

1918 Influenza



SPANISH INFLUENZA TAKES HEAVY TOLL

In Fall 1918, Katherine Anne Porter, a reporter for the Rocky Mountain News in Denver, Colorado, contracted influenza. She was 28 years old. She was hospitalized for several months and nearly died. Her hair whitened as a result of her illness and it stayed that way for the rest of her life. A young army lieutenant with whom she was acquainted also caught the flu, but he did not survive. Her white hair was not the only lasting effect from her illness. The experience led her to write a book, nearly an autobiography, describing a young woman's flu illness and that of her fiancé, who cared for her and then sickened and died of the ailment. The book was "Pale Horse, Pale Rider," published in 1939.



Katherine Anne Porter

Miss Porter's experience was like that of millions around the world who contracted influenza in the worst pandemic in modern history. World-wide, it is estimated that about one third of the world's people contracted the illness and at least 50 million people died from it. About 675,000 died in the United States.

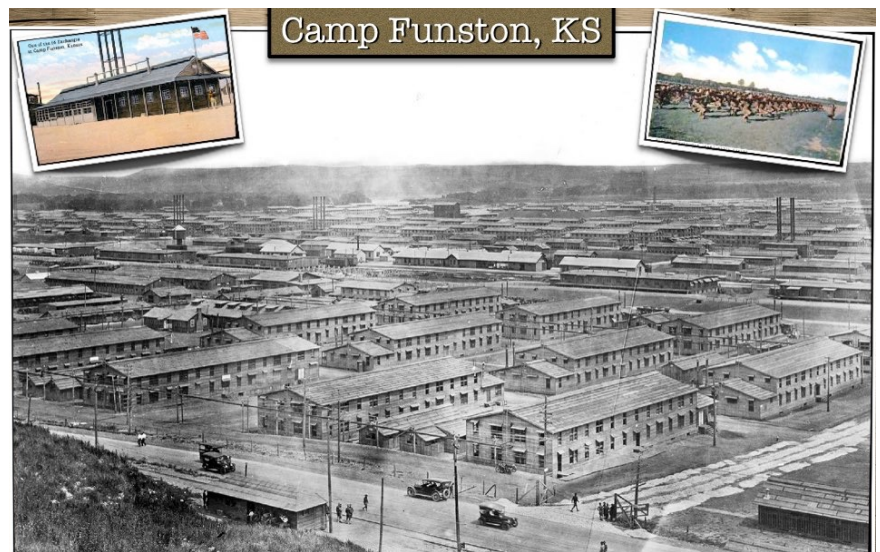
The origin of the influenza virus that caused the pandemic is unknown. It is a kind of virus found in birds and pigs, so one of these may have transmitted it to humans. The site of the first cases is also unknown. Though it came to be called the "Spanish flu," it did not start there. It was called that because, unlike the US and most western

European countries, Spain was not involved in World War I and so did not enact press censorship that prevented publication of news that would demoralize people. Consequently, the Spanish press was the first to report flu outbreaks, so other countries blamed the flu on them.

A site of early reports of severe flu-like deaths in the US was at Camp Funston in rural Kansas in the early spring of 1918. The militarization of the US population led to the spread of the illness to bases around the country and then to Europe. From there it spread world-wide.

In this first phase, the illness was somewhat more severe than the annual influenza we experience today. Some very violent cases were noted, but most were relatively mild. It was more contagious than annual flu, so it spread easier. It was also unusual in that it more often infected younger adults, in contrast to annual flu that affects older adults more often.

The number of new cases abated a bit by the end of summer, but a second wave rose in the US



“If the epidemic continues its mathematical rate of acceleration, civilization could easily disappear from the face of the earth.”

Dr. Victor Vaughn, in 1918 General Medical Board executive committee and Colonel, US Army.

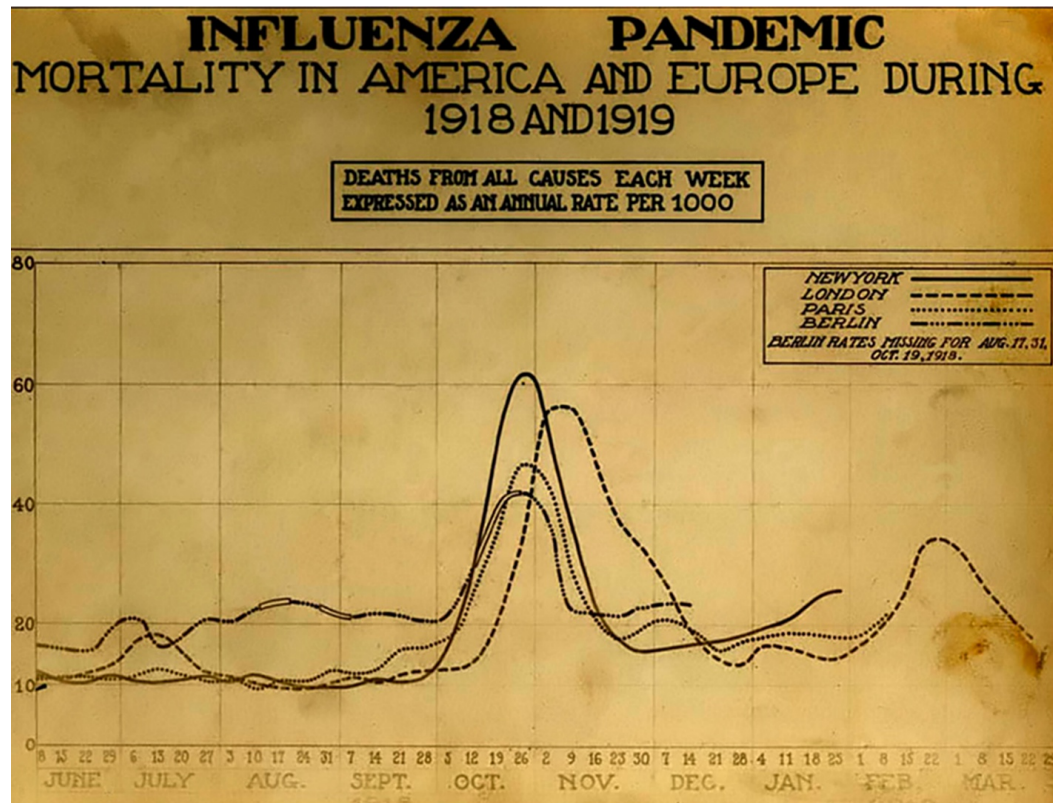
diminished in the US again, but a third, much smaller and less severe, wave appeared in the spring of 1919. Smaller outbreaks continued sporadically across the country into 1920.

On September 30, 1918, in the midst of the tragic second wave, the National Swine Show and Exhibition began in Cedar Rapids, Iowa. Within days, farmers in attendance began to find their pigs coming

down with an illness that passed among the pigs. It was an illness none had seen before. Hog producers were sensitive to the name "swine influenza" that some were calling it, but it turned out to be influenza. Later research showed that the virus in the pigs came from humans, so we had served as an intermediary vector between birds and pigs. Similar outbreaks of influenza in swine in Europe and Asia in 1918 were shown to be from humans and not international transmission among pigs.

and Europe in late August. Cases in this wave were frequently much more severe and the worst rates of death in the pandemic were achieved in the fall of 1918. This is when Miss Porter and her lieutenant friend became ill.

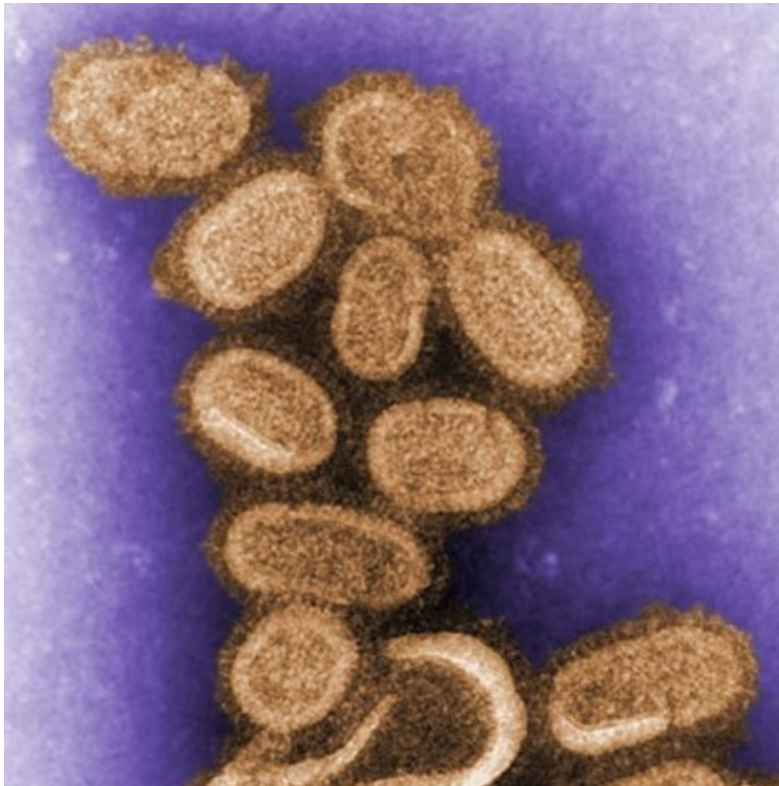
After the new year, cases



Influenza's Deadly Trail

The spread of influenza around the world was undoubtedly facilitated by World War I. After apparently arising in the US, it spread among military personnel there and then was carried to Europe. Troops were being transported between Australia and Europe, so it was likely introduced there afterwards. Britain was a colonial power at this time, so it had substantial trade with its colony India. Influenza was one inadvertent export. Trade with Europe and the US introduced it to Asia, Africa, and Latin America. In the West, it occurred in three waves, spring 1918, fall 1918 and winter-spring 1919. The second was the worst in the West. The third wave was worse in other parts of the world. It has been estimated that at least 50 million people died of pandemic-related illnesses worldwide.

How does Influenza affect the body?

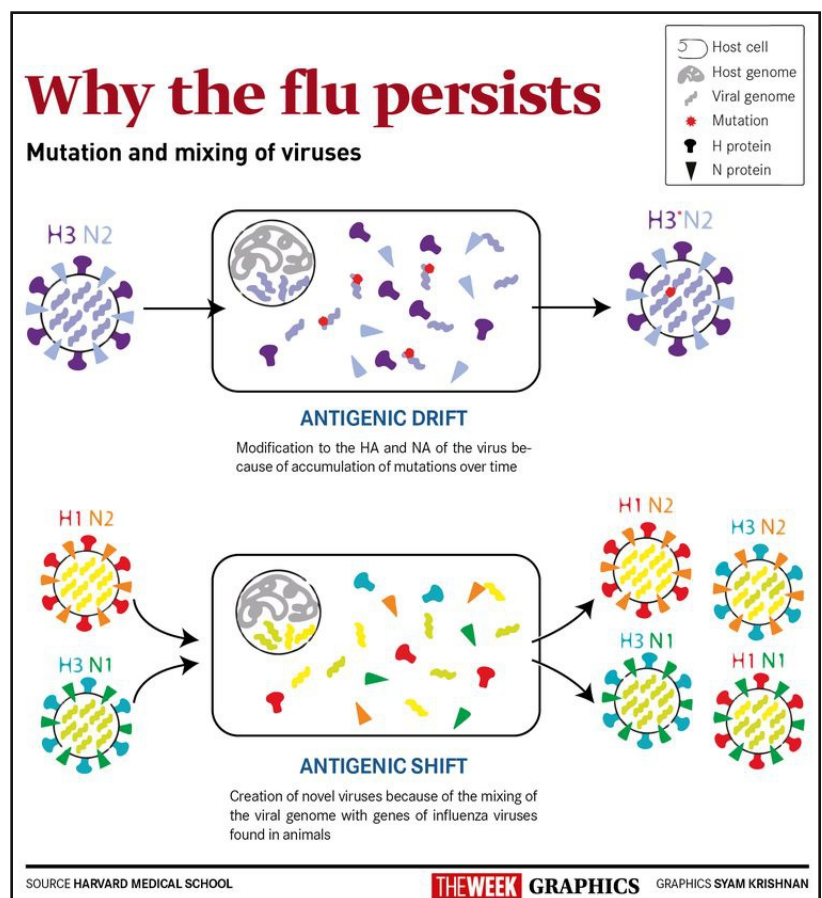


Influenza is caused by a virus, not a bacterium. Influenza viruses are microscopic packages containing the instructions to make more viruses. The package's envelope is composed of two protein molecules, one of which, the hemagglutinin (H) protein allows the virus particles to attach to the surfaces of lung cells inside the bodies of their hosts. The other protein, the neuraminidase (N) protein, assists the release of newly-made virus particles from the infected cells and prevents unnecessary reinfection of infected cells.

Inside the virus envelopes are the instructions used to cause the host cell to construct new virus particles. Those instructions are made of ribonucleic acid (RNA).

Production and release of viral particles kills lung cells, but this is usually not the direct cause of an

infected person's symptoms. The cell death and presence of viral particles elicits a response by the body's immune system. Defense cells are produced to consume the viral particles and chemicals are produced to alert other systems in the body. The production of new cells in the lymph nodes causes them to swell and cause pain. Mucus production increases in the breathing passages causing a runny nose and coughing. These are meant to remove viruses from the body. Unfortunately, it also leads to spreading the virus to others. The chemicals made by the immune system can affect the part of the brain that regulates body temperature, so a fever, body aches, and chills result. This is intended to inhibit the growth of the virus. Another chemical that has direct antiviral properties also has the side effect of causing appetite loss, fatigue and weakness.



Why was the 1918 influenza so much worse than annual influenza?

Influenza is caused by the influenza A virus, one of a family of three influenza viruses. Influenza A viruses can infect humans, birds, and other mammals. Aquatic birds are the most important reservoir for viruses that can affect humans. These viruses can have one of 16 H proteins and 9 A proteins in their envelopes. These are each numbered to indicate their type that is recognized by immune systems.

The 1918 influenza virus was the H1N1 strain. A 1952 pandemic arose from a change in the surface proteins by what is called "antigenic shift" to form an H2N2 strain. This strain changed again to an H3N2 strain to cause the 1968 pandemic. In 1977 a pandemic appeared and was caused by a re-emergence of the original H1N1 strain, but it was a weaker form than the one at the height of the 1918 pandemic. All these strains are circulating at low levels among the human population. Antigenic shifts rarely occur and are caused by new genes



replacing old genes and so changing one of the protein types. These new genes come from different strains of viruses infecting a host simultaneously so that their genes can shuffle during the production of new viruses. A more frequent kind of genetic change, called "antigenic drift" also occurs and causes small changes in the genes encoding the two proteins. Those changes can change the characteristics of the virus that affect its ease in spreading or the seriousness of the disease. It also changes the ability of the immune system to recognize the new variant and this is why we need to get a new flu vaccine every year since the viruses

"drift" from infection to infection, making new variants over the course of a year. It is not clear what features of the two proteins in the 1918 strain caused it to be more deadly.

Unlike other influenza pandemics, the 1918 influenza caused more pneumonic complications so that most deaths resulted from secondary lung infections. This caused the mortality rate in the US to be 2.5% rather than the more common rate of less than 0.1%. Streptococcus bacteria were the most common secondary infectants that caused bacterial pneumonia. These bacteria normally live in the nose and upper airways, but were able to move down into the lungs after the virus destroyed the surface of lung cells and minimized their defenses. The prevalence of bacterial pneumonias complicated efforts to discover the cause of the pandemic. Several scientists thought that some bacterium was responsible and they spent many years in futile efforts to isolate the bacterium they thought caused it and to develop vaccines and treatments against it.

The 1918 H1N1 strain likely arose from an avian source and may have been a new variant that had not been seen in human before. The 1918 H1N1 strain still circulates today and is an ancestor of the seasonal flu viruses.



CAMP DEVENS

A doctor stationed at Camp Devens, a military base just west of Boston, writes to a friend and fellow physician, of the conditions to be found there as influenza was making its presence felt.

29 September 1918

Camp Devens is near Boston, and has about 50,000 men, or did have before this epidemic broke loose. It also has the base hospital for the Division of the Northeast. This epidemic started about four weeks ago, and has developed so rapidly that the camp is demoralized and all ordinary work is held up till it has passed. All assemblages of soldiers taboo. These men start with what appears to be an attack of la grippe or influenza, and when brought to the hospital they very rapidly develop the most viscous type of pneumonia that has ever been seen. Two hours after admission they have the mahogany spots over the cheek bones, and a few hours later you can begin to see the cyanosis extending from their ears and spreading all over the face, until it is hard to distinguish the coloured men from the white. It is only a matter of a few hours then until death comes, and it is simply a struggle for air until they suffocate. It is horrible. One can stand it to see one, two or twenty men die, but to see these poor devils dropping like flies sort of gets on your nerves. We have been averaging about 100 deaths per day, and still keeping it up. There is no doubt in my mind that there is a new mixed infection here, but what I don't know. My total time is taken up hunting rales, rales dry or moist, sibilant or crepitant or any other of the hundred things that one may find in the chest, they all mean but one thing here — pneumonia — and that means in about all cases death.



...

I don't wish you any hard luck Old Man, but do wish you were here for a while at least. It's more comfortable when one has a friend about. The men here are all good fellows, but I get so damned sick o' pneumonia that when I eat I want to find some fellow who will not "talk shop" but there ain't none, no how. We eat it, sleep it, and dream it, to say nothing of breathing it 16 hours a day. I would be very grateful indeed if you would drop me a line or two once in a while, and I will promise you that if you ever get into a fix like this, I will do the same for you.

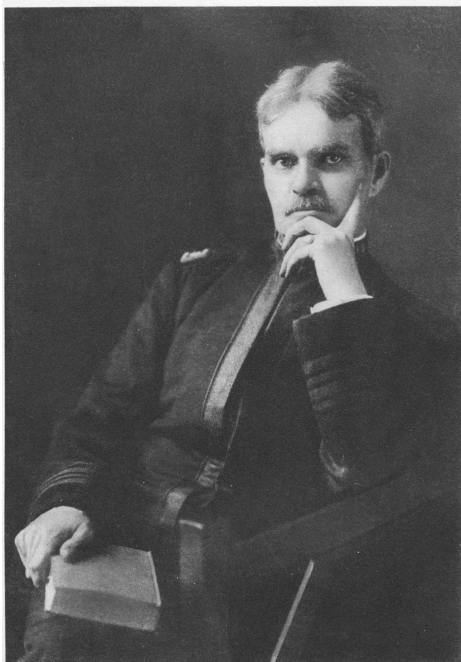
"Kansas Flu"? A Tale of Two Haskells



Dr. Loring Minor

The origin of the 1918 influenza pandemic is not known. The most recent and popular claim is that it appeared in rural west Kansas Haskell county. This claim was put forward by author and journalist John Barry. He claimed that local physician Dr. Loring Minor treated several cases of a severe influenza in January and February of 1918 according to Barry's interviews with his descendants. As evidence of his theory, Barry cited a few notices of influenza in Haskell county in a regional newspaper and a report of "18 cases of influenza of severe type, from which 3 deaths resulted, was reported at Haskell, Kans." in the April 5 edition of the United States Public Health Service's "Public Health Reports." Barry suggested that Dr. Minor had provided that report to the USPHS. A few young men visited home in Haskell county at this time and then returned to Camp Funston in eastern Kansas. An outbreak of influenza occurred there in March. Barry suggests those soldiers carried the flu to the base from Haskell county and then, from there, the disease spread among other military cantonments throughout the US and then went overseas. An

alternative theory contests Barry's evidence for this and point out that some of his evidence actually was about an influenza outbreak at the Haskell Institute in Lawrence, Kansas in April. The Haskell Institute was an off-reservation boarding school for native American children.



Dr. Charles Banks, Assistant Surgeon General, USPHS

The Institute was visited by Dr. Charles Banks, Senior Surgeon of the USPHS and he reported back to the director afterwards that

they had done a fine job of dealing with the outbreak. In that report he wrote, "...at your Institute, a like condition ensued with 18 cases of this severe type. The three deaths directly attributable to that severe lung involvement is not a large proportion... ." Consequently, it is more likely that the USPHS report was made by Dr. Banks. The outbreak at the Institute came after the one at Camp Funston. How the disease got to Funston is unclear. Despite all the uncertainties, the early appearance of a severe form of influenza in Kansas is certain, so it is likely the origin of the pandemic.



Haskell Institute

Simon Flexner

by Ana Depino

Before the common flu we know today, influenza was a significant problem in early 20th century America. Medical technologies were beginning to progress, and key scientific figures were coming to light, as seen in Simon Flexner, a physician in the early 1900s. He grew up in Louisville Kentucky, the middle son born into an immigrant Jewish family. He quit school in sixth grade, and spent his days hopping between jobs, and at 19 years of age working with a druggist washing bottles. Flexner became fascinated with what he saw at the druggist's microscope. This led him to pursue a 2-year program at the Louisville College of Pharmacy, followed by medical school. Flexner sent his microscopic observations of cells and bacteria to Dr. William Welch, Dean of the Johns Hopkins University School of Medicine, where Flexner's younger brother Abraham had previously graduated. Flexner soon found himself a student at Hopkins and a protégé of Welch. In several years he became a professor of pathology at Hopkins. Flexner later become a professor of pathology at the University of Pennsylvania.

In 1901, the Rockefeller Institute for Medical Research was created. Welch was the founding president of its Board of Scientific Directors and was offered the position to become the Director of the institute. Welch declined the position and offered it to Flexner and he accepted Welch's offer. Under Flexner, the institution opened its own lab in 1903 to research independently and opened its own hospital in 1910. Flexner saw the hospital as a large testing center that would allow its scientists to better control their clinical experiments. Flexner was said to have given Rockefeller the edge and sharpness it needed to compete with other institutions.



During the meningitis outbreak in the US in the early 20th century, he found that an antiserum that had shown minimal effectiveness could be made much more potent if it were injected into the spinal fluid, but only if some spinal fluid had been removed before the antiserum was injected. His method with the serum cut the death rate of meningitis to a mere 18% percent from an 80% rate for untreated patients. Flexner was a man who enjoyed being recognized for his deeds, and quite enjoyed the publicity he received after this groundbreaking result. However, as cold, and as sharp as he could be at times, he firmly believed in openness in expressing different ideas and theories. He relished in discussions over lunch of different opinions from colleagues working in different areas, going as far to having Fridays being routine to present findings, suggest experiments, and give new ideas.

When the flu epidemic began in 1918, Flexner, now 54 years old, joined the Army as a Colonel and the Rockefeller Institute became U.S. Army Auxiliary Laboratory No. 1 with most of its staff becoming commissioned officers. The Rockefeller Institute hosted a number of scientists who made contributions to the fight against the influenza pandemic in areas of the associated pneumococcal pneumonia and efforts to develop influenza antisera and vaccines.

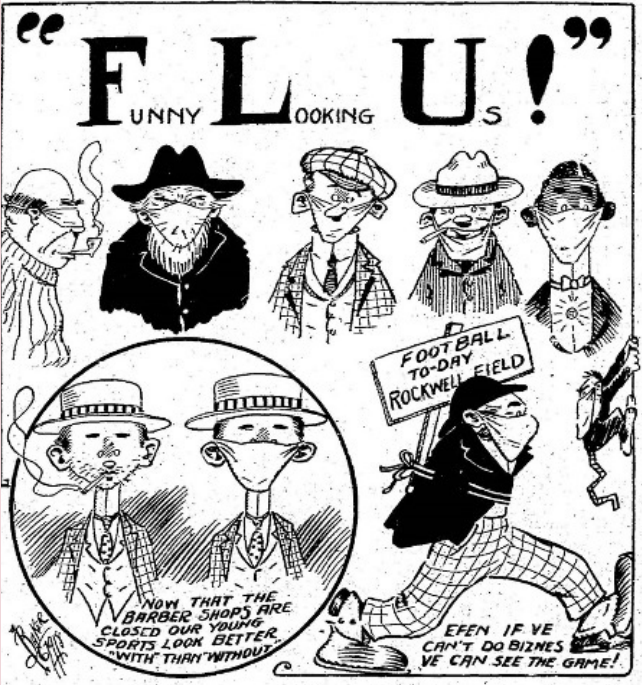


ANTI-MASK MEETING
TONIGHT (Saturday) JAN. 25
DREAMLAND RINK
To Protest Against the Unhealthy Mask Ordinance
Extracts will be read from State Board of Health
Bulletin showing compulsory mask wearing to be a failure.
Eugene E. Schmitz and other interesting speakers.
Admission Free.



Scenes from a pandemic

They Improve the Looks of Some People



The Tragic Tale of Dr. Paul Lewis

In fall 1918, Philadelphia was one of the worst-hit cities in the world. A greedy and corrupt city government ignored warnings to prepare for the rising influenza pandemic and not only failed to prepare, but exacerbated the spread by holding a parade on September 28 to stimulate sales of Liberty Bonds to support the war. 200,000 people attended, and, as expected given the virus' incubation period, in three days every bed in Philadelphia's 31 hospitals was filled. In the week ending October 5, some 2,600 Philadelphians had died from the flu-related illnesses and 47,000 were infected.

Dr. Paul A. Lewis was, at the time, laboratory director of the Henry Phipps Institute for the Study, Treatment, and Prevention of Tuberculosis. The Institute was established in 1903 and attracted the best minds in the study and treatment of infectious diseases.

Like many such researchers, Lewis was enlisted in the military during World War I and was a Commander in the U.S. Naval Reserve.



Paul Lewis

Lewis's research at the Institute started with studies of the tuberculosis bacillus, but his focus changed in 1918 to examining the cause of and treatments for influenza. He had made his name in 1910 as co-discoverer of the cause of polio when identified it as a virus. In 1918, some thought it was caused by a virus, but more were convinced it was caused by a bacterium. Pneumococcal bacteria were often found in flu patients, but it was thought these arose secondarily in ill patients and these lead to lung infections (pneumonia). A bacterium called *Bacillus influenza* had been identified many years earlier and was thought to be the cause. It could be isolated from some, but not all, of those afflicted with the 1918 influenza.

Lewis focussed on this bacterium and developed a vaccine against it. In trials of the vaccine, some immunity to infection was observed, but it did little to stem the outbreak. Lewis doggedly pursued this avenue of investigation, like others were doing, but it did not lead to any breakthroughs by the time the pandemic waned

in 1919.

Lewis continued on this non-productive path until 1923 when he tired of his overwhelming administrative duties and left the Institute and returned to where he had worked previously, the Rockefeller Institute for Medical Research in Princeton, New Jersey. He resumed his work on tuberculosis there, but he never returned to his previous levels of productivity.

While at Rockefeller, Lewis hired an assistant, Dr. Richard Shope, to work on tuberculosis. Neither of them, however, could move the research forward. While visiting home in Iowa, Shope observed swine influenza first hand. Influenza had been circulating in the Iowa swine since 1919. Unbeknownst to researchers, the ailment was transmitted to pigs from humans. Shope took samples back to Lewis' laboratory and they were able to isolate a strain of the influenza-related bacterium Lewis had previously worked on. This did not lead to any advances in human influenza research.

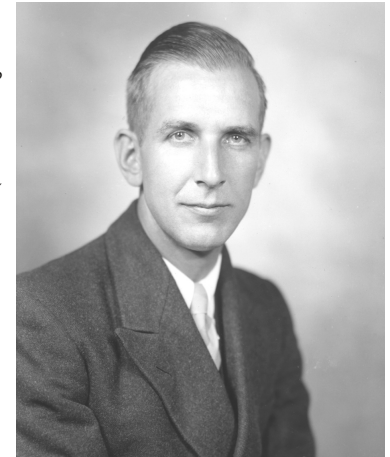
About this time, Lewis learned that a colleague had isolated a bacterium that he claimed caused yellow fever. While investigating it in the field, the colleague contracted the disease and died. The open question about the cause of yellow fever was too attractive to Lewis, so he volunteered to take up the question. He travelled to Brazil where an outbreak was occurring to look for this bacterium. While there, he, too, contracted the disease and died in June 1919. Following his death,

Shopes again took up the swine influenza problem and, in 1931, reported that instead of a bacterium, it was caused by a virus. He listed Lewis as author on one of the three papers he wrote on this finding. Shopes also showed that the bacillus he had isolate was a secondary infectious agent so that if a pig were infected with the virus and then secondarily acquired an infection with the bacterium, the disease was much more likely to lead to death. Years later other investigators, using Shope's methods, showed that human influenza is caused by a similar virus and *Bacillus influenzae*, today called *Haemophilus influenzae*, was shown to be a secondary infectious agent.



Paul Lewis

Perhaps, had he focused on Shope's work earlier, he might have been able to apply his expertise with viruses and discovered the swine influenza virus earlier and, then examining human samples, have discovered the human influenza virus, too. Hindsight in research is easy. Knowing which research avenues to follow while in the heat of the pursuit is very difficult.



Richard Shope, 1936

Paul A. Lewis
1879-1929

Paul A. Lewis died at Bahia, Brazil, on June 30, 1929. He was an associate member of the Rockefeller Institute for Medical Research, attached to its Department of Animal Pathology near Princeton, New Jersey. His death from yellow fever, contracted while studying the disease, came as a sudden shock to his many friends, and to his intimates has caused a sense of irreparable loss. Under any circumstances death is a tragedy to those left behind, even when they are near at hand to comfort and make easier the last hours. The tragedy becomes more poignant when it occurs in a distant land and when the inevitable must be faced alone and without the comforting presence of those one holds dearest.

The death of Dr. Lewis from yellow fever adds another to the illustrious list of those who have sought to solve the mystery of this disease. Furthermore, his death again emphasizes the fact that the profession of medicine has never lacked those who are willing to risk all that a more complete knowledge of disease may be obtained.

America



The United States declared war on Germany and its allies on April 2, 1917. President Woodrow Wilson, though originally opposed to getting involved in the European crisis, wholeheartedly endorsed the resolution and, until the war ended in November 1918, he devoted all his attention and efforts to the war effort. Consequently, he never publicly acknowledged the influenza pandemic that ravaged his country in 1918-19. The federal response to the pandemic was weak and slow. Even within the military,

which was affected by it early on in the pandemic, the response was slow and inconsistent, despite the warnings and efforts of several prominent doctors and scientists who were advising the Army.

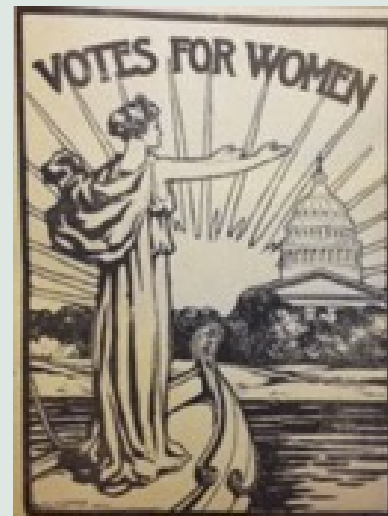
The American public was largely left in the dark about the extent of the pandemic as it began sweeping the country and the world. This was because of a massive US propaganda effort headed up by investigative journalist George Creel who Wilson appointed to head up the new United States Committee on Public Information. This agency promoted the war effort and dismissed any mention of the growing pandemic in newspapers, public advertisements, pamphlets, and patriotic social groups. The Espionage Act of June 1917 followed by the Sedition Act of May 1918 both prevented newspapers and others from mentioning the pandemic as this was seen by the government as an effort to hinder the war effort. The illness was called the "Spanish flu" because, unlike the countries involved in the war, Spanish newspapers reported cases in May, so propagandists pointed to these reports to show it came from Spain.

Military encampments, or cantonments, have always been breeding grounds for infectious diseases. This was the case in spring 1918 when measles and typhus cases were on the rise. The appearance of a flu-like illness in early spring did not raise too much concern, until it began to appear in several cantonments. By summer, the number of cases began to wane.

It is not known where the new, more infectious, strain of the influenza virus appeared in the summer of 1918. In Europe, Germany attempted a new attack on allied lines in July, but its forces were too diminished by influenza to be effective. Case numbers in the cantonments in the US began to rise in August. By September it came roaring back, not just in the cantonments, but in the civilian population,

too. It became too hard to ignore and those who did, like city officials in Philadelphia who went ahead with a Liberty Parade on September 28, paid the price.

The pandemic took place in a US different than today's country, but very recognizable. The country was experiencing a large immigration of people from Europe, particularly southern and eastern Europe. Racial relations were poor and



in 1918

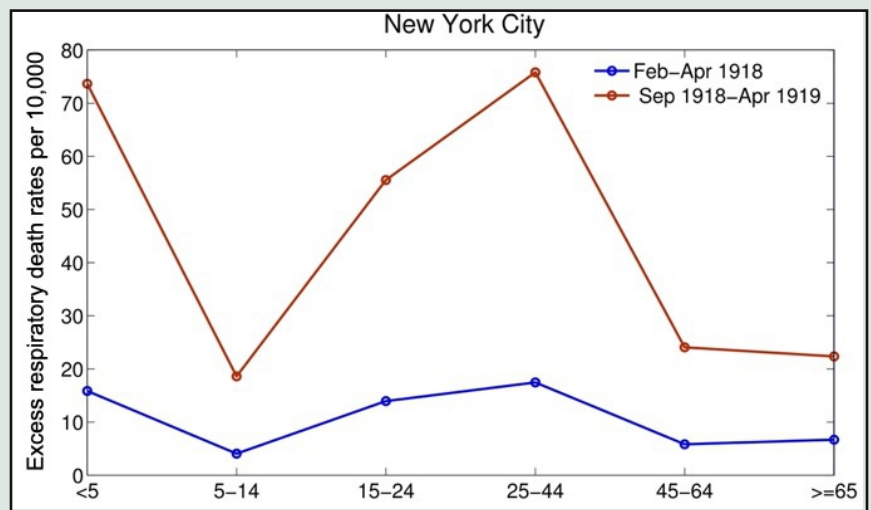


declining as the Ku Klux Klan was experiencing a resurgence and anti-Asian feeling in the West was rising. The war economy was very good, but the living conditions for the poor, particularly in the rapidly growing, grossly overcrowded industrialized areas of cities, were like those in 19th century slums. Agriculture was being industrialized with new cultivation machines and large markets needed to feed soldiers.



One of the most frightening aspects of the 1918-20 pandemic was the population that was most affected by it. Influenza is typically most prevalent in the elderly portion of the population, with a smaller incidence in the very young. This pandemic was different, affecting the young adult segment, as shown in this graph. This affected those in the military and young families most profoundly.

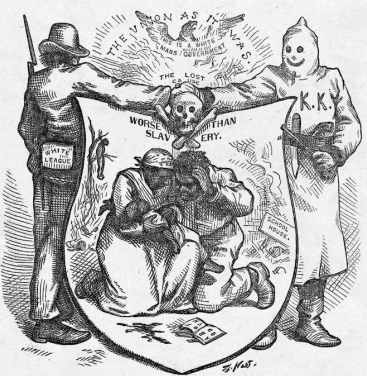
The pandemic claimed at least 50 million lives in the world, after having infected around half a billion people—one-third of the world's population at the time. Approximately 675,000 people died in the U.S. Influenza continued to appear in outbreaks in the US until April 2020. Many of those who survived the infection



found themselves afflicted by strange maladies long afterward. Coronary heart disease was found to be more prominent in those who lived through the pandemic. Depression, dizziness, and sleep disturbances were common. Lower educational achievements and increased likelihood of being income poor have been found following this pandemic.

Perhaps one of America's last victims of the pandemic was Notre Dame's All-American football star George Gipp. Gipp fell ill with pneumonia on November 20, 2020. After a fourth relapse, he died on December 14.

Hopefully we can remember Gipp along with all the millions of victims of this devastating pandemic and use their courage to help us fight off future pandemics. We can follow the pleas of Gipp's coaches, "Let's get one for the Gipper!"



Articles in this flipbook were written by **Kenneth Noll**, Professor of Microbiology at the University of Connecticut. He is also responsible for the layout and editing of this publication. The Simon Flexner article, however, was written by Ana Depino, a student in Prof. Noll's spring 2021 UConn course *Pandemics: History and Perspectives*. Students in that course wrote a story about each pandemic we discussed with the goal of publishing some of those on this website. I am pleased to be able to showcase the writing of these students here.

Resources

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Interviews with John Barry

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Fresh Air interview with Terry Gross: <https://www.npr.org/2020/05/14/855986938/what-the-1918-flu-pandemic-can-tell-us-about-the-covid-19-crisis>

Origin of the 1918 influenza pandemic

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Website, 2020: "The Haskell County origin story and the other "Haskell" in Kansas – Spanish Influenza in Victoria, Canada, 1918-1920" <https://spanishfluvictoriabc.com/spanish-flu-origin-spread-character/the-haskell-county-origin-story-and-the-other-haskell-in-kansas/>

Photo and image credits

- Cover: Mrs. Brown suffers from influenza with her child, Nov. 29, 1918 Library of Congress Call Number: LC-A6195- 3525: <https://www.loc.gov/item/2017668532/>
Dr. Loring Miner: https://www.athensnews.com/news/local/miner-man-of-medicine-and-ohio-university-grad-played-research-role-in-1918-pandemic/article_8f362c77-3367-5d40-8d95-cbcea57507ad.html
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Haskell Institute: <https://www.1918fluks.com/episodes/ep4>
World spread of influenza: <https://africacenter.org/spotlight/lessons-1918-1919-spanish-flu-africa/>
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Epidemic influenza: a survey. Chicago: American Medical Association, 1927.

Foolproof covid posters: <https://genomicsin.blogspot.com/2020/05/part-2-of-foolproof-april-series-myth.html>

Dr. George Banks: <https://www.findagrave.com/memorial/57202501/charles-edward-banks>

Camp Funston, Kansas: <https://history.army.mil/html/bookshelves/resmat/wwi/pt02/ch09/pt02-ch09-sec06.html#lg=1&slide=0>

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Spanish flu headline: https://www.nola.com/news/coronavirus/article_d430f5f8-657e-11ea-8e4a-8b5b4e421afe.html

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Funston hospital: <https://www.downtoearth.org.in/blog/governance/between-1918-19-spanish-flu-and-covid-19-not-much-has-changed-71425>

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