

The Spanish Lady's Underwear

Peeking under the dress of the “Spanish Lady” in an analysis of the virulence of the 1918 influenza pandemic and comments on the current COVID-19 pandemic.

Introduction

On September 28th, 1918, Philadelphia held a grand Liberty Loan parade in hopes of raising public funds for World War I. A mere 10,000 people were expected to attend, but with the end of war in sight, public excitement was at an all-time high, so thousands of military personnel, relief workers, scouts, and veterans marched as a crowd of 200,000 thronged in Center City. The parade was deemed an incredible success. The Philadelphia Inquirer ran a large photo on the front page and an accompanying article full of hope saying “...in every stride and in every voice there was to be seen and heard the first premonition of —victory.” The Evening Bulletin similarly praised the event, announcing that “This is a great day in Philadelphia” (Kopp and McGovern).

But both newspapers held hints of the horror to come. Tucked under that Inquirer photo of hundreds of uniformed men with rifles over their shoulders marching in orderly lines is a smaller headline that reads “Congress Votes \$1,000,000 to Curb Spanish Influenza”. Buried in the folds of the same Evening Bulletin that lauded the parade on its front page is a story about Thomas Harlacker, a policeman in his 30s who died from the Spanish flu. The story cites the city's health director who warned that “if people are careless, thousands of cases may develop and the epidemic may get beyond control” (Kopp and McGovern).

The warnings went unheeded, and the parade became grounds for a massive and sudden infection outburst of the Spanish flu. By the time the epidemic ran its course weeks later, more

than 12,000 lives were lost to the scourge. Philadelphia's only morgue, with a normal capacity of 36 bodies, was soon overrun and forced to stack bodies three or four deep. A report described the situation saying, "Some bodies were mortifying and the stench was nauseating... In the rear of the building, the doors were open and bodies lying all over the floor..." (Kopp and McGovern). The Commonwealth Brewing Company offered its cold storage plant as a temporary facility for 500 bodies.

Undertakers began price gouging families for burials and auctioning off coffins to the highest bidders. Families dug the graves of their loved ones as they grieved their deaths. Rabbi I. Rosenfeld built a casket for his own son, a senior at South Philadelphia High School, and then helped lower him into a grave in Mt. Carmel Cemetery (Kopp and McGovern).

The pressing needs of the city quickly overwhelmed hospitals and emergency services.

By early October, forty nurses of the Philadelphia Hospital begged for assistance. Despite the sheer number of cases, five nurses refused to stop care from roughly 1,200 patients. The nurses died a few days later (Kopp and McGovern).

Although Philadelphia was one of the hardest hit cities by the Spanish flu pandemic, similar situations played out across the United States and across the world. In St. Louis, roughly 40 nurses from the American Red Cross cared for about 3,000 patients that otherwise didn't have access to private nursing ("1918 Historical Image Gallery"). Boston's Red Cross chapter assigned 300 people, a mix of graduate nurses, practical nurses, and aides, to care for nearly 3,000 patients at emergency hospitals in Allegheny County, and roughly 12,000 home-bound cases (Jones).

Fear ran rampant across the country. Decades after the pandemic, Valeria Gawron asked her grandfather who two children in an old photograph were, and her grandfather said they were

his sons who died in the Great Epidemic. When Valeria asked what an epidemic was, he responded, "It is a thief who steals the young and the old and gives no time to mourn. It stole Joseph, my namesake, after he helped his friend pull a wagon up the street. It stole Stanley who had just learned to throw a ball to the other side of the yard."

"Why couldn't you mourn, Grandpa?" Valeria asked.

"We were too busy burying our dead and keeping our families safe. We barely had time for a priest to bless your uncles' bodies before we put them in the ground. We were so afraid someone else would get sick if we did not bury our dead within hours" (Gawron).

As the sick either recovered and developed immunity or passed away, the virus ran out of bodies to infect and it died out by the summer of 1918 (History.com Editors). When the pandemic had finally finished its course, conservative estimates calculated that over 50 million people died worldwide. Higher estimates ran up to 100 million (Johnson and Mueller). It was especially lethal for the young and healthy with the mortality rate peaking at 28 years old (Gagnon, Alain, et al). In fact, more U.S. soldiers died from the disease than from battle in World War I (History.com Editors). The Spanish flu was one of the deadliest infections in modern history, but what exactly was the virus that caused such widespread transmission and death? Why was it so much deadlier than the common flu that runs through households every year?

What was the Spanish Flu?

The exact origins of the 1918 influenza pandemic are unknown, with sources pointing to France, Britain, and the American Midwest. More recently, reports of an early influenza outbreak in China in 1917 suggest that Chinese workers might have experienced the original

strain. These workers came to Canada and then moved to Europe to replace European laborers as more young men joined the military for the ongoing World War I. Roughly 3,000 of these workers were put in medical quarantine in Canada, but despite this, many still arrived in England in January 1918 and then moved on to France. Once there, records from the Chinese Hospital at Noyelles-sur-Mer note hundreds dying from a respiratory illness (Vergano).

After this first wave, historians suggest that the Spanish flu mutated to become its final, deadlier form that ravaged Europe for the remainder of 1918. Despite the severity of the disease, cases among the Chinese laborers in Europe ceased at the peak of the pandemic (Vergano).

Despite the unknown origins, the popular name “Spanish Flu” or “Spanish Lady” was widely used. Spain remained neutral in World War I, and thus remained free from wartime censorship. While other European countries stifled reports of the disease to protect morale and prevent enemy countries from receiving information of their weakness, the Spanish media covered it in depth, especially when King Alfonso XIII fell ill in May of 1918. Since nations with censored media only saw reports of a deadly disease from Spain, many assumed that was where the pandemic originated (Andrews).

The Spanish flu came in three waves: spring 1918, fall 1918, and winter/spring 1919. There were two common forms of the disease. The spring of 1918 heralded the milder form, slightly worse than the seasonal flu. It included a cold, sore throat, cough, fever, muscle aches, and severe exhaustion. It typically only lasted a few days (Jester, Barbara, et al).

The severe form of the disease occurred in the fall of 1918. So deadly was the illness, many people didn't believe it was the same as the spring version. Patients languished in extreme respiratory distress. Chests heaved with heavy, bubbling rales as their lungs filled with pus. Patients starved for air. Hospitals hallways were filled with the sound of rapid and labored

breathing as each inhale and exhale became less and less efficient. As the lungs failed, a blue-purple discoloration first tainted the patient's lips and ears before spreading to the rest of the face. So dark and severe was the discoloration, that one physician noted it became difficult to tell the patient's original skin color (Jester, Barbara, et al). Blood gushed from the mouth and nose. Patients could barely remain conscious (Jester, Barbara, et al). Those that survived first few days often succumbed to a secondary bacterial infection that caused pneumonia. Many died less than two weeks after the initial infection (Jester, Barbara, et al). Yet this is only an average. Stories about the sudden onset of symptoms and then death were common. In Bellflower, Missouri a young, farm wife with eight children woke up healthy one morning and died by sunset of the next day (Brownell). In Mountain View, Missouri, four children went out on a sleigh ride. All four caught the flu and died within a week. The family placed the bodies outside as they died to preserve them in the cold. As one boy lay dying, he told his family to "just put me outside, I will join the others in a few days" (Piper).

When doctors looked at the lungs of the diseased, they described them as "livid, swollen, and distended" and notes they were filled with a thin, bloody pus (Jester, Barbara, et al). One pathologist described them as the "lungs of the drowned" (Jester, Barbara, et al).

What made this virus deadly? Why doesn't the seasonal flu kill nearly as many people? Understanding these questions means first looking at the genetic level of the influenza virus.

Unpacking the Influenza Virus

The two basic parts of a virus are nucleic acids, and a casing of proteins known as the capsid. Some viruses, including the influenza virus, have an additional envelope protecting it. The nucleic acids can either be RNA or DNA, and these provide the instructions for replication. But the virus itself cannot replicate on its own. It doesn't have the proper tools or resources and

this is what leads to disease. In order to replicate and spread, the virus infects host cells and releases its genetic material. These instructions hijack the protein building machines known as ribosomes. The cell stops normal cellular activity and instead begins to make capsids, RNA or DNA, and membranes to construct more viruses (Vidyasagar).

Influenza (flu) viruses have a roughly circular outer membrane, an inner, rigid, protein capsid, and viral RNA strands (Racaniello). There are four types of the influenza virus: A, B, C, and D. The A and B types cause the seasonal flu and can lead to pandemics. Type C infections cause only a mild illness and do not lead to epidemics and type D do not infect humans (“Types of Influenza Viruses”). On the outer membrane are proteins connected to a sugar, known as glycoproteins. On influenza viruses, there are two types of glycoproteins: hemagglutinin (HA) and neuraminidase (NA). These two proteins are important to the virus's ability to infect host cells, so cells have developed ways to defend themselves by manipulating these outer proteins.

The HA molecule acts as a key to begin the infection of a host cell. Their particular geometry allows them to bind to receptor proteins on specific host cells (Taubenberger et al). Then the entire influenza virus is engulfed by the cell (Vidyasagar). One way the human body fights against the influenza is by producing antibodies that prevent the HA molecule from binding to the receptor. The key no longer fits, and infection doesn't occur (Bouvier and Palese).

If the HA molecule is the entry key, the NA molecule acts as the exit key. After the host cell has built enough virions, the infectious virus particles, the NA molecules tear open the host cell's membrane to release the new virions that go on to infect more cells. Without the NA molecule, the virions clump at the cell membranes surface, and they can't infect other cells. Antibodies to the NA prevent the virus from leaving infected cells. Without more cells to produce more virions, infection is inhibited (Bouvier and Palese).

Vaccines are also incredibly effective in protecting individuals from the flu. Based on World Health Organization assessment of the most likely influenza strains, inactivated viruses are grown in hens' eggs or other cells. When introduced to the human body, the immune system designs antibodies for those particular HA or NA molecules. Since the virus is inactive, the host can build up immunity without actually getting sick. Then if a vaccinated individual contacts the live virus strain, their body can quickly build effective antibodies to prevent infection and illness ("How the Flu Vaccine Works").

While antibodies to the HA and NA molecules are the body's best defense against influenza, these molecules constantly make minor changes. Original antibodies will still function with small adjustments to the molecules, but these mutations accumulate in a process called antigenic drift to produce multiple strains and subtypes. Over time, these differences become too great, and the original antibodies are no longer effective against these drifted strains (Bouvier and Palese).

Influenza strains can also mutate through antigenic shift. This mutation occurs when an influenza virus strain picks up instructions for the HA and NA molecule from a different influenza subtype. This new strain will have novel HA and NA molecules. The keys have been changed and the prior antibodies don't work (Bouvier and Palese).

This antigenic drift and shift is the reason why the flu still comes every season despite the use of vaccines. The yearly flu vaccine only protects against that season's most likely strains. Even then, vaccines need to be produced beforehand, so there could still be a novel strain that the vaccine doesn't protect against (Taubenberger et al).

But what caused this particular influenza strain to become so virulent? Since scientific and medical technologies were limited or nonexistent in 1918, when the virus disappeared after the

spring of 1919, its deadly secrets were considered lost. However, modern technology enabled a full reconstruction of the virus to allow scientists to study its mysteries.

Reconstructing the Spanish Lady

In order to determine why this flu strain was so deadly, the virus needed to be brought back from the grave. Despite modern advances, it took over fifty years before scientists were able to completely sequence the original 1918 influenza strain. One problem was lack of material.

The first person to try and seriously revive the virus was Johan Hultin. In 1950, Hultin was 25 years old and in need of a Ph.D topic. A virologist mentioned that the way to solve the secrets of the 1918 pandemic would be to revive the virus from a victim buried in permafrost (Shreeve). Soon after, Hultin left for the Brevig Mission on the Seward Peninsula of Alaska. There lay a small ocean village with less than 400 people and a tragic past. In the November of 1918, be it via dog-sled traders from the neighboring town or via the local mail delivery person, the Spanish flu arrived in that small town of mostly Inuit Natives. It killed 72 of the 80 adult inhabitants in only five days (Jordan).

A mass grave site with white crosses scattered upon it rests on a hill beside the town, frozen in permafrost. The village elders gave permission to Hultin and his colleagues to excavate part of the grave to search for the intact lungs of the frozen victims in hopes of finding a live virus strain. It took days, with small campfires slowly thawing the land to enable digging. After two days, there was a stark reminder of the endeavor's seriousness. A body of a young girl. Perfectly frozen in a little blue dress. Hair done lovingly with red ribbons (Jordan).

The men kept digging.

After Hultin got the samples he needed, he returned to the University of Iowa, attempting to refreeze the tissue each refueling stop with the carbon dioxide from a fire extinguisher. Once back to the continental US, Hultin attempted to grow the virus in chicken eggs. He failed on this attempt, and abandoned this thesis in favor of another topic (Jordan).

Then came Dr. Jeffery Taubenberger, a molecular pathologist at the Armed Forces Institute of Pathology in Washington D.C. Taubenberger and his team used lung tissue from a U.S. service member stationed in Fort Jackson, SC in 1918. He became ill with the flu September 20th, 1918 and died six days later and a sample of his lungs was preserved. From this, Taubenberger sequenced fragments of the viral RNA and published his findings (Jordan).

After reading Taubenberger's research, Hultin became determined to recover the full viral RNA sequence once again. Forty-six years after his first attempt. Now 72 years old, Hultin once again returned to the Inuit gravesite armed with meager tools, including his wife's garden shears to cut through bone, but plenty of determination. This time he obtained a perfectly frozen pair of lungs from a woman he dubbed "Lucy" (Jordan).

Hultin sent the lungs to Taubenberger's team, who were able to successfully extract 1918 virus genetic material (Jordan). From there, Taubenberger sent the gene blueprints to Terrence Tumbej of the C.D.C. to reconstruct the genes to create the final virus. It took nearly ten years (Shreeve).

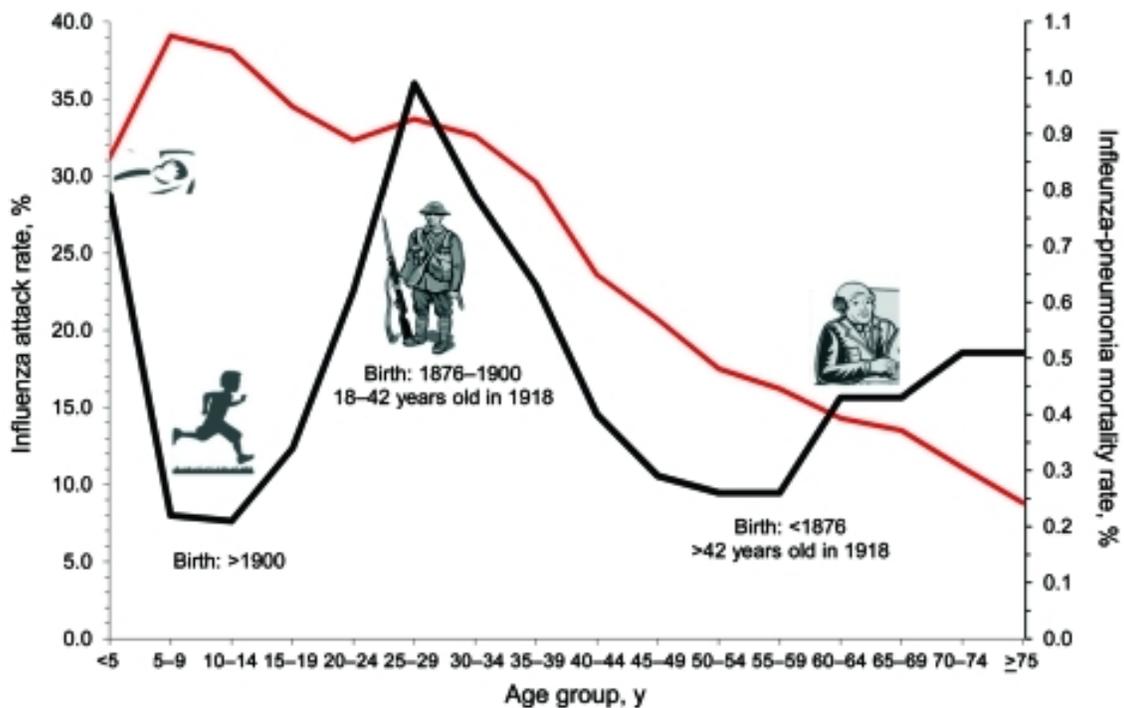
More than fifty years later after she vanished, the Spanish lady dances once more. Only this time, she's limited to Biosafety Level 3+ labs.

But as more and more genes were sequenced, Taubenberger was shocked by how incredibly... ordinary the sequence was. Indeed, of the 4,000 amino acids that make up the RNA strand, only 25 or 30 of them were different from the normal, seasonal flu. Yet these changes

made a difference. Remember the HA and NA molecules that acted as entry and exit keys?

When Trumpey replaced the 1918 HA gene with the HA gene from the common seasonal flu, the virus replicated at less than 1100th the original rate. While some viruses use the cell's own enzymes to destroy the membrane and infect other cells, the 1918 virus had its own NA gene, meaning it brought its own way out of the cell. These two factors contributed to the disease's virulence (Shreeve).

Yet the actual genetic code didn't explain why the virus killed off the young and healthy in such large numbers. Normally the mortality rate for the flu has a U-shaped curve, with more infants, children, and the elderly dying than the young and middle-aged. As indicated below, the mortality rate for the 1918 virus had a W-shaped curve with a peak at only 28 years old. This indicated that the disease affected infants, the young to middle-ages, and the very elderly more than other age groups (Shanks and Brundage).



“Illness attack rate (red line) and overall mortality rate (black line) for influenza-related pneumonia, by age groups of selected US populations, during the 1918 influenza pandemic period” (Shanks and Brundage).

One hypothesis for the unusually high young adult mortality is the concept of “original antigenic sin” (Jester, Barbara, et al). As an individual grows, their immune system is exposed to new subtypes of the influenza virus and novel bacteria strains. This memory of all the prior illnesses is essentially the immune systems armory. With the old blueprints, the body can produce effective antibodies quickly against recognized or similar diseases. But this can backfire if the playbook is limited.

Say there are three boxers. One is quite the ring hound. He's been in it for years and years and well, it kind of shows. He's getting on in years and he's maybe a bit slower and got a few more scars but he has the most experience by far. The second guy is young (maybe a little too young), and this is his first match. But he's quick and he's got energy to burn. The third one is also young but not as young as the second. He's coming off from his first match where he won in a terrific knockout in the first round. He's confident and it shows.

Each of them will be fighting against a strong opponent.

The matches begin.

The match with the older boxer looks like it's going badly at first, but the old boxer simply puts his hands up and wears the other guy down. Slowly biding his time until he can return on the attack. In the end, it's the old boxer that lifts his glove into the air, and experience wins out.

The youngest boxer doesn't know what to expect, but he's smart. The first two rounds are kind of dull as the young boxer studies his opponent. Soon the boxer's got a handle on his style and particularly his weaknesses, and releases all that pent-up energy he saved in a flurry of precise and effective attacks. At the sound of the last bell, the youngest boxer also raises a fist into the roaring crowd.

The last boxer steps into the ring. He's got a cocky kind of smile on his face. He saw this kind of fighter in his last match, or at least, he's pretty sure it's the same, no it will definitely be the same. He knows exactly what to do. Come out aggressively. Be first. Hold nothing back and finish it quickly. At the sound of the bell, hell releases its fury. But at the end of two rounds, the other guy is still standing and hell has run out of fire. Dripping sweat and worn out, the boxer realizes he should've bided his time and this guy is not like the other one. But it's too late and it's all over in the next round.

What went wrong? The older boxer had the experience to win the fight, and the youngest boxer knew to wait and see what his next moves should be. But the last fighter became so exhausted from his initial attack, that left him defenseless and unable to mount a new offense. This exhausted third boxer is the key to the "original antigenic sin".

In 1889-1890, there was a different pandemic where the "Russian flu" spread across the world causing an illness similar to influenza. This Russian flu was the original sin. Individuals born in 1875-1900 were exposed to this pandemic influenza strain and their bodies built up immunity to this particular subtype. Then during the 1917 pandemic, these persons would be between 18 and 43 years old, the most at-risk age category. Research done by Dr. G. Shanks and Dr. J. Brundage, and later work done by Dr. Alain Gagnon and his team suggests that the antibodies these individuals had against the Russian flu were not effective against the Spanish flu (Shanks and Brundage) (Gagnon, Alain, et al).

Despite this, their memory antibody response was faster and more intense than the new response, and essentially "out-competed" the new protective cells attempting to produce effective antibodies for the Spanish flu (Gagnon, Alain, et al). This memory antibody response is the third boxer's first attack, strong but ultimately ineffective and a waste of energy and

resources. This first wave left their bodies drained and susceptible to a secondary bacterial infection, for which they had no defense against, and the patients quickly succumbed to pneumonia (Shanks and Brundage). The body was essentially “trapped” by their inexperienced immune systems (Gagnon, Alain, et al).

So, why weren't the very young or the elderly affected nearly as much? After all, the elderly would have also experienced the same Russian flu strain. This is true; however, older individuals were more “immunologically experienced” and had “antigenic seniority” according to Gagnon (Gagnon, Alain, et al). They were the older boxers. Basically, their immune systems had been a couple rounds in the ring against the flu and knew every season brought new tricks and new players, even if they looked the same. Their immune systems were experienced enough to know the Spanish flu was different. They weren't overwhelmed by a preliminary and ineffective response that left them exhausted. Instead, new antibodies were created. Ones tailor made for the Spanish flu and no other. In addition to more experience fighting the flu, they also had more time to build up multiple portfolios of antibodies for bacteria and were less likely to die from a secondary infection (Shanks and Brundage).

For children who hadn't experienced the Russian flu before the 1918 pandemic, their immune systems weren't producing ineffective antibodies and instead created new antibodies that worked against the Spanish flu. They were the youngest boxer. The one new to the ring. They weren't weakened from an exhausting and useless first response and were able to overcome the infection.

Historical Context

Even though there are only hypotheses and guesses right now about the true nature of the virus's virulence, scientists and historians agree that the historical context surrounding the pandemic certainly contributed to its deadliness in the younger generation. Instead of death counts and warnings of influenza, war news filled most of the front pages of newspapers. Young men left for new places, either abroad or even to a faraway training camp in the U.S., and were contacted new bacterial strains that they had no protective antibodies for. Meanwhile, their grandmothers, grandfathers, and younger siblings stayed in their rural communities and were unlikely to contact novel strains of bacteria (Shanks and Brundage).

The conditions in trenches, training camps, and even industrial cities were perfect for spreading a fast-acting disease. Overcrowded. Unsanitary. Limited health services. Preparing for war quickly caused a massive influx of new soldiers to training camps. Recruits would move into half-finished camps, sleeping in tents with other men in cramped quarters (Jester, Barbara, et al). At Camp Dodge, IA, 13,000 recruits fell ill. A private named Arne Thompson helped dig the graves of 700 soldiers. According to his son, "He told me he remembered seeing the bodies of his comrades stacked like cordwood waiting for burial by the over-whelmed burial detail." When asked what he did during the Great War, Thompson simply responded, "I survived the flu" (Thompson).

With more than half the U.S. population living in cities already, the war industry only pulled more people in from rural communities. The population expanded too rapidly for housing to keep up. As many as four would share an apartment. Residents in boarding houses shared rooms and beds. The day shift slept through the night and swapped places with the night shift in the morning (Jester, Barbara, et al).

In addition to living conditions, health services were limited. Up to 30% of U.S. physicians were in military service, and medical technology was almost non-existent. Doctors didn't even know influenza viruses existed. The general conscious was that the illness was caused by a bacterium called "Pfeiffer's bacillus", so the development of vaccines, antibiotics, antiviral drugs, mechanical ventilation, intensive care support, and diagnostic tests were all years or decades away (Jordan).

While some cities implemented mitigation measures that encouraged social distancing like closing schools or banning public gathering, there was no coordinated pandemic plan among the national or state level (Jordan). Even when experts warned of the dangers of waiting to enforce quarantine measures, science still bowed to the politics of a nation at war. Many civil service personal believed news of a deadly influenza would dampen morale and cause wartime productivity to suffer. They pressured public health officials into delaying pandemic measures, which had disastrous consequences.

Despite several physicians and health personal urging the cancellation of the Liberty Loan Parade, the Director of Philadelphia's Department of Public Health refused to cancel or warn the public regarding the risk. Two days after the parade, the Director announced that the influenza epidemic in civilian populations was the same as the army and naval stations, which were completely overrun with deaths (Jester, Barbara, et al).

Conclusion and Comparisons

I would be naïve to write this essay explaining the Spanish flu pandemic and not consider the current pandemic of COVID-19 raging across the world at the time of this writing. A simple Internet search yields thousands of results comparing the two pandemics and what we should

learn from the mistakes of the past. It's true. Mistakes were made in 1918 that we would be better off to avoid. For example, when health experts caved to politicians, death often followed. In St. Louis, where social distancing and lockdowns were enforced, the death was half the death toll in Philadelphia to see the necessity for strict measures (Hearne).

Unfortunately, a true comparison of the two pandemics can only be completed when the COVID-19 pandemic runs out of steam, either through the development of a vaccine or when enough immune systems have effective antibodies to secure herd immunity, and the virus dies out.

So, what does the 1918 pandemic tell us about our own? As I wrote this essay, I had siblings, parents and friends ask me all kinds of comparison questions.

How long did the 1918 pandemic last? (Spring 1918-Spring 1919.) (Jordan)

Do you think there will be more waves of the coronavirus? (Unclear, but most likely.)

What was the fatality rate for the Spanish flu? (Exact is unknown but estimated to be 2.5%) (Jester, Barbara, et al).

Wait, what is the mortality rate for COVID? (Wide ranges based on country, age, and preexisting conditions. Current estimates for the U.S. indicate roughly 3.2%) (Roser, Max, et al)

What? And how many people died from the Spanish flu? (More than 50 million)
(Johnson and Mueller)

Then the fear and panic sets in.

While a good degree of caution is certainly recommended when dealing with a new influenza strain, I came across a major difference between the 1918 pandemic and the current COVID-19 pandemic while researching for this article.

Information.

Over and over again, article after article bemoaned the lack of information, reports, or documentation for the 1918 pandemic. The case-fatality rate? According to one article, “Estimates of the case fatality proportion are difficult to compare because it was not always clear if the denominators included only symptomatic individuals, both asymptomatic and symptomatic infected persons, or the overall population at risk.” The 2.5% is just one estimate. In fact, one English physician estimated an 8% fatality rate based on his treatments of thousands of patients (Jester, Barbara, et al).

The number of people that died? Estimates range from 50 million to 100 million. That paper notes that, “Limitations of these data can include nonregistration, missing records, misdiagnosis, and nonmedical certification, and may also vary greatly between locations” (Johnson and Mueller).

Another paper notes that “The exact extent of morbidity and mortality from the 1918 pandemic is unknown because without laboratory confirmation, influenza is indistinguishable from other respiratory diseases” (Jester, Barbara, et al). Once cities realized the extent of the pandemic, they began to require physicians to report cases, but doctors were so overwhelmed with patients that timely or accurate documentation was all but impossible (Jester, Barbara, et al). “In the January 1919 American Journal of Public Health issue, the editor wrote that data in many cases were incomplete and confused as ‘so great was the pressure for action, that very few were able to devote any time to observation for the sake of the future’” (Jester, Barbara, et al).

Even when overwhelmed by the ill and dying, health officials still had to bow to military and political interests. In a review of the Spanish flu affecting the American military in World War I, the author reports that governments of “belligerent” countries may have edited the counts of illness for the sake of morale or wartime appearances (Jester, Barbara, et al).

Today, information regarding the COVID-19 pandemic besieges ordinary citizens. There are constant updates and reports on the number of cases and deaths that include details such as age, prior health conditions, location, and travel history (Roser, Max, et al).

The flow of news never seems to stop, and while it is certainly stressful, it is also an advantage. While comparisons to the 1918 pandemic can be useful in some regards, we also need to remind ourselves the modern world is a vastly different place, especially with regards to healthcare. Many countries now have access to antibiotics, antiviral drugs, mechanical ventilation, intensive care support, and diagnostic tests to help fight this disease. Even though the development of a vaccine may take time, at least we know it will one day be an option.

The important takeaway from this is that this information is constantly changing. We do not have enough information about the current pandemic to start making comparisons, and even if we did, we still don't know enough about the 1918 pandemic.

Staying informed is important.

Being cautious is important.

Learning from pandemics of the past is important.

But panicking over a repeat of the Spanish flu isn't beneficial, because it is a different virus in a vastly different world.

Works Cited

- “1918 Historical Image Gallery.” *Centers for Disease Control and Prevention*, U.S. Department of Health & Human Services, 2 Mar. 2018, www.cdc.gov/flu/pandemic-resources/1918-commemoration/historical-images.htm.
- Andrews, Evan. “Why Was It Called the 'Spanish Flu?'” *History.com*, A&E Television Networks, 12 Jan. 2016, www.history.com/news/why-was-it-called-the-spanish-flu.
- Brownell, Dave. “Amos Brownell and Mary Palmer Hensley.” *Centers for Disease Control and Prevention*, U.S. Department of Health & Human Services, 19 Sept. 2018, www.cdc.gov/publications/panflu/stories/memorial_brownell.html.
- Bouvier, Nicole M, and Peter Palese. “The biology of influenza viruses.” *Vaccine* vol. 26 Suppl 4,Suppl 4 (2008): D49-53. doi:10.1016/j.vaccine.2008.07.039
- Gagnon, Alain, et al. “Age-Specific Mortality During the 1918 Influenza Pandemic: Unravelling the Mystery of High Young Adult Mortality.” *PLOS ONE*, Public Library of Science, 5 Aug. 2013, www.journals.plos.org/plosone/article?id=10.1371%2Fjournal.pone.0069586.
- Gawron, Valeria. “Joseph and Stanley Garas.” *Centers for Disease Control and Prevention*, U.S. Department of Health & Human Services, 20 Sept. 2018, www.cdc.gov/publications/panflu/stories/memorial_garas.html.
- Hearne, Shelley. “A Tale of Two Cities: The Catastrophic 1918 Flu Pandemic Can Guide City Policymakers Today.” *CityHealth*, De Beaumont Foundation and Kaiser Permanente, 5 Dec. 2018, www.cityhealth.org/blog/2018/12/5/a-tale-of-two-cities-the-catastrophic-1918-flu-pandemic-can-guide-city-policymakers-today.

History.com Editors. "Spanish Flu." *History.com*, A&E Television Networks, 12 Oct. 2010,
www.history.com/topics/world-war-i/1918-flu-pandemic.

"How the Flu Vaccine Works." *NHS*, National Health Services, 16 July 2019,
www.nhs.uk/conditions/vaccinations/how-flu-vaccine-works/.

Jester, Barbara, et al. "Historical and Clinical Aspects of the 1918 H1N1 Pandemic in the United States." *Virology*, Academic Press, 16 Nov. 2018,
www.sciencedirect.com/science/article/pii/S0042682218303313?via%3Dihub.

Johnson, NP and J Mueller. "Updating the Accounts: Global Mortality of the 1918-1920 'Spanish' Influenza Pandemic." *Bulletin of the History of Medicine*, U.S. National Library of Medicine, 2002, www.ncbi.nlm.nih.gov/pubmed/11875246.

Jones, Marian Moser. "The American Red Cross and local response to the 1918 influenza pandemic: a four-city case study." *Public health reports (Washington, D.C. : 1974)* vol. 125 Suppl 3, Suppl 3 (2010): 92-104. doi:10.1177/00333549101250S312

Jordan, Douglas. "The Deadliest Flu: The Complete Story of the Discovery and Reconstruction of the 1918 Pandemic Virus." *Centers for Disease Control and Prevention*, U.S. Department of Health & Human Services, 17 Dec. 2019, www.cdc.gov/flu/pandemic-resources/reconstruction-1918-virus.html.

Kopp, John, and Bob McGovern. "100 Years Ago, 'Spanish Flu' Shut down Philadelphia – and Wiped out Thousands." *PhillyVoice*, WWB Holdings, LLC, 12 Mar. 2019,
www.phillyvoice.com/100-years-ago-spanish-flu-philadelphia-killed-thousands-influenza-epidemic-liberty-loan-parade/.

Piper, Alice A. "Evelyn Cox Flood." *Centers for Disease Control and Prevention*, U.S.

Department of Health & Human Services, 20 Sept. 2018,

www.cdc.gov/publications/panflu/stories/memorial_flood.html.

Racaniello, Vincent. "Structure of Influenza Virus." *Virology Blog: about Viruses and Viral*

Diseases, 2 Feb. 2011, www.virology.ws/2009/04/30/structure-of-influenza-virus/.

Roser, Max, Hannah Ritchie and Esteban Ortiz-Ospina. "Coronavirus Disease (COVID-19) –

Statistics and Research." *Our World in Data*, 4 Mar. 2020,

www.ourworldindata.org/coronavirus.

Shanks, G Dennis, and John F Brundage. "Pathogenic Responses among Young Adults

during the 1918 Influenza Pandemic." *Emerging Infectious Diseases*, Centers for Disease

Control and Prevention, Feb. 2012, www.ncbi.nlm.nih.gov/pmc/articles/PMC3310443/.

Shreeve, Jamie. "Why Revive a Deadly Flu Virus?" *The New York Times*, The New York Times,

29 Jan. 2006, [www.nytimes.com/2006/01/29/magazine/why-revive-a-deadly-flu-](http://www.nytimes.com/2006/01/29/magazine/why-revive-a-deadly-flu-virus.html)

[virus.html](http://www.nytimes.com/2006/01/29/magazine/why-revive-a-deadly-flu-virus.html).

Taubenberger, Jeffery K, and David M Morens. "The pathology of influenza virus

infections." *Annual review of pathology* vol. 3 (2008): 499-522.

doi:10.1146/annurev.pathmechdis.3.121806.154316

Thompson, David A. "Arne Thompson." *Centers for Disease Control and Prevention*, U.S.

Department of Health & Human Services, 9 Apr. 2013,

www.cdc.gov/publications/panflu/stories/warstories_thompson.html.

“Types of Influenza Viruses.” *Centers for Disease Control and Prevention*, U.S. Department of Health & Human Services, 18 Nov. 2019, www.cdc.gov/flu/about/viruses/types.html.

Vergano, Dan. “1918 Flu Pandemic That Killed 50 Million Originated in China, Historians Say.” *National Geographic*, National Geographic Partners, LLC, 29 Feb. 2020, www.nationalgeographic.com/news/2014/1/140123-spanish-flu-1918-china-origins-pandemic-science-health/.

Vidyasagar, Aparna. “What Are Viruses?” *LiveScience*, Future US, Inc, 6 Jan. 2016, www.livescience.com/53272-what-is-a-virus.html.