

SCOWCROFT INSTITUTE OF INTERNATIONAL AFFAIRS WHITE PAPER • 2021

LESSONS FROM **PAST EPIDEMICS AND PANDEMICS**





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INTRODUCTION

The COVID-19 pandemic was the first major, truly global pandemic since the Great Influenza pandemic of 1918 and, as such, drew numerous comparisons to the infamous pandemic a century ago. These comparisons also prompted people to speculate whether we had learned anything about pandemic response since 1918. The short answer to this is "yes." We have made tremendous scientific advancements and improved communication and technology.

Incidentally, however, there were lessons we didn't learn. Disagreement over things like face masks and social distancing in 2020, were reminiscent of reactions 100 years earlier. The federalist system, which extends to the American public health system, also created similar patterns in the uniquely different paths that response and case numbers took in different states. In 2020, as in 1918, some states opted for stricter measures than others and some states experienced worse outbreaks than others -- sometimes as a direct result of pandemic response policies and sometimes for entirely different reasons. Such similarities demonstrate that, while we have come a long way, there is still progress to be made.

While the 1918 pandemic is remembered as the most terrible and destructive pandemic in modern human history, there are other notable epidemics and pandemics that have occurred over the last 100 years that can offer insight as we look to future pandemics beyond COVID-19. In this paper we sought to examine several critical epidemics and pandemics, including the 1918 flu, the 2003 SARS outbreak, the 2009 H1N1 pandemic, and the 2014 Ebola outbreak in West Africa. In examining these events, we explored the in-country government responses, miscommunication and misinformation, and focused on the response within the United States. We also specifically examined the response from the State of Texas in each of these outbreaks. In some instances there were no reported cases in Texas and in others there were. It is our hope that through analyzing these previous epidemics and pandemics, we can expand our knowledge and better prepare the world, the country, and our state for the next pandemic.

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LESSONS FROM PAST EPIDEMICS AND PANDEMICS

To say, "I have the flu" is common in American culture. Most of the time when this phrase is used, however, the individual does not, in fact, have the influenza virus. Instead, they are more likely infected with a rhinovirus or coronavirus. There are over 100 viruses that cause the illness known as the common cold, but rhinoviruses and coronaviruses make up 70 percent of cases. Influenza, or "the flu," is a very different virus. Many of the most notable modern pandemics have been caused by influenza, though coronaviruses have become increasingly deadly with the emergence of SARS, MERS, and SARS-CoV-2. Below is a table that outlines the basic estimates of four past epidemics and pandemics.

Pandemic	Causative Agent	Cases per Million	Deaths per Million
1918 Influenza	H1N1 influenza virus	333,333	33,333 - 60,000
2003 SARS	SARS coronavirus	1.27	0.12
2009 H1N1	H1N1 influenza virus	291,003	41.3
2014 Ebola	Ebola Zaire	3.9	1.55

1918 Influenza

Waterfowl serve as the primary reservoir for all influenza viruses, but most influenza viruses that infect humans experience recombination events with animals more similar to humans (Kim et al., 2009). In particular, pigs have the ability to assist influenza viruses in becoming more transmissible from human-to-human (Ma et al., 2009). How the 1918 flu spilled over into the human population is still a mystery, but many scientists believe that it did not start as a disease in pigs and then spillover to humans, but rather it was in humans and then spilled over into pigs (Taubenberger, 2006). Today we also understand that the 1918 pandemic was caused by a H1N1 virus and is estimated to have killed 50-90 million people worldwide and approximately 675,000 in the United States (Johnson & Mueller, 2002). The virus also had a disproportionate impact on healthy individuals between the ages of 15-34 (Jester et al., 2018).

The origins of the 1918 influenza pandemic are not officially known, but a leading theory is that the virus emerged in Haskell County, Kansas and became recognized at Camp Funston in Fort Riley, Kansas. The first known case occurred on March 11, 1918 and the spread of the virus was likely aided by global troop movements over the months of March and April in 1918.

The French army reported their first cases in April 1918, but overall the impact on French soldiers in the spring of 1918 was small (Erkoreka, 2010). About a month later the virus had reached Spain and caused significant mortality. The impact of the pandemic in Spain and the country's willingness to openly discuss the pandemic are the primary reasons it was termed the "Spanish flu" (Erkoreka, 2010). By June or July of 1918, the virus had arrived in Portugal, leading to a number of outbreaks in cities across the country.

Entering the fall, the 1918 flu virus hit France, Spain, and Portugal harder than it had in the spring. Within the United States it had killed 195,000 Americans by October 1918 alone (CDC, 2018), but the major peaks didn't come until October and November. A second wave emerged in the fall of 1918 and a third wave occurred in the spring of 1919. The virus is even believed to have infected President Woodrow Wilson at the Versailles Peace Conference in April 1919 (CDC, 2018). Cases continued into the first months of 1920, but the age distribution began to level out with greater numbers of deaths among children under 5 years of age and adults over 60 years of age (Erkoreka, 2010). The change in age distribution made the 1918 pandemic flu more similar to seasonal influenza compared to the earlier pandemic waves. In 1921 there is evidence that just over 200 people in Madrid died of the 1918 flu virus, but overall the pandemic was over by that time (Erkoreka, 2010). Despite its devastation, however, it was left out of the history books and has occasionally been referred to as the "forgotten pandemic" (Crosby, 2003). Why the pandemic was forgotten in the years between the 1920s and the 1970s is unclear, but in the 1970s historians, policy makers, and scientists began to take interest in the catastrophe.

During the pandemic's forgotten period, there were some scientists that sought to learn more about the virus. One in particular was Johan Hultin, a researcher from Sweden who obtained permission from leaders in a remote Inuit village that had been hit hard by the 1918 flu to excavate the burial site for those who had died from the flu (Jordan et al., 2019). After days of excavation, Hultin obtained tissue samples from the lungs of four individuals. The technological and scientific barriers of the 1950s prevented Hultin from learning much from the samples, but decades later, in 1997, Dr. Jeffrey Taubenberger and his team successfully sequenced the 1918 flu genome, or the complete set of viral RNA, after collecting tissue samples from a young man from South Carolina who had died of the virus (Jordan et al., 2019). Following the publication of their findings, Dr. Hultin contacted Dr. Taubenberger to see if he would be interested in additional lung tissue samples from the Alaskan village he had visited almost 50 years previously. Upon Taubenberger's answer, Hultin traveled to the village and obtained a perfectly preserved lung tissue sample from a woman who likely died in her mid-20s (Jordan et al., 2019).

From the newly obtained samples, discoveries were made about the 1918 flu including its "striking ability to quickly replicate" (Jordan et al., 2019). Researchers found that the amount of virus in the lung tissue of mice was 39,000 times higher with the 1918 virus than with one of the other comparison flu viruses (Tumpey et al., 2005). The virus also had a much more devastating effect on chicken embryos than did other closely related H1N1 viruses (Jordan et al., 2019; Tumpey et al., 2005). The most important thing that was learned from the experiments done on the extracted 1918 virus was that its lethality did not come from one single gene, but rather from the unique combination of genes (Jordan et al., 2019). The discoveries made by Drs. Hultin, Taubenberger, Tumprey, and other scientists helped to explain why the virus had been so deadly and how it spread easily in the crowded conditions of WWI.

The Role of Misinformation

The 1918 flu emerged during the First World War and this timing had a large impact on discussion of and reporting on the virus. The most commonly known impact of this is the fact, discussed above, is that it was termed the "Spanish flu." Due to extensive wartime censorship, Allied and Axis powers censored information about the virus and its spread. When the virus was discussed, it was often downplayed or outright false information was printed. A newspaper in London stated that the flu threat was not serious and the British government said it was "unpatriotic" to be concerned about the flu during wartime (Little, 2020). The United States newspapers also downplayed the flu and President Woodrow Wilson resisted efforts to contain it (Little, 2020). Italy took it one step further and denied that there were any cases of the flu at all in the country during the fall of 1918 (Little, 2020).

Aside from a lack of accurate reporting or outright denial, there were conspiracy theories circulating. A newspaper in Rio de Janeiro, Brazil claimed that the Germans were traveling around the world in submarines and spreading the virus (Mawdsely, 2019). The Brazilian newspaper seemed to imply that the Germans had created a virus as a biological weapon of war. This was not the only conspiracy theory, however. Throughout the world there were a number of theories about what had led to the virus, including the tendency to blame it on foreigners and Jewish people (Mawdsely, 2019). Other theories claimed that the virus was the result of dancing, or Jazz music, or even "the bombing of the soil as a result of the war" (Mawdsely, 2019). In a world where scientific information was scarce and governments were largely removed from response, conspiracy theories about the virus filled the void.

1918 Influenza in the United States

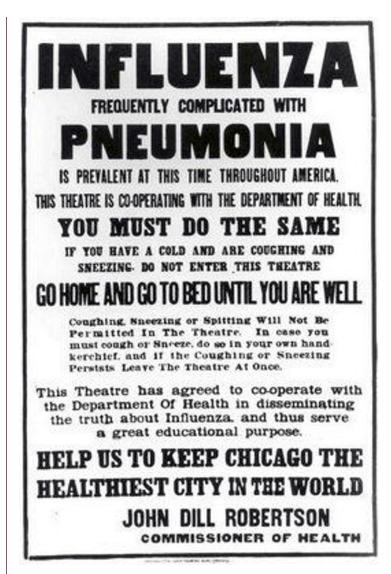
In early 1918, several young men from Haskell County reported to Camp Funston for basic training. Because



the Camp was a major training base for men preparing to deploy to Europe and the men there experienced crowded living and working conditions, the virus spread easily. By mid-March there were 1,100 men admitted to the hospital and 38 men had died (Barry, 2017). The movement of Camp Funston soldiers to other military camps across the country facilitated the spread of the disease. This initial outbreak in the spring of 1918 was small, however, compared to the outbreak several months later.

In September 1918, a second wave emerged at Camp Devens, located about 40 miles from Boston, and this wave proved to be more fatal than the first wave in the spring (CDC, 2018). It is estimated that approximately 15,000 soldiers in camp were infected and more than 800 died (Lepiarz, 2020). Outside the camp, the city of Boston was also impacted by the virus. In late September 1918 the Boston Globe wrote that hospitals in the area were at their limits (Lepiarz, 2020). By October 1918 the virus had taken hold in most of America's major cities. In Chicago, 8,510 people died from the virus in just 8 weeks (Fornek, 2005). The city, along with many other cities, closed live theaters, movie theaters, and schools (CDC, 2018). Chicago also discouraged kissing and outlawed public dancing (Fornek, 2005). The city canceled football games and banned gatherings, such as union meetings (Fornek, 2005). Social distancing became a standard measure in many cities fighting 1918 influenza. Additionally, numerous cities, including San Francisco which suffered 3,500 deaths (PBS, n.d.), required anyone working to wear a mask and encouraged all residents to also wear masks (CDC, 2018). In November 1918, Salt Lake City guarantined infected individuals by placing signs on their doors (CDC, 2018).

Though many closures, social distancing, and quarantine measures were put in place in the fall of 1918, Americans quickly became tired of staying home (Baskar & Kwong, 2020). In a study of 43 cities across the United States during the 1918 flu pandemic, it was found that 23 of those cities decided to reopen - or return to normal before cases were in sustained decline (Baskar & Kwong, 2020). Those 23 cities experienced a second epidemic of 1918 flu, whereas the 20 cities that remained closed for longer did not. Additionally, cities that implemented closures, social distancing, and quarantine measures early and kept them in place a sufficient amount of time



A Chicago Public Health poster outlines flu regulations during the pandemic. (Photo Credit: origins.osu.edu) PER OSU: Source: Public Domain

for cases to experience sustained decline, experienced less economic impact than those that opened businesses and removed social distancing requirements before there was a sustained decline in cases (Correia et al., 2020). Cities that implemented early and extensive non-pharmaceutical interventions, such as closures, had lower mortality rates and higher economic growth following removal of the measures (Correia et al, 2020). Specifically, cities that implemented early and forceful non-pharmaceutical interventions experienced increases in manufacturing employment, manufacturing output, and bank assets after the pandemic had ended (Correia et al., 2020), whereas states that removed non-pharmaceutical interventions before the virus was sufficiently controlled did not experience the same level of economic growth.

Philadelphia and Seattle provide contrasting examples of how non-pharmaceutical interventions impact outcomes from the 1918 flu. Philadelphia resisted non-pharmaceutical interventions until the virus was widespread, whereas Seattle implemented interventions early and kept them in place until cases had experienced a sustained decline. Philadelphia witnessed the damage caused by the virus in Boston, but the city did not take any steps to protect itself and, instead, held its Fourth Liberty Loan parade as originally planned (Davis, 2018). Within weeks, Philadelphia was overwhelmed with the size of the epidemic they were experiencing. Morgues overflowed (CDC, 2018) and there was a shortage of coffins (Bristow, 2020). At the end of the pandemic, Philadelphia suffered one of the highest death rates from the 1918 flu in the country (Barry & Dickerson, 2020).

By contrast, the city of Seattle began taking precautions before the virus arrived. In late September 1918, the Commissioner of Health told city residents that if the virus appeared, they would need to isolate people (Bristow, 2020). When the first cases appeared at Fort Lewis in Tacoma, WA, the entire camp was quarantined. Shortly after, more cases appeared at a naval training station at the University of Washington (Bristow, 2020). Within two days of the discovery of these cases, Seattle closed "churches, schools, and shows" and banned "public assemblies" (Seattle Daily Times, 1918). Additionally, the city prohibited public spitting, making it punishable by jail time, required the wearing of masks in public, and put restrictions on the number of hours



a business could be open. Experts hypothesize that the actions taken by the city of Seattle is one of the reasons that it had one of the lowest death rates (Bristow, 2020).

While these are just two examples of differing responses, other research has found that the early implementation of measures like those taken by Seattle, reduced transmission rates by 30-50% (Pambuccian, 2020). Additionally the amount of time such measures were kept in place, correlated with reduced total mortality (Markel et al., 2007).

1918 Flu in Texas

When the 1918 flu reached Texas, some areas implemented more stringent non-pharmaceutical interventions than others and, as a result, experienced markedly different mortality outcomes. By most measures, Texas was largely spared from the worst of the 1918 pandemic with an estimated 106,000 cases statewide and around 2,000 total deaths (Hlavaty, 2018). The hardest hit areas appear to have been North Texas, Houston, and San Antonio.

In Sherman, a town located north of Dallas, the virus likely appeared some time before officially reported, with initial cases misdiagnosed (Redshaw, 2013). When the outbreak was recognized as influenza, the local newspaper printed a series of precautions which included avoiding crowds, wearing masks, frequent handwashing, and consulting a doctor when symptoms presented (Sherman Daily Democrat, 1918). In addition to the public health advice shared by the newspaper, a grocery store also ran an advertisement which falsely claimed their store was free of influenza because flu germs were only able to live in dirt (Redshaw, 2013). The town of Sherman imposed a quarantine in September 1918, but it was lifted a few weeks later.

In Houston, the virus hit Camp Logan from September 13 to October 8, 1918 (Hall et al., 1918). As a result of this outbreak, 2,487 individuals were admitted to the base hospital for influenza infection. Of those admitted for influenza, 416 developed pneumonia of which approximately 75 died (Hall et al., 1918). As a result of the outbreak at Camp Logan, the State Health Board instructed area schools to sweep and disinfect floors daily, clean desks, chairs, tables, and doors with linseed, kerosene, and turpentine, and stated that spitting on the floor or coughing and sneezing without covering one's mouth would lead to suspension (Medley, 2020). Isolation, quarantine, the use of disinfectants, and the banning of public gatherings were widespread throughout the city in the fall of 1918. Overall, Houston experienced an estimated 111 deaths from the 1918 flu (CDC, n.d.j), the majority of which were concentrated at Camp Logan.

Disease containment measures in San Antonio and the surrounding areas experienced the greatest resident pushback against public health measures compared to other parts of the state. In October 1918, when the area had its first spike in cases, there were bans on public gatherings, school closures, prohibition against church services, and closures of many businesses (Sauer, 2020). Restrictions were eased after a few weeks and Seguin and the rest of the San Antonio area experienced another spike in December. The city again issued closures and bans on gatherings, but residents rebelled against the orders (Sauer, 2020). It is estimated that by January 1919 the city had between 12,000 - 86,000 cases as a result of removing the social distancing orders before cases had experienced a sustained decline.

THE 1957 INFLUENZA PANDEMIC

The first case in the 1957 H2N2 influenza pandemic was reported in Singapore in February (CDC, 2019). A few months after this initial reported case, Hong Kong also reported cases of the virus. The cases in Hong Kong quickly became a major epidemic with approximately 250,000 infections (Honigsbaum, 2020). Over the next several months the virus moved westward, appearing in India in June and causing more than one million cases. Shortly thereafter the United States experienced their first cases.

As the 1957 virus spread across the world it raised high levels of concern in the public health and medical communities because genetic testing of the virus showed that it was unlikely to have been previously found in humans (Kilbourne, 2006). And while the virus was deadly, killing an estimated 1.1 million worldwide and 116,000 in the United States (CDC, 2019), it was not as severe as the 1918 flu.

The United States' response to the 1957 pandemic really began before the virus even reached the country. In the spring of 1957, a microbiologist at the Walter Reed Army Institute of Research named Maurice Hilleman, was concerned by what he was seeing in Hong Kong and obtained a specimen of the virus to study (Zelovich, 2020). As a result of this early research, vaccine development was already well underway by the time the virus reached the United States and on July 26, 1957 doctors began vaccinating recruits at Fort Ord in California (Zelovich, 2020). In fact, by the time the virus began spreading in the United States during the summer of 1957, factories in the United States had already produced 40 million doses of the vaccine (Zelovich, 2020). This quick action on the part of Dr. Hilleman helped the country largely avoid the pandemic.

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2003 Severe Acute Respiratory Syndrome (SARS)

In late 2002, reports of a "pneumonia-like" illness emerged around Foshan, China and the Guangdong Province. This mysterious disease, which would later be named Severe Acute Respiratory Syndrome, or SARS, was characterized by a fever followed by respiratory symptoms (Berger et al., 2004). Many of the symptoms were similar to influenza, with some severe enough to lead to respiratory failure and death. The severity of the SARS virus took many scientists and public health officials by surprise. Until the outbreak in 2003, coronaviruses that infected humans produced only mild symptoms and were not considered a serious infectious disease threat.

Throughout late 2002 and early 2003 SARS spread throughout China, into Hong Kong, and eventually, to

over a dozen countries throughout the world. Almost 20 years later, there is still no clear understanding of the origins of SARS, but two distinct genotypes have been identified. The first genotype is responsible for the infections in the Hotel M in Hong Kong, which led to a large number of infections in Canada, Singapore, Taiwan, and many other countries. The second genotype is responsible for the outbreaks throughout the rest of Hong Kong, the Guangdong Province, and Beijing. In addition, scientists determined that the genotype responsible for the worldwide SARS pandemic was "neither a mutant of a known coronavirus nor a recombinant between known coronaviruses" (Holmes, 2003: Berger et al., 2004). SARS is a previously unknown fourth lineage of the coronavirus family (Marra et al, 2003) and may even have split from the group two lineage several decades ago (Snijder et al., 2003).

CoV Group	Characteristics	Virus Examples
Alpha	Mostly mammalian viruses	HCoV-229E (human) HCoV-NL63 (human) Transmissible gastroenteritis virus (pig) Porcine epidemic diarrhea virus (pig)
Beta	Viruses that infect mammals, but can also infect rodents	Mouse Hepatitis Virus (mouse) Bovine Coronavirus (cow) SARS-CoV (human) MERS-CoV (human) SARS-CoV-2 (human)
Gamma	Mostly infect birds	Avian infectious bronchitis (chicken) Turkey coronavirus (turkey)
Delta	Mostly infect birds, but some mammals	Porcine coronavirus HKU15 (pigs) Sparrow coronavirus HKU17 Magpie robin coronavirus HKU18

Prior to SARS, there were no documented incidents of animal-to-human transmission involving coronaviruses, but the concept was not unthinkable. "Coronaviruses have a high frequency of mutation and a high frequency for recombination, which can result in the rapid development of new strains within an individual" (Romich, 2008). The ability of coronaviruses to mutate had hinted at their zoonotic potential long before the outbreak of SARS in 2003. It should be noted, however, that despite coronaviruses' usual plasticity and remarkable ability for mutation and recombination, all evidence indicates that the SARS coronavirus remained extremely stable throughout the SARS outbreak. There was little to no mutation in the strains recovered from patients worldwide.

Stability was an important feature of SARS. It maintained a consistent genotype as it moved through the population, which scientists suggested meant that it was well-adapted to humans (Ruan, 2003). Its excellent adaptation to humans suggests that SARS has most likely existed for many decades if not centuries, but has only recently achieved human-to-human transmission.

One interesting component of SARS transmission was that most patients did not transmit the virus to additional individuals. In other words, the majority of people infected with SARS never infected another person. What allowed SARS to spread so broadly was the fact that dozens of infected people acted as superspreaders. Individuals such as the Guangdong doctor, who infected most of his hotel floor, and the Hong Kong patient, who infected almost 200 people in a hospital, are what allowed SARS to maintain an R0 above 1. Without the presence of super-spreaders, it is possible that SARS would have gone unnoticed by the global community and would have died out on its own without ever spreading outside Guangdong province.

Response in China

During the earliest months of the SARS outbreak in China – November 2002 to February 2003 – the Chinese government did not acknowledge the outbreak and their denial of SARS extended to conversations with the international community. During these initial three months, China did not take any action to contain the spread of the virus. Additionally, there were a number of policies in place in 2003 that prevented healthcare



workers and local public health officials from reporting the disease. The National Law on Communicable Disease Prevention and Control, last revised in 1989, was the regulating authority for communicable disease in China at the time of the SARS outbreak (Balasegaram & Schnur, 2006). This law stated that specific notifiable diseases must be reported to varying levels of government depending on their severity. The list of diseases that were considered notifiable included diseases such as Yellow Fever and Typhoid Fever. Anything that was not a previously established disease was not included on this list. In the past, this system had worked well, but SARS was still being termed atypical pneumonia during the early months of the outbreak (November 2002-January 2003) and pneumonia was not a mandatory reportable disease. The law meant that the initial stages of the outbreak were dealt with by local health officials with no legal requirement to report it to any other level of government (Balasegaram & Schnur, 2006).

There was also the 1988 State Secrets Law, which prevented anyone other than the Ministry of Health from reporting an outbreak of infectious disease to the public or international community. More specifically, this law meant that health officials, or other governmental members, at lower levels of government could only report the SARS outbreak to the Ministry of Health. Reporting the outbreak externally would result in prosecution (Saich, 2006). If health officials in the Guangdong province had, on their own, reported that there was an outbreak of infectious disease in the region to the international media – or even the Chinese media – they were eligible to be punished by death. Another reason for the Chinese government's slow response to the new disease was a history of emerging influenza in southern China. Both the 1957 and the 1968 global influenza pandemics began in the region. Due to this history, some assumed that this may be another influenza outbreak. Distinguishing the SARS outbreak from influenza was further complicated by a small cluster of avian flu that occurred in Foshan around the same time as the initial SARS case (Kaufman, 2006). Although influenza pandemics can pose an extreme health risk, it was something the Chinese were accustomed to dealing with and the government likely viewed the disruption of the economic and political state of the country unwarranted.

In April the Chinese government acknowledged the extent of the SARS outbreak and began to take preventative measures in Beijing. Most of the control measures implemented in Beijing focused on "early identification and isolation of cases and reducing public crowding and the opportunities for transmission" (Kaufman, 2006, p. 61). The national government began to require daily reports from all provinces about new or suspected SARS cases and deaths (Kaufman, 2006). In late April, construction began on a SARS hospital in a rural county outside of Beijing. The purpose of the hospital was to contain all of the SARS patients in the area and prevent the disease from spreading in hospitals.

China also developed national infection control guidelines for healthcare workers to help prevent the spread of SARS from patients to healthcare workers and implemented a mandatory quarantine for suspected and confirmed SARS cases. The Chinese also implemented a series of daily checks desired to contain spread of the virus:

"The Beijing government also instituted morning fever checks for all students and established fever clinics to isolate and observe febrile persons, students and otherwise. Beijing cancelled most public gatherings and



closed elementary schools. The national government cancelled the annual week-long May Day holiday to minimize travel to and from Beijing, and instituted fever checks for travelers at major transportation points such as airports and bus and train stations." (Kaufman, 2006, p. 62)

These measures went beyond the recommendations of WHO, but did help to contain the epidemic within the country.

Response in Canada

SARS reached Canada in late February 2003. It entered the country when two different individuals arrived from Hong Kong with "atypical pneumonia" (Tyshenko, 2010). One infected individual returned to Toronto, Ontario and the other returned to Vancouver, British Columbia. After entering each city, however, the disease took different paths. SARS became a serious public health issue in Toronto, while in Vancouver, it was quickly contained.

During the early stages of the outbreak, physicians in Toronto were not aware of the disease, as the global alert from Health Canada's Global Public Health Intelligence Network had not been communicated to them. Why the mass communication network failed in this instance is not entirely clear. Regardless of the reason for failure, most found out about the disease, and the global health alert associated with it, through their own investigation into the strange cases of pneumonia appearing in local hospitals (National Advisory Committee on SARS and Public Health, 2003). Because there was little to no knowledge of the disease at the onset of the outbreak in Canada, few precautionary measures were taken in Toronto. In addition, many of the most severely ill patients had to be intubated, which proved to be a risky procedure for health care workers. There were several instances where doctors contracted the disease from a patient they had intubated even though they had worn personal protective equipment (PPE) during the procedure. It quickly became clear how infectious SARS was within hospitals, and Canadian officials began to implement strict containment procedures.

In late March 2003, Toronto hospitals began refusing new admissions and transfers from other hospitals. Hospital employees were banned from working at any other institution except for their current hospital assignment. These measures were taken in an attempt to keep SARS from spreading from one hospital to another. The ban on new admissions was another attempt to prevent susceptible individuals from coming into contact with those infected with SARS. Other emergency measures in the Toronto area allowed for patients to be tracked and for forced guarantines to be implemented if necessary. Most people complied with the guarantines without incident, but when there were individuals who resisted quarantine procedures public health officials resorted to legal means in order to get resistant individuals to comply (National Advisory Committee on SARS and Public Health, 2003). Hospitals also began keeping suspect and probable SARS cases in negative pressure rooms to decrease the chances of the disease spreading throughout the hospital. These rooms filled quickly and many Toronto hospitals had to build makeshift negative pressure rooms to accommodate the patients (Tyshenko, 2010).

Around this time, many of the hospitals dealing with SARS had declared a "Code Orange", meaning that they had suspended all non-essential services (National Advisory Committee on SARS and Public Health, 2003). At the height of the outbreak, healthcare practitioners received educational training on how to wear PPE and N95 masks properly. They also received training on how to remove them properly when leaving contaminated areas. During this time there was still confusion about whether "clean areas" were contaminated by the virus. Due to this confusion healthcare workers did not know what areas were safe to remove PPE. Additionally, there were not enough masks for all healthcare workers at the beginning of the SARS outbreak in Canada. When masks were available, they often did not fit properly. Ill-fitting masks offered little protection from the virus.

To control the virus outside of hospitals Toronto implemented work quarantines with a number of strict requirements. This included:

- Health care professionals had to wear a mask at all times while at work and practice good hand hygiene
- Healthcare workers had to commute alone. When this was not possible they had to wear a mask and sit on the opposite side of the vehicle from the other person.

- Healthcare workers could not enter any other hospital site except the one they were assigned
- Healthcare workers with other offices in the community were only allowed to see essential patients
- Healthcare workers had to monitor their body temperature twice a day and stop work immediately if they developed a fever
- Healthcare workers were only allowed to be at home or at work. They could not make stops anywhere in-between.
- Healthcare workers had to wear N95 masks around their family members
- Healthcare workers had to remain quarantined from other family members, if possible, and to sleep in a separate room from other family members
- Healthcare workers were not allowed to share any household items with other family members

By May 14, 2003, the World Health Organization removed Toronto from their list of areas that had recent local transmission of SARS (Tyshenko, 2010). This action was largely interpreted by Canada as an indication that the SARS outbreak in Toronto was over and the emergency SARS sanctions were lifted on May 17, 2003. Healthcare providers were no longer required to wear protective equipment when working with patients. Shortly after the emergency sanctions were lifted, on May 23, 2003, the North York General Hospital shut down due to the presentation of new SARS cases. During the days that followed 30 hospital staff members and doctors were admitted with SARS (Tyshenko, 2010). In total, this new outbreak included 11 probable cases of SARS and 41 suspected cases. These new cases resulted in approximately 2,200 people in self-quarantine.

The last new case of SARS in Toronto occurred on June 12, 2003, and towards the end of June only a handful of probable SARS cases remained in area hospitals (Tyshenko, 2010). Due to the steady decrease in SARS cases by mid-June, officials declared the Toronto outbreak over. From this declaration onward, Toronto's SARS cases continued to decline, and most of the hospitals in the area were fully functional again by September.

Vancouver took stringent containment steps from the beginning. The index patient in Vancouver, British Columbia returned from a trip to Hong Kong on March 7, 2003. During this return flight, he had begun to feel ill. Once back in Vancouver, his symptoms intensified. His family practitioner, unsure about the specific cause of the patient's illness, directed him to the emergency room at Vancouver General Hospital. Within approximately 5 minutes of his arrival, he was moved from the waiting room to a small room where he could be kept in isolation. Within 2 hours of his arrival, he was moved to a negative pressure room. All healthcare workers that assisted him during this time wore N95 masks and none of them became infected. The hospital staff was unaware that he was a SARS patient, but they had heard of a new virus that had originated in Asia. As a result of these initial precautions, the virus never spread any further in the Vancouver region.

Response in Texas

Though Texas had no confirmed cases of SARS the state did have a number of suspected cases (CDC, 2003). The first two suspected SARS cases in Texas were reported in April 2003 in Collin County (Evans, 2020). Within two weeks, additional suspected cases were reported in Fort Bend, Lubbock, Travis, and Harris counties (Midland Reporter Telegram, 2003). All six of the initial suspected cases were travel-related and those suspected of infection were asked to self-quarantine.

As of the end of April 2003, Harris County was looking into 11 suspected SARS cases, but continued to stress that there was limited risk to the general public (City of Houston, 2003). The City of Houston provided information about SARS symptoms to the public, as well as a breakdown of what classifies an individual as a probable case vs. a suspect case. Over the next month the State of Texas continued to track suspected cases of SARS and requested that individuals labeled as a suspected case quarantine themselves at home.

The first probable SARS case in Texas occurred in Travis County in early June 2003 (Reed, 2003). The individual labeled as a probable case did not require

hospital admission, but was quarantined at home and monitored by the health department. Given that this was the first probable case and there had only been 8 suspect cases at this time, local and state health departments continued to communicate that there was minimal risk to the public and no large-scale precautions were taken (Reed, 2003). Approximately a month later, as the global SARS outbreak was drawing to a close, 11 members of the Air Force stationed at Dyess Air Force base were quarantined in their homes for respiratory symptoms similar to SARS (Starr, 2003). Three of the airmen had recently traveled from Toronto, which elevated the concern among public health officials, but none of the individuals tested were positive for SARS. During the time that testing was occurring, the Texas Department of Health reiterated that it was unlikely the airmen had SARS, but they were being tested out of an abundance of caution (Starr, 2003).

Role of Misinformation

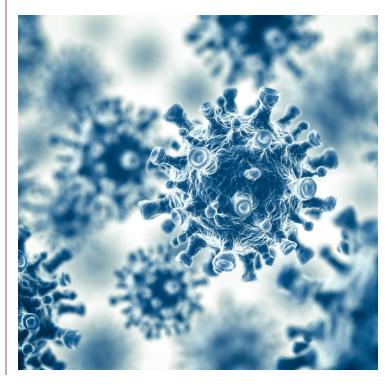
Almost as soon as the world started discussing SARS, misinformation about its origins emerged. Rumors inside Beijing began to spread that Chinese officials were planning to "seal off" the city and declare martial law (Chiu, 2003). Additional rumors claimed that officials were planning to disinfect the city with airplanes and that SARS was actually a biological weapon developed by the United States and Taiwan (Chiu, 2003). The bioweapon theory, in particular, gained traction after two Russian scientists stated that they believe SARS had been created in a lab (Chiu, 2003; Jennings, 2008). Some Chinese also believed that SARS was a US plot to distract China from war with Iraq (Chiu, 2003). Despite the false nature of these claims, they spread fairly widely throughout the country.

Outside of China, misinformation also spread. Some believed that SARS had been created to prepare the world for a future when disease would kill most of the population, while others thought SARS had been created by the ultra-wealthy as a form of global population control (Santa Clara University, 2003). There were also many that believed SARS was a hoax designed to make medical and pharmaceutical companies money. Since SARS, the idea that infectious diseases are hoaxes designed to make pharmaceutical companies money has continued to be one of the more commonly spread falsehoods.

2009 H1N1

In 2009, a new influenza virus that is believed to have undergone a triple reassortment with different swine viruses, emerged in Mexico. Swine play an important role in interspecies transmission of influenza and are oftentimes the middle point between an avian virus and a virus that is capable of infecting humans. H1N1, the influenza strain responsible for the 2009 outbreak, was first isolated in 1930. Since this time, scientists have been able to demonstrate that all strains of H1N1 known to be circulating in the United States have a common ancestor and antigenic similarities to that 1918 H1N1 flu (Tumpey et al., 2004). This means that all modern H1N1 influenza strains are in some way descended from the 1918 virus that killed millions.

Over the course of 3 months the 2009 virus worked its way across the world, eventually infecting people in over 40 countries. Thousands fell ill and there was a great deal of concern within the public health community that it may become this generation's 1918 flu. In 2009, a disproportionate number of young people fell ill and were hospitalized due to infection with the virus, similar to the 1918 flu. With seasonal influenza, the highest risk groups are young children or elderly people. By 2010 the virus is believed to have infected approximately 2 billion people worldwide and killed just over 230,000.



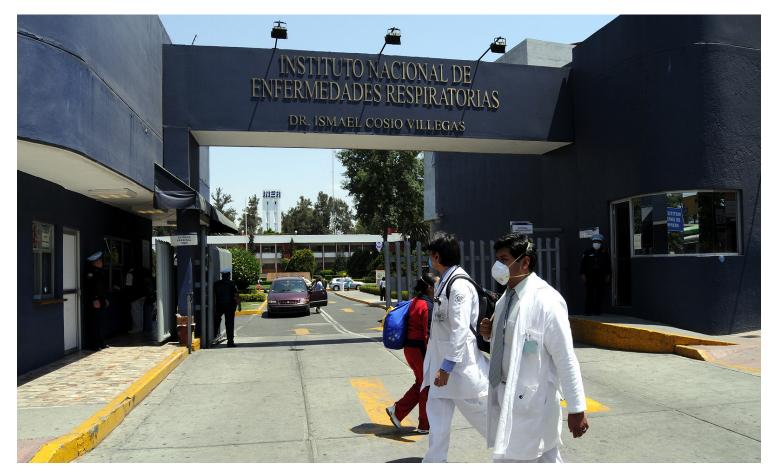
Response in Mexico

In mid-February 2009, in the town of La Gloria, Veracruz, Mexico, there was a cluster of "influenza-like respiratory illness." The respiratory illness continued to circulate in the town in early spring. At the time, many residents of the town believed that the illness was most likely caused by air and water contaminated from some of the local pig farms.

The first H1N1 death occurred in mid-April when a 39-year-old woman was hospitalized in Oaxaca, Mexico for acute respiratory disease. Health officials conducted contact-tracing but they determined the death of the woman was an isolated incident. By April 16, 2009, the Oaxaca Health Department began experiencing an unusually high number of atypical pneumonia cases. Realizing that it was likely something more serious than atypical pneumonia, Mexico contacted the Pan-American Health Organisation regarding the cases. Given that the SARS outbreak had occurred only six years prior, there were initially rumors that the cases of atypical pneumonia may be a coronavirus spreading throughout the hospital. Samples were sent to Mexico City to be identified.

On April 21, the Oaxaca Health Department confirmed a second death from the mysterious atypical pneumonia and established a quarantine of the emergency room at Hospital Civil Aureolio Valdivieso. Around this time health authorities also determined that neither a coronavirus or avian influenza were responsible for the deaths, and they hypothesized that the culprit was an "unspecified bacterial pathogen" (Crudo, 2015). This speculation led the Oaxaca State Congress' Permanent Committee on Health to begin an investigation into the deaths and for the National Ministry of Health to issue a health alert regarding the cases. It was also reported that 16 employees at the hospital had come down with a respiratory disease.

The high number of cases at the hospital sparked interest at the Instituto Mexicano del Seguro Social. They began sending patients showing respiratory or pneumonia-like symptoms to the epidemiological department for further study. It was decided that the



hospital's emergency room had to remain closed for 15 additional days so that preventative disinfection measures could be taken. They believed that this additional closure time would help to ensure that the disease, whatever it was, would not continue to spread within hospitals, as SARS had in 2003.

On April 23, 2009 government officials closed schools in Mexico City in order to prevent further spread of the disease. They also worked to limit public gatherings and got confirmation from Canada that samples they sent to a laboratory there were a novel strain of H1N1. Despite public awareness campaigns and school closures, by April 25, 2009 Mexico had identified approximately 1,300 people suspected of having H1N1. This large number led Mexican president, Felipe Calderon, to issue an emergency decree.

Throughout the rest of April 2009 and the beginning of May 2009, Mexican officials cancelled public events and encouraged people to avoid public gatherings. On May 1, 2009, Mexico began a five-day shutdown of most businesses throughout the infected areas of the country. As of May 11, there had been 48 deaths in Mexico, a number that increased throughout the next few months. The H1N1 outbreak continued internationally until late June, but much of the worst was over for Mexico in late May. Isolation and guarantine procedures were relaxed, businesses were allowed to reopen, and citizens were not mandated to avoid public gatherings. The strategy led to both social and economic consequences. Economically, Mexico lost 0.3 percent of its \$1 trillion GDP (Stevenson, 2010). Most of this loss was suffered in the tourism industry. The social cost of the Mexican response came mostly from political opponents of the President's party. They considered the threat to be exaggerated and accused the president of overreacting. These charges led many in Mexico to wonder if the level of the threat had been fabricated.

Response in the United States

In late April 2009, the CDC confirmed two cases of H1N1 in California. Two days later, the CDC announced that they had discovered a total of seven individuals diagnosed with H1N1. During these early stages of the outbreak in the United States, however, it was unclear if these individuals were connected to the outbreak that was taking place in Mexico. The same day that the CDC reported the first two cases of H1N1 in the United States, they also began development of a vaccine seed strain (CDC, n.d.k.). The US declared a public health emergency on April 26, 2009 in response to the growing number of cases. By this time, there were 20 confirmed cases of H1N1 across five states. The CDC also dispensed information about precautionary measures people could take to protect themselves from the disease. These recommendations included: 1) covering your nose and mouth when you sneeze or cough, 2) washing your hands, and 3) avoiding touching your eyes, nose, and mouth.

On April 27, 2009, the U.S. Secretary of Homeland Security, Janet Napolitano, gave a press briefing in which she said that she would be serving as the coordinator of America's federal response to the pandemic (Lister & Redhead, 2009). She argued that the National Response Framework, which served as a guide for federal response in various crisis scenarios, stated that the Secretary of Homeland Security would be in charge during national crises and that this H1N1 outbreak fell under those regulations. During this same time period, the Acting Health and Human Services Secretary, Charles Johnson, declared a public health emergency. This declaration provided the FDA legal authority to utilize emergency use authorization to use unapproved medical treatments and tests, or approved medical countermeasures against new indications", provided the benefits outweighed the risks during an emergency. Following this relaxation of restrictions, the FDA allowed the emergency use of oseltamivir and zanamivir for treatment of H1N1 (Lister & Redhead, 2009).

At this same time the United States issued travel warnings for all U.S. citizens traveling to Mexico. U.S. Customs and Border Protection began screening all people coming into the country. Any traveler that appeared to be symptomatic was taken to quarantine stations or a health official for further evaluation (Lister & Redhead, 2009). Cases in the United States continued to increase throughout the next few days, and the United States confirmed their first H1N1-related death on April 29, 2009, when a 23-month-old Mexican child died after its family had crossed the border into Texas. Not only was this the first H1N1-related death in the United States, but it was also the disease's first fatality outside of Mexico. President Barack Obama sent a letter to Congress in late April, requesting \$1.5 billion to help fight the outbreak (Quinn, 2009). He argued that this money would be helpful for general influenza preparedness measures such as vaccine development, increased monitoring and response capabilities, and stockpiling antivirals (Lister & Redhead, 2009). Many members of Congress had already begun looking into adding funds to the defense supplemental appropriation to support the H1N1 response (Lister & Redhead, 2009) and the President was able to receive the funds from Congress.

By June 1, 2009, although the rate of new cases was still dropping in the United States, there were now cases of H1N1 in all 50 states and 17 H1N1-related deaths. Influenza vaccine manufacturers supported by the Biomedical Advanced Development Authority in the United States accelerated R&D efforts to develop a vaccine for 2009 H1N1 throughout the summer and the first doses were made available to the American public on October 5, 2009. By this time, however, the influenza cases were dropping significantly and a supply/demand mismatch did not materialize in the United States once the vaccine became more widely available.

Response in Texas

Dr. Lakey, the Commissioner of the Texas Department of State Health Services at the time of the 2009 H1N1 learned about the initial case in Texas through informal channels - specifically a report published by the University of Minnesota's Center for Infectious Disease Research and Policy (CIDRAP) (Giles, 2011). Shortly thereafter, the state established a multi-agency coordinating center in Austin and directed the border health team to begin collecting data in South Texas. The Governor's Division of Emergency Management, the Texas Education Agency, and the Texas Department of Agriculture coordinated activities amongst themselves and with key partners throughout the state to make decisions about containment and response (Giles, 2011).

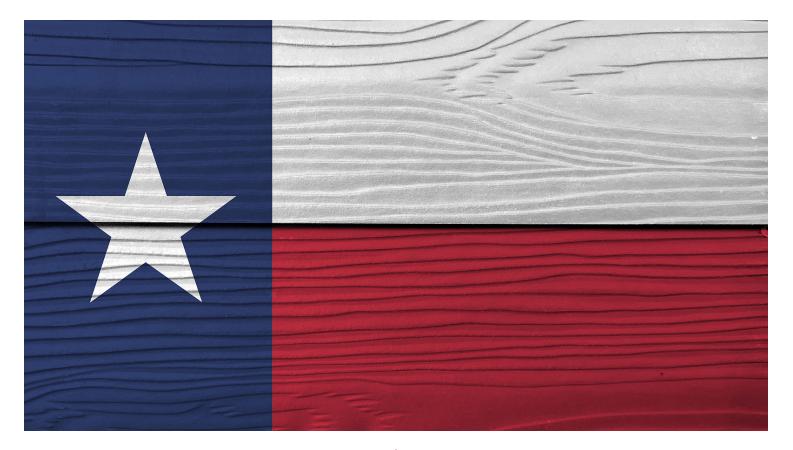
The Texas Education Agency followed the recommendations made by the CDC to consider closing schools with influenza exposure. By mid-May there were 858 closed schools, which affected more than half a million students in the state of Texas (Carlos, 2009). In Dallas County, where schools were nearly 100% compliant with reporting requirements, they reported 81 children with H1N1 at 35 different schools (Carlos, 2009).

The primary catalyst for closing schools in Texas during the 2009 H1N1 pandemic was the fact that influenza is known to spread easily in school settings. During the 2009 outbreak, the Texas Medical Association released a report that stated, "Schools remain the single most important communal setting for children aged 5 to 17. Because those in this age group are described as being the sentinel spreaders of seasonal influenza, targeting this population for control measures could potentially have the largest effect in reducing early disease transmission" (Carlos, 2009).

Surveillance of the 2009 virus in Texas evolved over the course of several months as more information was gained about the pandemic. Initial surveillance included all cases of influenza-like illness, but a month later that was narrowed to only confirmed cases. By June 2009, reporting was further narrowed to only include severe cases which required hospitalization or led to death (Texas DSHS, n.d.). Of the 11,625 specimens tested by the Texas Department of State Health Services Austin Laboratory, 3,086 tested positive for influenza. Of those 3,026 positive specimens, just over 92% tested positive for 2009 H1N1 (Texas DSHS, n.d.).

Since the 2009 H1N1 pandemic emerged in Mexico, Texas had to address issues of travel across the Texas-Mexico border. Initially, there were calls from some members of the U.S. Congress to close the border entirely, but that was dismissed at both the federal and state level (Dunham, 2009). Weighted in the decision of whether or not to close the border with Mexico was the economic outcome of such an action. In 2008, \$367 billion in trade had crossed the Texas-Mexico border and Texas alone had exported more than \$62 billion in goods to Mexico (Giles, 2011).

Some of the greatest challenges that the State of Texas encountered in their response to the 2009 H1N1 pandemic was the ability to distribute antivirals to all residents who needed them, regardless of insurance status. To solve this problem, the state turned to H.E.B., a Texas-based grocery store chain, to help develop an effective distribution strategy (Giles, 2011). In their partnership with H.E.B., the state made antivirals from the state stockpile available through 63 different H.E.B. stores (Giles, 2011). The partnership with H.E.B. in 2009 and the lessons that the company took from that



experience and others they have had in assisting with hurricane disaster response, helped them act quickly and serve as an example for other companies when the COVID-19 pandemic arrived in Texas (Solomon & Forbes, 2020). Entering the fall of 2009 the virus dissipated. Many experts believe that as it circulated, it became less virulent (Discovery Channel, 2018).

Role of Misinformation

The CDC acting director, Dr. Rich Besser, provided effective communications that are often lacking during infectious disease crises. Dr. Besser set the stage early in the 2009 pandemic for public expectations that were easy to understand. His message was clear and he repeated daily that there was much to learn about the 2009 H1N1. Every update included four major messages: 1) This is what we know today; 2) This is what we do not know, 3) this what we are doing to get a better understanding; and 4) we will keep you informed as soon as we do have a better understanding. Despite Dr. Besser's effective communication, conspiracy theories continued to spread among small groups of conspiracy theorists. At the beginning of the 2009 H1N1 pandemic, the disease was referred to as the "Mexican flu." In order to combat this image, Mexican officials spread and encouraged the spread of misinformation about the virus' origins. Some claimed that it had actually been imported from "Eurasia," while others said that it had come from China (Smallman, 2015). Within Mexico, and elsewhere in the world, citizens believed that the virus had been the result of a failed attempt by the US government to weaponize influenza and claimed that Secretary of Defense Donald Rumsfeld stood to benefit financially from the pandemic (Smallman, 2015).

On the US side of the border, theories spread equally rapidly. Videos began circulating of Mexican migrants with the claim that the migrants were disease vectors (Smallman, 2015). Another theory stated that the drug cartels had partnered with Al Qaeda to release the virus and that terrorists were then using Mexican migrants to walk the virus across the border in an act of "germ warfare" (Kahn, 2021; Smallman, 2015). Lastly, similar to theories during the SARS epidemic, many believed that the pandemic was a "scam" to make money for the medical and pharmaceutical industries (Evans, 2010).

THE 1968 INFLUENZA PANDEMIC

In July 1968 a large number of cases of influenza-like illness began appearing in government clinics throughout Hong Kong (Jester et al., 2020). The cause of the illnesses was quickly identified as H3N2 influenza virus and the World Health Organization issued a global warning in August (Jester et al., 2020). The first case of the 1968 flu in the United States occurred in a Marine returning from Vietnam, though shortly after military physicians all over the country began reporting cases.

The virus continued to spread within the United States military for many months and the first case was not reported in the civilian population until October 1968 (Jester et al., 2020). From this initial case in the civilian population, the virus spread quickly with early epidemic peaks occurring in Alaska, Nevada, Colorado, Wyoming, Washington, and Montana (Jester et al., 2020). Other states, such as Texas, Mississippi, and Kentucky, did not experience their peak outbreaks until January of 1969 (Jester et al., 2020). In all, approximately 100,000 people died from the 1968 flu in the United States and approximately 1 million died worldwide (CDC, 2019).

Since the end of the 1968 pandemic, the H3N2 virus has continued to circulate globally and its impact over the last 50 years has been significant. "The average estimated number of annual hospitalizations during the past six seasons for A(H3N2) virus" was 675,000, which is more than twice the number of annual hospitalizations that have resulted from the H1N1 virus (Jester et al., 2020). One of the reasons that the H3N2 virus continues to have such a large impact is that it undergoes antigenic drift (genetic mutations) at a higher rate than H1N1 viruses (Jester et al., 2020). Because of this, the virus continues to adapt in a way that allows it to evade host immunity.

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2014 Ebola

Ebola virus was first discovered in 1976 in what is today the Democratic Republic of Congo. The initial cases occurred along the Ebola River, which is what gave the disease its name. Since the initial discovery, scientists have identified six types of Ebola virus, four of which infect people. The most dangerous species is *Zaire ebolavirus*, which is the one that occurred in West Africa in 2014. This species has a case fatality rate ranging from 25-90% with the average around 50% (WHO, 2020e). The virus can be transmitted from animals-to-humans, but it can also be transmitted human-to-human through contact with bodily fluids.

Ebola is an acute illness, with only about a week between exposure to the virus and death. Infected individuals typically experience a sudden onset of symptoms which include fever, fatigue, muscle pain, headache, and sore throat. These symptoms quickly progress to vomiting, diarrhea, rash, and occasionally both internal and external bleeding. Many patients that die experience massive hemorrhaging. This has been attributed in popular literature, such as Richard Preston's *The Hot Zone*, as the "crash" of the patient.

The Ebola outbreak in West Africa was unexpected, as Ebola had been confined to East Africa up to that point. The first official case was an 18-month-old boy from a village in Guinea. It is still not known how the boy was infected, but the leading theory is that he was infected by bats while playing in a tree. From this initial case, several members of the boy's family were infected and the virus continued to spread. By March 2014, Ebola had reached the capital city of Guinea and the Ministry of Health issued an alert for an unidentified illness. A little over a week later, the Pasteur Institute, located in France, confirmed that the illness was Ebola virus.

Over the next several months the virus continued to spread. It quickly reached Liberia and Sierra Leone, where it spread widely. By July 2014, Ebola had taken hold in the capital cities of all three countries. WHO declared Ebola a Public Health Emergency of Concern in early August 2014. By the time the outbreak was contained, there were more than 28,000 reported cases and more than 11,000 deaths making it the largest Ebola outbreak in history. In fact, the 2014-2016 outbreak was 67 times larger than the largest previously recorded outbreak of Ebola (MSF, 2020). The size of the outbreak is partly due to its entrance into urban centers, something that had not yet occurred in previous Ebola outbreaks.

At the start of the outbreak, health care systems in Liberia, Sierra Leone, and Guinea were underequipped. Overall, case fatality rates varied between countries. Sierra Leone had a rate of 28 percent, Liberia had a rate of 45 percent, and Guinea experienced a case fatality rate of 67 percent.

Response from the United States

The US led the largest response to the 2014 Ebola epidemic of any country or organization. In total, the US provided over \$2.5 billion in support. Approximately \$1 billion of this support came from the United States Agency for International Development (USAID) and the Centers for Disease Control and Prevention (CDC) provided another \$980 million (USAID, 2016). Lastly, the Department of Defense (DOD) provided just over \$630 million (USAID, 2016). The total amount of funding provided by the US was several times greater than funding provided from all other countries combined.

The CDC arrived in July of 2014 and set up its Emergency Operations Center. The purpose of this operations center was to provide technical assistance to local and international partners, as well as support general logistics, staffing, communication, and disease management (CDC, 2019). During the peak of the outbreak the CDC trained more than 24,000 West African healthcare workers and assisted in expanding laboratory capacity in Guinea, Sierra Leone, and Liberia (CDC, 2019).

USAID activated one of its DART teams during the first week of August 2014 (USAID, 2014). The role of the



DART team was to identify the needs for controlling the outbreak, expand humanitarian response efforts, and coordinate all US efforts in the response to Ebola (USAID, 2014). USAID's Office of Disaster Assistance (OFDA) provided \$3 million to support CDC efforts, particularly in the areas of health education messaging, contact tracing, and data collection (USAID, 2014). Additional money was provided by OFDA to expand USAID outreach operations already in place and to provide personal protective equipment (PPE) to healthcare staff. Lastly, USAID worked with the World Food Programme, Mercy Corps, and Save the Children to deliver food aid, cash transfers, and food vouchers (USAID, 2016). This support was vitally important to the people of Sierra Leone, Guinea, and Liberia because Ebola had a negative impact on food security in each country.

Response in Sierra Leone

The Ebola outbreak in Sierra Leone circulated almost undetected for months before an explosion of cases at the end of May 2014. The first case was a woman who had visited Guinea in December 2013-January 2014. She became ill shortly after returning home to Sierra Leone in early January (WHO, 2016). This initial case did not get reported and the virus was able to gain a foothold in the country. By April, Sierra Leone's government began working to prevent importation of cases when individuals who had died in Guinea were brought back to Sierra Leone for burial. The surveillance that followed led to several suspected cases, but none of the suspected individuals tested positive (WHO, 2016). In early June there was a large cluster of new cases in Sierra Leone that could all be traced back to the funeral of a popular healer who lived near the border with Guinea. The healer had been treating Ebola patients from Guinea that had crossed the border seeking her guidance. All told, 365 cases were traced back to this one funeral and by mid-June the district of Kailahun had declared a state of emergency. This declaration led to the closing of schools, movie theaters, and gathering places. Additionally, it implemented checkpoints along the Guinea and Liberia borders.

In the city of Kenema, which was to the South of the original Sierra Leone epicenter, officials converted two full wards in the government-run hospital to Ebola treatment facilities. This allowed them to keep Ebola patients separate from the rest of the hospital population, but without proper PPE a number of health care officials became infected. Eventually there were more than 40 deaths of doctors and nurses in this single hospital. At the end of June, MSF arrived in Sierra Leone and opened an Ebola treatment center in Kailahun, the original location of the outbreak. MSF has since acknowledged that they took this action too late and that the virus had taken hold in the city.

Around this time, the Ministry of Health established an Ebola Operations Center, in which WHO served as a primary collaborator (Ross et al., 2017). The Ebola Operations Center was chaired by the Minister of Health and WHO, but also received input from the United Nations, IFRC, IRC and others. By the end of July, President Koroma declared a state of emergency and established the Presidential Task Force on Ebola. On July 29th, Sierra Leone's only expert on viral hemorrhagic fevers died of Ebola.

Approximately one week later, on August 8, 2014, the WHO declared Ebola a Public Health Emergency of Concern. They encouraged the international community to make resources available for all three of the affected countries and deployed teams to determine how healthcare workers were becoming infected. Despite the declaration, the outbreak continued to spread throughout the month of August. By September MSF began calling for greater support from the UK government. They request that military support is



provided in addition to civilian support (Ross et al., 2017). In response to this request, the UK sent 750 military personnel to Sierra Leone and Canada sent an additional 36 personnel to join the British military (Kamradt-Scott et al., 2015). Additionally, Sierra Leone makes the decision to lockdown the country for 3 days in September and to simultaneously do a house-to-house education campaign (Gostin & Friedman, 2015).

Despite these efforts, a new epicenter emerged in September in the city of Freetown. While cases in other cities around Sierra Leone had started to come under control, cases in Freetown and its surrounding districts spiked dramatically. On September 21, 2014 Operation GRITROCK was deployed as part of the UK civilianmilitary task force (Ross et al., 2017). This operation eventually went on to lead the entire international response and assist with the roll out of District Ebola Response Centers throughout the country. The number of weekly new cases increased to more than 500 by the end of October 2014 (Ross et al., 2017).

As the outbreak reached its peak in Freetown and the surrounding districts, supplies began to run out. There were no hospital beds, no PPE, no food, and no rehydration fluids (WHO, 2020e). In response to this crisis, the World Food Programme, with support from the World Bank, airlifted 20 ambulances and 10 mortuary trucks into Freetown (WHO, 2020e). This was in addition to food support for more than 300,000 people in the country who had been affected by Ebola.

Efforts, led by the UK in collaboration with WHO, MSF, UN and the government of Sierra Leone, continued throughout the next several months. At the end of November 2014 cases peaked in the country and continued a slow decline until March 2015, at which point the UK scaled back involvement in the response. The turning point in outbreak control was the development of new burial practices that eliminated the need to touch the body of the deceased, but still fulfilled the spiritual needs of the family. By March 2016, the outbreak was over and there had been a total of 14,122 confirmed, probable, and suspected cases in Sierra Leone and 3,955 deaths (WHO, 2016).

Response in Texas

On September 25, 2014 - approximately 6 months into the Ebola outbreak in West Africa - a man who had



recently traveled from Liberia presented in an emergency room in Dallas County (Chevalier et al., 2014). The patient had a fever of over 100 degrees as well as abdominal pain and a headache, but was discharged after being treated for a possible sinus infection (Chevalier et al., 2014). Three days later he returned to the hospital with more severe symptoms, including diarrhea, and tested positive for Ebola. Following the positive test, contact tracing was immediately implemented. Through this contact tracing, 48 individuals, including 21 healthcare workers were identified. All identified individuals were monitored by public health authorities.

On October 11, 2014 a nurse that had helped provide intensive hospital care for the first patient began to show symptoms of Ebola. Shortly thereafter, another nurse fell ill. In response the Ebola team in Dallas began working with hospitals to help them provide proper care to the Ebola patients. Additionally, the team established a medical transport plan for known or suspected Ebola patients, increased capacity for Ebola testing using molecular methods (PCR), and trained more than 100 healthcare workers on the proper use of PPE (Chevalier et al., 2014). The effectiveness of contact tracing was greatly aided by local and charitable organizations who were able to provide financial assistance for those asked to stay home, as well as food, clothing, textbooks, and other necessary school supplies (Smith et al., 2015).

In October, following the cluster of cases in Dallas, Governor Rick Perry created the Texas Task Force on Infectious Disease Preparedness and Response with Executive Order RP-79 (Legislative Reference Library of Texas, n.d.). The task force was originally composed of 17 members, which included representatives from state agencies, and experts in infectious disease and pandemic response. The Texas Task Force on Infectious Disease Preparedness and Response continues to provide guidance for public health emergencies in the state.

Ebola in Texas did not spread beyond the few isolated contacts of the initial patient. In all, the United States had 11 people who were treated for Ebola. Seven of these cases were individuals who were infected and fell ill while in West Africa treating Ebola patients. They were then transported from West Africa to hospitals in the United States. Of these seven, six recovered and one died. For the remaining four cases, two were tied to the patient in Dallas and one was a medical worker who fell ill after returning to New York City. Of these four cases, three recovered and one -- the initial patient that reported to the hospital in Dallas -- died.

Role of Misinformation

During the 2014 Ebola outbreak in West Africa, distrust and misinformation spread like wildfire. Some residents of Liberia believed that Ebola was a bioweapon that had been designed and developed by the United States with the goal of global depopulation (Feuer, 2014). Many residents of Guinea avoided hospitals because they believed that doctors were injecting patients with poison or were trying to lure people into their clinics so that they would harvest their blood and organs (Al Jazeera, 2014). On an international scale, there was the belief that the virus had been engineered by a group called the New World Order and their goal was to implement martial law (Feur, 2014). All of this misinformation led some people to avoid healthcare or not take the virus as seriously as they should.

In the United States, poor and ineffective communication allowed fear among the American public to grow irrationally large. While Ebola is a scary and deadly disease, the risk to the general public in the United States remained low. The lack of effective communication meant that Americans did not gain an understanding of the realistic nature of the threat and, thus, tended to view it as more extreme than it was (Andrew et al., 2018).





CONCLUSION

Past pandemics and epidemics offer an opportunity to observe and learn lessons that will help better prepare us for the next pandemic. These lessons may include a better understanding of viruses, the broad economic impacts of disease, or how misinformation can impact effective pandemic and epidemic response. While the COVID-19 pandemic was not covered in this white paper, lessons learned from the outbreaks discussed here helped to inform COVID-19 response and the responses to all outbreaks can inform our responses to future pandemics.

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In Memoriam

Lieutenant General Brent Scowcroft

(March 19, 1925 - August 6, 2020)



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