China was less distinctive in this way back in 1917. Not that Chinese agriculture was so different back then, just that there was more human contact with birds and pigs in other parts of the world.

China was a very large and very poor country at this time, and epidemics were all too frequent. In November 1917, the Chinese province of Shanxi experienced an outbreak of some kind of respiratory disease. In response, the Chinese government turned to its house expert on epidemics, the 38-year old physician, Wu Liande. Wu had been born to an ethnic Chinese family living in Penang, in Malaysia and had studied medicine at Emmanuel College, Cambridge, where he had a stellar academic career and in 1902 he became the first ethnic Chinese physician to earn his degree at that university. He continued his medical studies in Europe for a while before returning to Penang. His outspoken opposition to the opium trade got him in trouble with the powers that be in British Malaya, but also earned him the attention of Yuan Shikai, who was still a military

commander at this time. Yuan invited Wu to come to China to work at the Imperial Army Medical College and Wu accepted.

The decision to hire Wu proved more than sound in the winter of 1910, when there was an epidemic of some kind of respiratory infection in Manchuria. The Imperial government sent its most famous physician to investigate. Wu determined that the epidemic was in fact the pneumonic plague, a disease caused by the bacterium *Yersinia pestis*, which is also responsible for the better-known bubonic plague. He did this by performing an autopsy, which was very much taboo in traditional culture and actually a criminal offense in Imperial China, but the epidemic was killing people by the thousands, and Beijing was worried enough to grant Dr. Wu a special dispensation to go ahead and perform that autopsy. The fact that the corpse he was working on was of a Japanese patient, not a Chinese one, may have helped him to get that permission.

Wu was able to culture *Yersinia pestis* from tissue samples taken from the victim's lungs, so that settled that. What to do about it? Wu ordered strict quarantines, suspended most rail travel and, most controversially, ordered the remains of the plague victims to be cremated. In a Manchurian winter, the ground is frozen too hard for burial, so corpses were customarily preserved until spring. Cremation was another grave breach of traditional custom, but the Imperial government backed Wu on all these decisions. Sixty thousand people died in Manchuria during this epidemic, but Wu's drastic response limited the spread and prevented the disease from reaching the rest of China, perhaps saving millions.

The Qing Dynasty was overthrown and the Republic of China born the following year, but Wu's stock remained high, especially after his patron Yuan Shikai became President, and so it was that when the 1917 epidemic in Shanxi appeared, the government in Beijing sent China's now-even-more-famous doctor to investigate. As before, Wu did an autopsy on one of the victims. The people of Shanxi reacted badly to this and tried to set fire to the rail car Wu was using. He hightailed it back to the capital with his tissue samples and on January 12, 1918, he announced that he had again found *Yersinia pestis*, confirming that this was another outbreak of pneumonic plague.

So if it wasn't influenza, why am I telling you this? Well, Wu's diagnosis proved controversial. The mortality rate in this epidemic was under 50%, far less than the 99%+ death rate the 1910 plague in Manchuria had wrought. Other doctors and officials in Shanxi insisted Wu was wrong, and that the disease was something less severe than plague. They proposed that it was an unusually severe form of "winter sickness," that being the Chinese term for what we call "influenza."

Was this the origin of the 1918 influenza epidemic? It's possible. For most of the century between the epidemic and our day, this was the preferred hypothesis. It satisfies the need of people like me to blame the disease on people far away. On the other hand, accepting this

hypothesis requires that you dismiss China's pre-eminent physician and epidemiologist, Dr. Wu Liande, as a fraud or an incompetent.

On the other other hand, the fact that Chinese laborers were moving in large numbers through China to Shandong Province and from there across the oceans in packed ships, some across North America in equally close accommodations aboard railroad cars, and on to Europe provides us with an easy way to explain how a Chinese epidemic could become a global one.

If either of these hypotheses are correct—if patient zero was a poor but hardworking farmer or farmwife or son or daughter of a farm family, either in Haskell County, Kansas or Shanxi Province in the Republic of China, take your pick—then we cannot blame the Great War for the emergence of a new and more dangerous strain of the influenza virus. We would have to conclude that it came about naturally, although we can still blame the war for spreading it across the world.

But there is one more hypothesis to consider, and this one also is emerging only in the 21st century. This hypothesis points the finger directly at the Western Front, or, to be more precise, about 120 kilometers behind the Western Front. That's where you'll find the French fishing village of Ètaples-sur-mer, where the River Canche empties into the English Channel amid marshes and sand dunes tufted with sea grass. It lies about 60 kilometers south of Calais and the Strait of Dover. Its pre-war population of about 5,000 swelled to over 100,000 when the British Army made Ètaples its staging area for the shipment of soldiers, supplies, and equipment to the front. It was a port town with good rail connections and far enough behind the front to make it suitable. There also stood the hated "Bull Ring," as the Tommies called it, the exercise grounds where recruits newly arrived from Britain were put through a demanding two-week training regimen so grueling that the soldiers undergoing it looked forward to being sent on to the trenches. So hated was the Bull Ring that it sparked a brief mutiny in the fall of 1917.

Ètaples and the surrounding region hosted not only British Tommies and Colonial troops from every corner of the Empire but also German POWs, French Colonial soldiers, and no small number of Chinese laborers.

Ètaples also had a hospital. Multiple hospitals, actually, totaling some 23,000 beds in all, including a special hospital dubbed "Number Three Native Labour General Hospital," for the Chinese. During the second half of 1916 and the Battle of the Somme, dozens of trainloads of wounded soldiers were brought to these hospitals at Ètaples. Many of them had been victims of gas attacks and had damaged lungs and respiratory passages.

During the winter of 1916-17, a year before the outbreaks in China and Kansas, doctors observed the appearance of a peculiar lung disease among the patients at these hospitals. They called it "purulent bronchitis," and wrote up a report of it, which was published in the *Lancet* in the summer of 1917. By that time, other cases had emerged in British hospitals in France and in the barracks at Aldershot, in England, but they had receded with the coming of warm weather, as

influenza typically does, although no one knows for sure why influenza does that, not even in our day.

The British encampment at Étapes had hog pens, where pigs were brought and slaughtered to feed the troops. And the marshy, low-lying Channel coast is a haven for migratory sea birds, so we have here all the ingredients to produce a new strain of influenza, with the added twist of hospitals full of thousands of patients with damaged respiratory passages who may have provided a helpful breeding ground for new strains of the virus.

The Étapes hypothesis was first advanced in the early 21st century by the English virologist John Oxford. If this hypothesis is correct, then the Great War was very much a factor in the emergence of this new strain, and patient zero was probably some poor, twice-unlucky British or Colonial soldier, whose lungs were assaulted first by a German gas attack and soon after by a new strain of influenza virus.

But this hypothesis also comes with some unanswered questions. If the virus emerged in the winter of 1916-17, why did it take more than a year to develop into an epidemic? That could be because the British soldiers who were carrying the virus did not travel very far. Most of them traveled no farther than Aldershot to Étapes to the trenches and back again over this period. A more difficult question is why the disease doesn't seem to have spread to the civilian population, either in France or in England. British soldiers would have been fraternizing with their civilian neighbors in both countries, and so you would expect to see a spike in influenza cases in nearby communities, and there is no evidence of any such thing, at least none anyone has yet discovered.

So there you have it. Three hypotheses, each offering an attractive explanation, but each also raising questions that we can't answer, at least not as of the date I release this episode. But in our time, genetic analysis is becoming ever more sophisticated, and new evidence may someday emerge that will confirm one of these explanations and refute the others. Or perhaps new evidence will surprise everyone and lead the investigation in a wholly new direction.

But we'll have to stop there for today. Thank you for listening, and I'd like to thank Stephen for his donation, and thank you to John for becoming a patron of the podcast. Donors and patrons help keep the podcast going, for better or for worse, in sickness and in health, and if you have a few local currency units to spare, please consider becoming a donor or a patron. It's easy. Just visit the website, historyofthetwentiethcentury.com, for more information.

And I hope you'll join me next week, on *The History of the Twentieth Century*, because now that we've set the table, as it were, it's time to examine the impact of the influenza epidemic. Pale Horse, Pale Rider, part two. Next week, on *The History of the Twentieth Century*.

Oh, and one more thing. In 1918, when doctors barely knew what a virus was and had no means of detecting one, the medical community generally accepted that influenza was caused by a

bacterium. The presumed culprit had been identified by the German biologist Richard Pfeiffer during the last major influenza epidemic, in 1892. In 1918, it was called *Bacillus influenzae*, or sometimes Pfeiffer's bacillus, after its discoverer.

In our time, it is called *Haemophilus influenzae*, and we now know it to be an opportunistic pathogen, that is, the bacillus commonly lives in the noses and throats of many of us without causing symptoms, but it can multiply and become dangerous when its host's immune system is weakened, say by a virus, and it can cause illnesses such as ear, eye, or sinus infections, meningitis, or even pneumonia. The fact that many people, sick or well, carry the bacillus in their noses helps explain Pfeiffer's misunderstanding. It would not be until the 1930s that medical science would abandon Pfeiffer's explanation and come around to the suggestion that influenza was caused by a virus.

In the 1980s, a vaccine for one form of *Haemophilus influenzae*, type b, or "Hib" for short, was developed and is now routinely administered to children in many countries. Still, the word "influenza" remains in the organism's name, an historical reminder of an old medical misunderstanding.

[music: Closing Theme]