

# WWI and the 1918 Flu Pandemic

*[Announcer] This program is presented by the Centers for Disease Control and Prevention.*

[Sarah Gregory] Hi, I'm Sarah Gregory, and today I'm talking with Dr. Terence Chorba. He works at CDC in Tuberculosis. We'll be discussing the 1918 influenza pandemic and the October 2018 EID cover essay, "Concurrent Conflicts—the Great War and the 1918 Influenza Pandemic." Welcome, Dr. Chorba.

[Terence Chorba] Thank you for having me. It's a pleasure.

[Sarah Gregory] What happened with the 1918 flu? What occurred that allowed this flu to spread so quickly?

[Terence Chorba] Well, in the 1918 flu pandemic, sometimes referred to as the Spanish flu, it was an exceptionally deadly pandemic, and it was the first of two pandemics in the past hundred years that involved the H1N1 subtype of influenza A virus. The term "H1N1" refers to two specific glycoproteins, or antigens, on the virus's surface: hemagglutinin, hence "H," and neuraminidase, hence "N," on the viral capsid, or on the surface. And, for this reason, different strains or subtypes are referred to as H1N1, H1N2, etc., depending on the types of H or N glycoproteins that are expressed.

And in this pandemic, there were about 500,000,000 people—about a half a billion—around the world who were infected, eventually, including people even out in the remote islands of the Pacific. And the toll in deaths has been estimated to be somewhere between 50 and 100 million, which at the time, which would have been about three to five percent of the world's population. In terms of the total lives lost, it must have been the deadliest disaster in the history of the human race. As to what it was that allowed this flu to spread so quickly, there was a strong sentiment that the massive troop movements and close quarters for soldiers in cramped congregate settings, as in the circumstances of World War I, hastened the spread of the virus.

[Sarah Gregory] So, what strain was it and why was this flu strain so virulent?

[Terence Chorba] The strain of influenza A involved was a subtype of avian strain H1N1, which genetic sequence has been successfully determined using tissue samples recovered from a female influenza victim buried in the Alaskan permafrost, and from samples preserved from American soldiers who died of flu at the time. As to why it was so virulent, we could talk for hours. There are some papers that support the hypothesis that the viral infection itself was not more aggressive than other well-known influenza strains, but rather the special circumstances of the war scene, with malnourishment, cramped quarters, crowded medical emplacements in hospitals, and lack of hygiene, all fostering bacterial superinfection that killed many of the victims. Remember, it was not until 1935, when we first had the first sulfa drugs, that the . . . we had the first antibiotic useful for treating streptococcal and staphylococcal infections. The majority of deaths in the great Spanish flu pandemic were from superinfection, mostly bacterial pneumonia, for which we now have antibiotics to intervene. Unfortunately, at the time of the First World War, other than some antifungal agents, the only antibiotics that were available were the arsenicals, which were used to treat syphilis.

[Sarah Gregory] So what finally stopped the tide?

[Terence Chorba] Well, region by region, this epidemic had an abrupt pattern of ending locally. For example, I've read that in Philadelphia, there were about 5,000 people per week in the middle of October in 1918 who were...who died. That is a massive amount, amounts of death. But by the time of the armistice, just a month later, new cases of flu were pretty much gone from the city. And, we...so we don't know why it came to such an abrupt halt, but one theory holds that the virus mutated extremely quickly to a less lethal strain. There was no vaccine against the agent at the time, so it's assumed that there were mutations occurring that considerably reduced the transmissibility and the lethality of the virus very quickly.

[Sarah Gregory] What lessons have we learned from this pandemic?

[Terence Chorba] Well, the principle lessons that we learned from the pandemic were really programmatic lessons. The accounts of the public health authorities and descriptions of the interventions that were put into place to curb the effects of the pandemic at the time, really demonstrated a total lack of preparedness that pervaded all the attempts to deal with the overwhelming morbidity and mortality that was occurring. Clear orders were generally not being given to public health officials and transparency in what was going on was often lacking. There was little in the way, in terms of consistent advice with respect to the wearing masks, mixing in public places, allowing congregate settings to occur, like church services or community gatherings. And the pursuit of activities of daily living in the community, there was very, very little guidance that was being given consistently, consistently. And so, what we did learn is that, to prepare for situations like this in future, community plans need to be thought through ahead of time, with clear lines of authority and responsibilities, and the appropriate roles spelled out for who's supposed to be doing what.

[Sarah Gregory] Now, I know with the swine flu and H1N1, in the last 10 years here at CDC, the big concern was, "Could this happen again?" Can this happen again?

[Terence Chorba] Well, for H1N1, derivatives of the Spanish flu strain resulted in an H1N1 epidemic happening again that was swine origin in its...it really came from flocks of swine. And it occurred in 2009. That strain contained genes from, actually from five different flu viruses. And within a year, the World Health Organization was able to declare that that pandemic was over. And it probably reflected the fact that there were mutations that existed in this virus, that resulted in most of the infections, in 2009 and 2010, as being mild, similar to seasonal flu, with most of the infectees having a pretty rapid recovery.

However, it is thought that influenza viruses readily resort, recombine, have point mutations, due to their segmented RNA genomes. And so, pandemics can occur. But we're in a much better position today, with our understanding and surveillance of the spread of influenza viruses. We now have some helpful antiviral drugs, we also have anti-cold medications, and we have the ability to ramp up production of flu vaccines targeting the specific flu virus strain that may be the particular challenge, in a very short time.

[Sarah Gregory] Okay. So there are better drugs and treatment now, so it's not likely that millions of people will die again, right?

[Terence Chorba] Right. Fortunately, we have the flu vaccine with modest-to-high protection against flu, season to season. And the vaccine is intended to address different antigens, so different Hs and different Ns as they occur in the circulating strains of the virus, with a new

version of the vaccine really being developed twice a year now, depending on the anticipated circulating variance of the virus. One must keep in mind that virus prevalence varies widely between years, so even if all other things were equal, we'd still see effectiveness measures that would still vary from year to year.

Fortunately, for those who do get infected, we also have these two principle classes of new antiviral drugs used against influenza. The first group first became available in the 1970s, called M2 inhibitors, that disrupt the function of proton channels within the virus, such as amantadine and its derivatives, like rimantadine. And these have activities against some strains of influenza A. The second group, that became available in the late 1990s, are called neuraminidase inhibitors, and you may know...you may be familiar with one of these—oseltamivir. These are known to have some activity against strains of both influenza A and B. And when taken soon after infection, these drugs can reduce the severity of the symptoms, and they may, perhaps, can be taken in certain settings prophylactically to reduce the risk of infection.

However, not surprisingly, there are drug-resistant strains of flu that have emerged to both classes of drugs. And, of course, fortunately, we have a host of antibiotics that were not available at the time of the First World War, for treating secondary infections that could otherwise result in considerable morbidity and mortality, as was seen in 1918. Back then, we did not have antibiotics for treating staphylococcal or streptococcal infections, which accounted for the majority of deaths in the face of that epidemic.

[Sarah Gregory] So, what's your area of expertise at CDC?

[Terence Chorba] Well, I've actually have wide experience at CDC with infectious diseases, but my current area of expertise is tuberculosis, which was another big killer at the time of the First World War. And I have no involvement in influenza. What pulled me into schooling myself about influenza and writing this article, was my fascination with World War I. And that was fomented by four things.

First of all, I had two uncles who served in the American forces in that war, and one of them was a machine gunner who got influenza at the time, and he was wounded in an artillery exchange. And so, a bit of the family mythology revolved around him.

Second, I have always had a great admiration for the poets of that war: Joyce Kilmer, Siegfried Sassoon, Rupert Brooke, Rudyard Kipling, Wilfred Owen, and my favorite, John McCrae, who was a Canadian poet and physician who served in combat in Belgium. And McCrae was best known for writing the poem, "In Flanders Fields." And many of you in the audience, I'm sure, know the poem.

In Flanders fields the poppies blow  
Between the crosses, row on row,  
That mark our place; and in the sky  
The larks, still bravely singing, fly  
Scarcely heard amid the guns below.

We are the Dead. Short days ago  
We lived, felt dawn, saw sunset glow,  
Loved and were loved, and now we lie,

In Flanders fields.

Take up your quarrel with the foe:  
To you from falling hands we throw  
The torch; be yours to hold it high.  
If ye break faith with us who die  
We shall not sleep, though poppies grow  
In Flanders fields.

And, you know the poppy is always used for remembrance of, around times like Memorial Day and Veterans Day.

McCrae wrote those words back in 1916, early in the conflict, at a time that there was romanticism for involvement in the war, and before the truths of the awesome carnage, the huge pandemic, and the suffering all came to light. Sadly, he died of meningitis in a tent hospital in Belgium in early 1918.

The third inspiration to...to learn more about, about the First World War and how it had its, not just its causes, but its effects and what transpired in the war, was when I read a bestseller, a Pulitzer Prize-winning history by Barbara Tuchman, written in 1963, *The Guns of August*. And the beginning starts with the funeral of Edward VII and the crowned heads of all, of all Europe coming together in 1910. And then, a fascinating book, it then carries you through the entirety of the war. It's a great read, and if you haven't read it, go read it, and you'll be glad you did.

And the fourth, of course, was really the substance of this article, is just the art of John Singer Sargent. That's always fascinated me. Sargent was an American who persisted as a realist in Europe during the golden age of impressionism. And in his mid-60s, he was commissioned by the British to document the Anglo-American effort at the end of the Great War in northern France, only himself to develop influenza during the peak of the epidemic. And he documented his hospital stay with a wonderful watercolor, that's the subject of the article that prompted this interview that we're having now.

[Sarah Gregory] How does the painting speak to you in relationship to the 1918 pandemic?

[Terence Chorba] In relation to the pandemic, Sargent's painting—the hospital tent itself—could be viewed as a bit of commissioned propaganda, an activity in which hundreds of artists, on commission or on orders, were engaged on both sides of the trenches. The painting portrays no carnage, no deprivation, no disorder; the setting is inspirational in keeping with the desires of the British war ministry that had commissioned Sargent. And it is devoid of the horrors of the conflict, where the ubiquitous amplification mechanisms for infectious disease transmission that war provides: mass migrations, crowding, forced congregational living, poor ventilation, malnutrition, and poor sanitation. I chose the painting because its theme was one of a field hospital from the perspective of a convalescing patient in that epidemic and gave me a springboard to talk about the centenary of the pandemic itself.

[Sarah Gregory] So, on that note, would you care to read the October 2018 cover essay that you cowrote, "Concurrent Conflicts—the Great War and the 1918 Influenza Pandemic"?

[Terence Chorba] Sure. I'd be happy to.

The generation that endured through what was perhaps the most devastating epidemic ever—the great influenza pandemic of 1918—is now gone. The influenza strain of that pandemic infected about 500 million people, one third of the world’s population, with extraordinarily high pathogenicity and virulence. The result was staggering mortality: an estimated 20 to 100 million lives were lost worldwide. The estimate of deaths of Americans attributable to influenza in that pandemic is 675,000, the majority of whom were among those from ages 20 through 40 years. During World War I, the “Great War,” three influenza-associated mortality waves occurred in northern Europe, beginning in early summer of 1918 and extending over the course of a year; influenza accounted for more fatalities than military engagement. The highest point of combined influenza and pneumonia mortality occurred in October 1918. At that time, the pandemic strain became known as “the Spanish flu,” so called because neutral Spain lacked war censors and was the first country to report on the pandemic publicly; however, the geographic origin of the causative organism remains an enigma.

Among that generation was the artist John Singer Sargent, who was born in Florence in 1856 and raised principally in France, the child of two Americans: an eye doctor turned medical illustrator father, and an amateur artist mother. Home-schooled and trained at the *École des Beaux-Arts* in Paris, he enjoyed a storied career, principally as a portraitist. In early summer 1918, late in his renowned career when living in England, Sargent was invited back to France on commission by British Prime Minister Lloyd George, Field Marshal Douglas Haig, and the British Department of Information, War Memorials Committee, to depict the Anglo-American effort in the war.

During late September, while preparing sketches for his iconic painting *Gassed* in a military camp near Roisel, in Péronne, France, Sargent fell ill with influenza. Sargent was cared for and convalesced in a hospital tent in France. He wrote that he lay there, “with the accompaniment of groans of wounded, and the chokings and coughing of gassed men, which was a nightmare. It always seemed strange on opening one’s eyes to see the level cots and the dimly lit long tent looking so calm, when one was dozing in pandemonium.”

Sargent’s hospital experience inspired the image featured on this month’s cover, *Interior of a Hospital Tent*. This watercolor depicts the interior of a hospital tent with military cots arrayed in file on the side, covered with blankets in a mix of red (for the contagious cases) and brown (for the convalescing or noninfluenza cases), two colors that must have dominated the entire war hospital environment. The scene is actually one of tranquility, a respite from the chaos and carnage of war. In one bed, a soldier lies reading, his head bolstered by pillows; in another, a soldier sleeps on his side with an open tent flap behind him, his bed bathed in light from the world of the healthy. Beyond them, there are three or four more cots with soldiers reclining in varying amounts of darkness and light. Above all, in varying shades of military brown, is a great propped tent canopy.

Sargent remained hospitalized for a week, but unlike so many of the much younger soldiers, he recovered and returned to his task of documenting what he saw. At the outset of his journey through France in July 1918, he had written that the best material for his commission would be to see “a big road encumbered with troop and traffic...combining English and Americans.” By mid-October in northern France, Sargent had had his fill of war. He wrote, “I’ve wasted lots of time going to the front trenches. There is nothing to paint there—it is ugly, meagre, and cramped.... I have seen what I wanted, roads crammed with troops on the march. It is the finest spectacle that war affords...”

By the end of October, Sargent returned to Britain to complete the several works for which he had been commissioned. What he had seen firsthand and documented from his experiences in the conflict were the amplification mechanisms for infectious disease transmission that war provides: crowding, migration, and poor ventilation and sanitation. Because there were no vaccines available with proven safety and efficacy to protect against influenza and no antibiotics to treat secondary bacterial infections from influenza or wounds, there were few public health measures available to counter the spread and devastation of the pandemic.

Whether the great pandemic tipped the balance of power toward the cause of the Allies, such that surrender came in November 1918, remains a matter of debate. The theory that one conflict had a significant impact on the outcome of the other is supported by data published from archives in Austria, which indicate that waves of morbidity and mortality from influenza were experienced both to a larger extent and earlier among the Central Powers (that is, Germany, Austria-Hungary, the Ottoman Empire, and Bulgaria) than among the Allies. Unfortunately, the pandemic was not limited in its geographic reach, and through 1920, it exerted a tremendous toll on morbidity and mortality and created economic and social burdens, both elsewhere in Europe and throughout the Americas, Africa, Asia, Australia, and the Pacific.

[Sarah Gregory] Thank you so much for taking the time to talk with me today, Dr. Chorba.

Listeners can read the October 2018 article, “Concurrent Conflicts—the Great War and the 1918 Influenza Pandemic,” online at [cdc.gov/eid](https://www.cdc.gov/eid).

I’m Sarah Gregory for *Emerging Infectious Diseases*.

[Announcer] For the most accurate health information, visit [cdc.gov](https://www.cdc.gov) or call 1-800-CDC-INFO.