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Learning from the 2009 H1N1 Influenza Pandemic

RMS Special Report

RMS®

EXECUTIVE SUMMARY

- The 2009 H1N1 influenza pandemic illustrated how fast a new infectious disease can spread through modern society. The speed at which the H1N1 virus spread to infect many millions of people was unprecedented for a disease of this scale. The 2009 H1N1 influenza virus became well-established in large numbers of countries within a few weeks.
- Though the novel virus was relatively mild when compared to previous pandemics, it reached nearly every country in the world and infected many millions of people.
- The pandemic H1N1 virus exhibited several unusual characteristics. Most flu viruses have strong seasonality and a preference for temperate regions, but the pandemic H1N1 virus emerged in the spring in Mexico and transmission continued throughout the summer in the northern hemisphere. Most mild flu viruses have a more significant impact on the elderly, but the pandemic H1N1 virus had a higher-than-average death toll in young and otherwise healthy people.
- The presence of the 2009 H1N1 pandemic does not decrease the probability of another pandemic occurring in the near future. Viral reassortment can occur at any time, and flu viruses will constantly continue to evolve.
- Pandemics are inevitable and their economic impacts can be significant, with significant insured losses. Life and health insurance companies and other businesses must assess the likely impacts of mortality and morbidity from infectious diseases, in order to determine the risk capital needed for extreme scenarios.
- Analytical and statistical models are critical in understanding and managing infectious disease risk. Using historical data, as well as established principles of epidemiology, virology, mathematical analysis and modeling, the range of possible pandemic events and their impacts can be quantified. Models provide a quantitative framework to evaluate risk and make informed decisions.
- There are significant lessons from the 2009 H1N1 influenza pandemic—from how this pandemic spread to how people attempted to deal with it—that can be applied in the event of a future, more severe disease outbreak.

INTRODUCTION

In the spring of 2009, a new type of influenza virus, never before linked to human disease, emerged and rapidly spread throughout the world, causing the first influenza pandemic of the 21st century. The novel strain of influenza virus A (H1N1) causing the pandemic was identified in April 2009, after outbreaks of a deadly "swine flu"[1] were reported in Mexico and the United States. The World Health Organization (WHO) first declared the pandemic in June 2009, when the virus had spread to most regions of the world.

During the early stages of the pandemic, before the characteristics of the virus became more clearly established, many feared the world was facing an unstoppable disease of unknown severity. Health agencies and governments around the world strove to limit the impact of the virus, sharing medical resources accordingly.

Though the new virus proved to be relatively mild when compared to the previous three pandemics of the past century (in 1918, 1957, and 1968), it reached more than 200 countries and territories around the world and infected hundreds of millions of people. The pandemic H1N1 virus also exhibited several unusual characteristics. Most flu viruses have strong seasonality and a preference for temperate regions, but the pandemic H1N1 virus emerged in the spring in Mexico and transmission continued throughout the summer in the northern hemisphere. Most mild flu viruses have a more significant impact on the elderly, but the pandemic H1N1 virus had a higher-than-average death toll in young and otherwise healthy people.

Risk Management Solutions (RMS) monitored the pandemic from its initial outbreak in March 2009 through its development, providing regular assessments and modeled outlooks of the potential future impacts. The RMS® Pandemic Influenza Model, developed to estimate the spread and overall impact of pandemic influenza and other infectious diseases, was used both as a framework to understand the unfolding events and as a guide to the likely progression of the pandemic. In the forty-one years since the last influenza pandemic, the world has changed significantly. Medical science has advanced, with better pharmaceutical resources, improved disease surveillance, and increased public health preparedness. However, regardless of where diseases emerge, they spread more quickly today due to major globalization, greatly increased passenger travel between countries, and larger, denser, and more interconnected cities. The RMS model considers evidence from historical outbreaks as well as modern data to estimate the global impacts of an outbreak.

There are significant lessons from the 2009 H1N1 influenza pandemic—from how this pandemic spread to how people attempted to deal with it—that can be applied in the event of a future, more severe disease outbreak. Life insurance companies and other businesses must assess the likely impacts of mortality and morbidity from infectious diseases, in order to determine the risk capital needed for extreme scenarios. This report presents a synopsis of the 2009 H1N1 pandemic event, discussing its impacts, and highlights the role of analytical and statistical models in understanding and managing infectious disease risk.

[1] H1N1 influenza was referred to as "swine flu" because of the genetic similarity of the H1N1 virus to influenza viruses normally found in pigs.

THE 2009 H1N1 INFLUENZA PANDEMIC

Background

Influenza is a contagious disease caused by an exceptionally adaptable RNA virus. Influenza viruses are classified by type (A, B, or C). Type A, the most virulent among the three types to humans, has been responsible for the four influenza pandemics since 1900: the 1918 ”Spanish flu,” 1957 “Asian flu,” 1968 “Hong Kong flu,” and the current 2009 H1N1 “swine flu.” Each of these pandemics was the result of major genetic changes that created novel strains of influenza, with enhanced transmissibility and/or resistance to standard treatments. There are several subtypes of influenza A, named according to their surface proteins (e.g., H1N1, H5N1, or H3N2). The “H” stands for hemagglutinin and the “N” for neuraminidase.

Antigenic variation is the evolutionary mechanism by which viruses evade host immune systems. Influenza viruses accomplish this through minor, incremental genetic changes (i.e., “antigenic drift”), as well as through major genetic alterations (i.e., “antigenic shift”). Antigenic drift specifically refers to frequent, discrete mutations that occur within the genes (e.g., the hemagglutinin or neuraminidase genes) of a given influenza subtype, leading to new strains that escape host immune surveillance. These new strains drive the need to produce annual seasonal flu vaccines. Antigenic shift, on the other hand, refers to the exchange of whole genetic segments between different flu viruses, sometimes resulting in the introduction of novel, transmissible viral subtypes to the immunologically naïve (i.e., lacking protective antibodies) human population. Antigenic shift events are the cause of global pandemics. Epidemiological models can simulate general patterns of sickness and mortality caused by both types of antigenic variations.

Antigenic drift works by natural selection. Over time, the virus evolves into a new strain with novel characteristics, such as drug resistance, increased transmissibility, or increased virulence. This process typically involves trade-offs such that the overall characteristics of the resulting virus are rarely significantly different from existing strains. Evolutionary drift occurs at a rapid rate; as a result, humans are unlikely to cure or develop immunity to all influenza viruses and influenza vaccines will only be effective against a limited number of strains over a short period of time.

Antigenic shift occurs when distinct flu viruses co-infect the same host and mix their genetic material. This process of reassortment produces novel virus subtypes—some of which may be particularly virulent and easily transmissible between humans. Reassortment may occur in animal intermediates or reservoirs, such as birds or pigs, which transmit the reassorted viruses to humans in close contact with the animals, such as farmers or butchers. Reassortment may also occur in humans, when a bird or swine flu virus directly “jumps” to a person already infected with a human influenza virus. Unlike advantageous single mutations that are selected over time, reassortment is an instantaneous process that facilitates dramatic changes in influenza properties, with potentially devastating consequences to naïve human populations.

Both the process of viral replication within an animal host and animal-to-animal transmission can lead to the mutation and evolution of an influenza virus. To trace the origins of the H1N1 strain, scientists computed the number of mutations in the 2009 pandemic H1N1 virus and compared it to the number of mutations in older influenza viruses. Influenza viruses are composed of eight genes. Of the eight H1N1 virus genes, laboratory techniques confirmed the association of six virus genes to North American pig influenza viruses, and two genes to influenza viruses common to Eurasian pigs. The comparison reveals that the 2009 pandemic H1N1 strain most likely had evolved in nature—possibly in an unknown animal host—for a number of years before being detected in humans.

Development and Synopsis of the 2009 Pandemic

In mid-March 2009, reports of an outbreak of a highly transmissible, influenza-like illness began circulating throughout Mexico, originating from the state of Veracruz. By mid-April, "swine flu" outbreaks led to the hospitalization of over 1,600 people and the deaths of over 100. On April 21, 2009, laboratory studies confirmed that two influenza cases in U.S. children were caused by a novel influenza virus. Additional cases of the illness began appearing in the U.S., and individuals were soon confirmed to be infected by the same viral strain as the patients in Mexico. The new virus was infecting large numbers of people and was highly transmissible between humans. It was also evidently virulent, with deaths each day being reported in Mexican hospitals. By the end of April 2009, the chance the virus could spread to the worldwide community was high, but not inevitable. It was still possible that the epidemic in Mexico could be contained, if the number of people who were infected was still small; and if the infectiousness of the virus (i.e., the rate at which it spread from one person to another) was not too high. Given the limited amount of information available at the time, both the total number of infected people and the infectiousness of the virus were unknown and difficult to estimate.

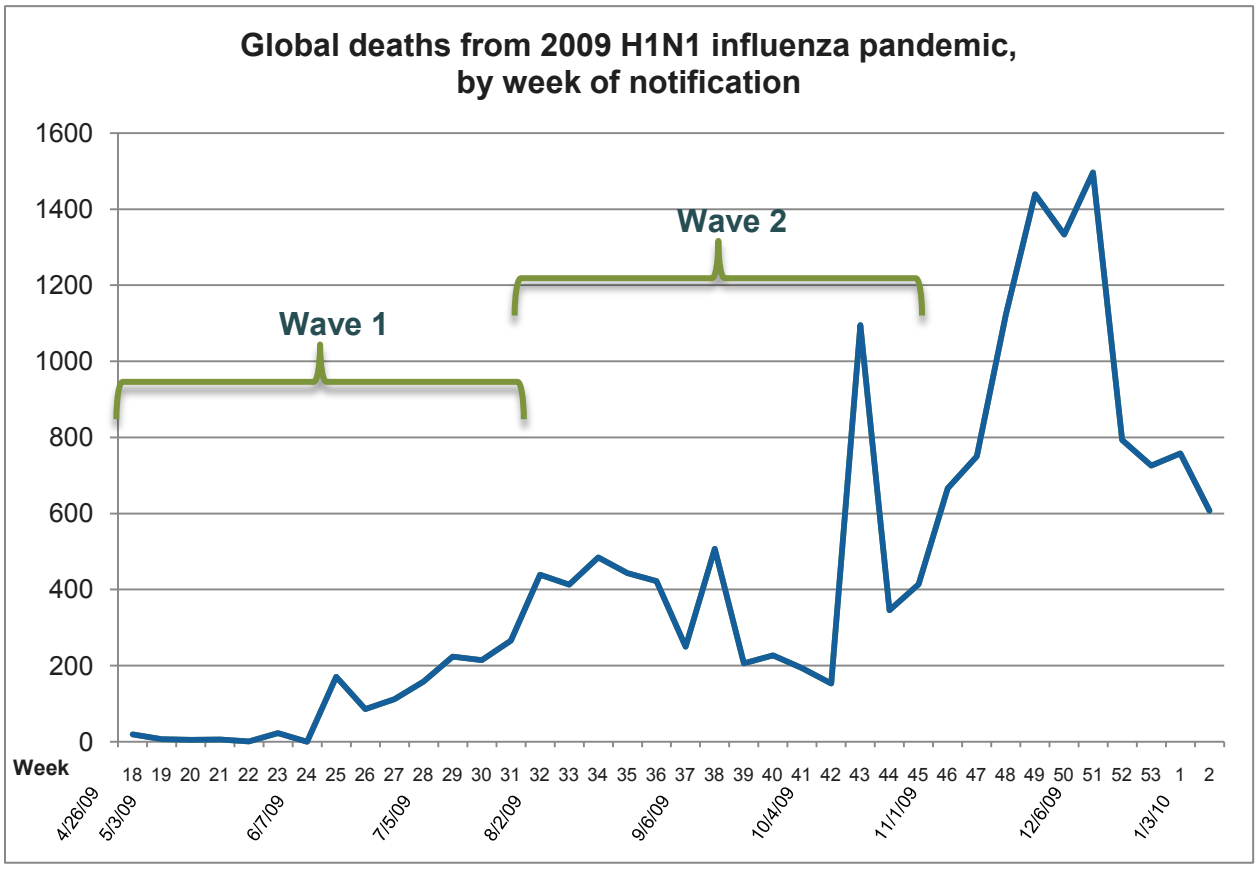

Figure 1: Global deaths from the 2009 H1N1 influenza pandemic, by week (Data Source: ECDC, 2010)

As of April 2010, the pandemic H1N1 virus continues to be the predominant circulating influenza virus worldwide, with laboratory-confirmed cases reported in more than 200 countries, as illustrated in Figure 1.

World Health Organization (WHO) Pandemic Alerts

The WHO alerted the global community when there was laboratory confirmation of the new virus, but did not raise its pandemic alert status. At the time, the WHO pandemic alert level was Phase 3, due to sporadic human cases and limited human-to-human transmission of avian influenza H5N1. RMS issued its first Pandemic Client Advisory on April 26, 2009, warning clients of a "strong possibility that this will be a pandemic."

The novel H1N1 virus then quickly spread locally and across North America and Europe, demonstrating efficient human-to-human transmission. The WHO raised the pandemic alert level from Phase 3 to Phase 4 ("Evidence of Increased Human-to-Human Transmission") on April 27 and to Phase 5 ("Evidence of Significant Human-to-Human Transmission") on April 29. It finally declared a Phase 6 ("Pandemic") on June 11, 2009. Table 1 shows the timeline for the WHO alert announcements.

The WHO was naturally cautious and based its alert decisions on carefully-weighed evidence. It was very aware of the potential for public panic, and the negative global economic impact of declaring a pandemic. The official pandemic preparedness measures for most countries are determined by the WHO alert status levels, and a declaration of a pandemic often means the large-scale and costly mobilization of public health resources within a country. Therefore, while the WHO was careful in its declaration of a pandemic, for businesses implementing protection measures, or for national public health agencies being proactive in mitigating the impacts, the WHO pandemic status notices were lagging behind the evidence, rather than acting as preparedness warnings.

Table 1: World Health Organization pandemic influenza phases and timeline of the emergence of the 2009 H1N1 influenza pandemic

WHO Pandemic Influenza Phase	H1N1 Timeline of Events
PHASES 1-3 **Mostly animal infections**	18 March 2009- Mexico reports increase in ILI
	23 April 2009- Mexico reports first H1N1 case to WHO
	23 April 2009- 7 cases in US (Texas & CA)
	24 April 2009- Mexico speaks of "epidemic"
	25 April 2009- WHO declares Public Health Emergency of International Concern (PHEIC)
	26 April 2009- 20 confirmed H1N1 cases prompts "health emergency" in US
PHASE 4 **Human-human transmission**	**27 April 2009- WHO raises level from 3 to 4**
	27 April 2009- First confirmed cases in Europe, Spain, Scotland
	28 April 2009- All 5 continents affected
PHASES 5-6 **Pandemic** **Widespread human infection**	**29 April 2009- WHO raises level from 4 to 5**
	29 April 2009- First confirmed death in US (23 m/o toddler)
	29 April 2009- Cases confirmed in Germany and Austria
	1 May 2009- First confirmed case in Asia (Hong Kong)
	3 May 2009- 898 total cases reported in 18 countries
	17 May 2009- 8480 total cases, 72 deaths reported in 39 countries
Current	**11 June 2009- WHO raises level from 5 to 6 and "The world is now at the start of the 2009 influenza pandemic"**
	9 April 2010- Over 17,000 total deaths reported in over 213 countries and territories
POST PEAK **Possibility of recurrence**	
POST PANDEMIC **Seasonal**	

When the WHO raised the pandemic alert level to Phase 6, novel H1N1 cases had been reported globally and efficient and sustained human-to-human transmission had been occurring for months. Evidence that the virus could cause high mortality was nominal, as the pattern of morbidity and mortality that emerged was akin to a seasonal flu strain of mild to moderate virulence. As caseloads continued to amass in countries around the world, in July 2009, the strain on surveillance and laboratory diagnostic facilities caused the WHO and the U.S. Centers for Disease Control and Prevention (CDC) to discontinue both the testing of individuals with mild symptoms and the regular reporting of specific case counts. As a result, official counts produced by health agencies during the pandemic are likely significantly less than the true number affected.

Epidemiological studies have demonstrated that actions taken to combat the spread of an epidemic are much more effective if they are implemented earlier in the disease cycle. The WHO is now re-evaluating its system of pandemic alert notification. One important but absent dimension of the warning system was a metric for the severity of the pandemic.

The WHO scale identifies the level of evidence that a pandemic is in process, rather than how severe it is likely to be. Other scales, such as the Pandemic Severity Index (PSI) proposed by the CDC in 2006, which predicts severity and communicates response measures appropriate to the severity of the pandemic, were never officially issued. A future pandemic would benefit from an early assessment of likely severity and alerts issued earlier in the process.

Case counts began to diminish in the northern hemisphere during the summer months, but the WHO and other health organizations were concerned about a resurgence of the virus in the fall, fueled in part by continued transmission in the southern hemisphere. The typical flu season in the northern hemisphere runs from October through May. As illustrated in Figure 1, the second wave of illness in the U.S. began in August 2009, and incidence peaked in late October. The number of deaths from October to December 2009 also shows a steady increase, with a spike in week 43 (October 18 to October 24), which was attributed to delayed and aggregated reporting from official sources. The early onset of the pandemic H1N1 resurgence in August meant that several months of transmission would occur prior to the widespread availability of a vaccine. This, coupled with reports of severe and fatal illness in children and pregnant women, created panic around the need to be vaccinated.

Two major concerns emerged early in the monitoring of the 2009 H1N1 pandemic. First, the age demographic of victims was different from the pattern typically seen in seasonal flu. The new virus appeared to be causing high mortality in relatively young people in Mexico and the U.S. Second, the virus was successfully spreading during a time of year normally characterized by limited transmission in the northern hemisphere. The proximity of the origin of outbreak to the U.S. also increased the potential impact on populations with high life and health insurance penetration.

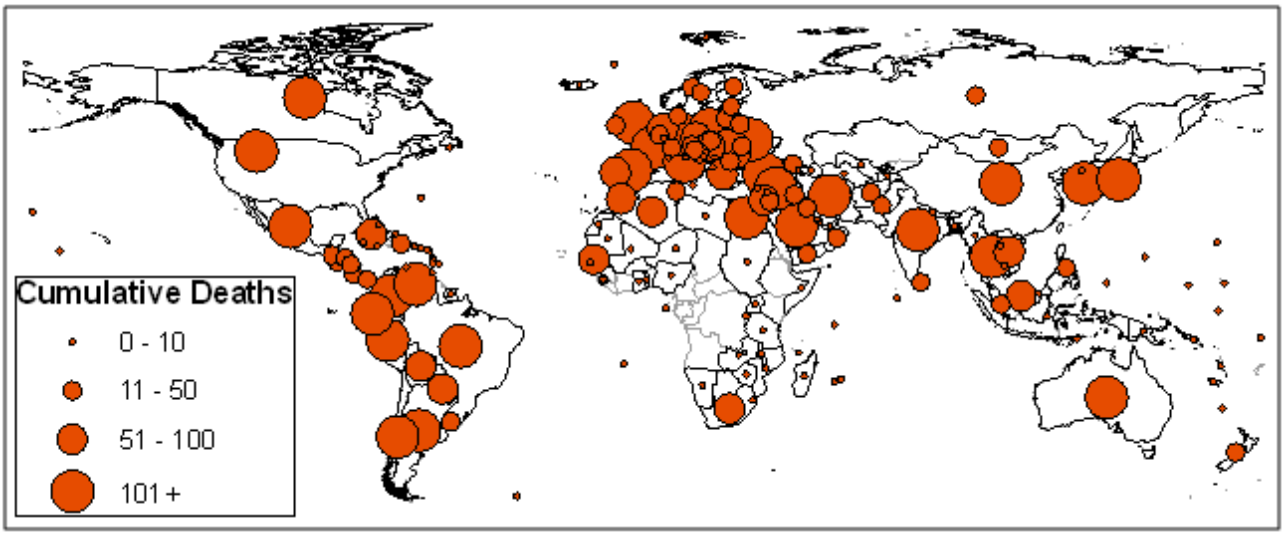

Figure 2: Map of cumulative global deaths from the 2009 H1N1 influenza pandemic, as of February 2010 (Data source: ECDC, 2010)

Estimation of Severity

Early estimation of the future severity of a pandemic relies on assessments of the infectiousness and virulence of the virus. An assessment of the caseloads (i.e., how many people were infected and how rapidly the disease was spreading) and the case fatality rate (i.e., the number of infected people who die) remained highly uncertain over a long period of time. If public health authorities had a good idea of the infection rates at the early stage of the epidemic, it would have been possible to contain the outbreak from spreading across the world. However, in Mexico, by the time officials were able to estimate the caseload, the outbreak had already spread and its containment became impossible.

The primary difficulty throughout the early development of the pandemic was to distinguish novel H1N1 cases from other types of influenza, colds, and respiratory infections. However, H1N1 cases could only be definitively identified through laboratory testing. With media coverage of a new pandemic virus, many people naturally sought medical advice for flu-like symptoms. Laboratories were quickly inundated with tests for the H1N1 virus. Statistics on caseloads were constrained by laboratory capacity and speed of processing. By mid-June, official statistics on confirmed caseloads were abandoned as misleading, as laboratory testing was limited to a small proportion of actual cases. In some countries, the statistical progression of the pandemic was assessed based on the volume of people seeking consultation with a physician.

Death statistics were less ambiguous than caseload assessments, due to more thorough laboratory testing in fatal cases. However, without an accurate assessment of the total number of people infected with the virus, the case fatality rate—the critical assessment of virulence—was difficult to estimate. During the early days of the pandemic, RMS took a probabilistic approach to assessing the case fatality rate, assuming a level of uncertainty and a binomial distribution for the conditional likelihood of case fatality rate, based on the observational data.

Case Fatality Rate

Observationally, the case fatality rate diminished significantly and rapidly over the first few months of the pandemic. The initial cluster of hospitalized cases in Mexico in March and April of 2009 led to at least 100 confirmed deaths. Although the number of infected people in Mexico during these same months is highly uncertain, it is likely that the caseload was in the thousands, suggesting a case fatality rate on the order of a percentage point. Once the disease spread to the U.S. and Europe, the case fatality rate was far lower. During May 2009, when laboratory testing was relatively comprehensive, there were more than 1,000 people testing positive for novel H1N1 for every confirmed death, suggesting a case fatality rate an order of magnitude lower than that seen in Mexico two months earlier. By April 2010, after the disease had spread through the general population and had largely run its course, the total deaths relative to the estimated total number of people infected with the virus was more on the order of three in ten thousand, which represents another order of magnitude lower than seen in Mexico at the start of the outbreak.

This trend in case fatality rate is not unprecedented, as case fatality rates have diminished over time in previous pandemics. However, this reduction was much more rapid than previously observed. Early projections from public health authorities overestimated the mortality impact of the pandemic, largely because the magnitude of the case fatality reduction, as the virus spread through the population, was not taken into consideration.

The reduction in observed case fatality rate is due to a number of factors. The initial infection wave in Mexico was unexpected, and occurred both in a population with low seasonal flu immunity and in a region of poor public health infrastructure. Delays in seeking treatment, availability of antiviral drugs, and other circulating respiratory diseases contributed to the high death toll. As the disease spread to the U.S. and Europe, the public health measures in place in these areas allowed for shorter time to treatment and plentiful supplies of antiviral drugs. In addition, these populations had generally higher levels of immunity. The world population ultimately affected by the 2009 pandemic influenza virus may have benefited from more preparedness time, the sharing of stockpiles of antiviral drugs and the availability of the vaccine, particularly for high-risk individuals.

Models of pandemic influenza should incorporate estimates of the changes in case fatality during the life cycle of the disease. It is possible that the virus lost some of its virulence as it reproduced during the pandemic's progression. It is also possible that changes in the virus could have resulted in increases in virulence. For example, Tamiflu-resistant strains of the virus were identified by July 2009, which could have increased the lethality of the disease had these strains become more common. Other mutations of the virus could have similarly changed the overall case fatality rate seen in the 2009 pandemic.

Attempts at Containment

The speed and effectiveness of response to contain pandemic influenza is a key variable in mitigating the overall impact. Early national, state, and local mitigation measures included school closures, travel restrictions to and from Mexico, and surgical mask and respirator stockpiling. In addition, personal response measures included frequent hand washing, crowd avoidance, and self-quarantine when contagious, through the end of the incubation period (typically 1–7 days in duration).

More formal measures to contain the spread of the new virus were implemented in the spring months of 2009; however, these measures were ultimately ineffective. Mexico instigated a national period of quarantine starting on May 1, 2009, closing restaurants and schools, as well as banning public gatherings. This quarantine reduced the spread of the virus, and after several days with no new reported cases and severe economic losses, the ban was lifted on May 5. Unfortunately, the following week, record numbers of new cases were reported.

In April of 2009, cases reported in the U.S. were mainly traced to individuals who had traveled from Mexico. Contact tracing (i.e., isolating the carrier individuals and identifying all of their contacts) and putting carriers and their contacts on a course of antiviral drugs was implemented in many places. European and Canadian health organizations similarly focused on containment around the identified infected individuals.

By the end of May, as the number of cases looked set to double every few days, authorities abandoned attempts at containment and switched to a new strategy of pandemic mitigation.

International Spread Patterns

The international spread of the 2009 H1N1 virus was more rapid than that observed in previous pandemics. The last influenza pandemic, in 1968, took almost a year to proliferate around the globe from its initial outbreak in China to the last infected countries in South America. Further back in time, the 1918 pandemic spread more slowly, by ship. In contrast, the 2009 pandemic spread quickly via international passenger air traffic, infecting 74 different countries on every continent within five weeks of the Mexico outbreak.

The volume of air passenger traffic dictated the pattern of the spread of the virus around the world. During the initial outbreak period, over 300,000 people flew internationally from Mexico each week. Infection rates in Mexico likely reached 1 in 10,000 during early April, so that hundreds of infected individuals had already traveled abroad before the virus was even identified, making containment impossible.

Of the 1.3 million people who flew out of Mexico during April 2009, 68% of individuals flew to the U.S, 16% flew to Canada, 8% flew to Europe and 7% flew to Latin America. In the first two weeks of May, 3,000 new cases were identified outside of Mexico, with 74% of them in U.S., 11% in Canada, and 7% in Europe, almost exactly mirroring the air passenger traffic. The exception was Latin America, which reported less than 1% of the new laboratory-confirmed cases—possibly due to less rigorous disease surveillance in these countries rather than a slower spread of the virus.

Infectious disease is now a more rapidly spreading international threat than ever before, driven by international air passenger traffic. The modeling of the potential spread of infectious disease using air traffic volumes was well-validated by the observations seen in the 2009 pandemic.

Declining Case Rates in Spring 2010

As of spring 2010, surveillance data indicates that disease levels in most countries are dropping. However, the pandemic H1N1 virus continues to cause illness and death in several parts of the world, with evidence of new community transmission in some countries. The WHO is holding at the current highest phase of pandemic alert (Phase 6), determining that it is still too soon to declare that all parts of the world have experienced peak transmission. The possibility of resurgence in caseloads remains, and future waves of transmission are feared as the southern hemisphere enters its winter flu season, which typically lasts from May through October. While a move to the post-peak phase does

not signify an end to the pandemic, it could potentially impact surveillance and control actions in some countries. The WHO continues to monitor the situation and has communicated that it will reconvene its panel periodically to review the epidemiological evidence and revisit its recommendations.

As of April 2010, virtually all countries around the world have reported laboratory-confirmed cases, and the pandemic H1N1 virus continues to predominate among all subtyped circulating influenza A viruses. The 2009 H1N1 virus is likely to continue circulating for many years. Moreover, as there are indications that the pandemic virus will continue to displace other influenza strains, the WHO has recommended the inclusion of the pandemic strain as one component of the multivalent seasonal flu vaccine for the northern hemisphere's 2010 influenza season.

UNIQUE CHARACTERISTICS OF THE 2009 H1N1 PANDEMIC

In 2004, the discovery of human cases of influenza A/H5N1 ("avian flu") redefined the virulence scale for influenza and caused the global community to re-evaluate the threat posed by influenza viruses. The subsequent emergence of the 2009 H1N1 virus has again caused the re-evaluation of what is known about these viruses, including the impacts on the younger population, the use of antiviral drugs to reduce the severity and duration of flu symptoms, and the availability of a vaccine against the H1N1 virus.

Impact on the Younger Population

Greater risk for influenza complications has been noted for seasonal influenza in pregnant women, the elderly, the very young, and those with chronic underlying medical conditions (such as chronic lung disease, cardiac disease, immunodeficiency, and diabetes). During this pandemic, the same groups of individuals were considered to have a higher risk for complications from the 2009 H1N1 virus.

In typical flu seasons with multiple circulating viruses, the elderly suffer a disproportionate number of flu-related fatalities, as compared to the young and healthy. As illustrated in Figure 3 (in green), seasonal flu typically exhibits a “U” shaped mortality curve, with the very young and the elderly being affected disproportionately relative to the total population. The 1918 pandemic exhibited a “W” shaped curve due to an additional peak in mortality around age 30 (Figure 3 in red), while in the 2009 H1N1 pandemic (Figure 3 in blue), the elderly seemed to have conferred immunity, resulting in lower infection risk (Taubenberger, 2006). The 2009 pandemic saw a disproportionate amount of cases in the younger demographic.

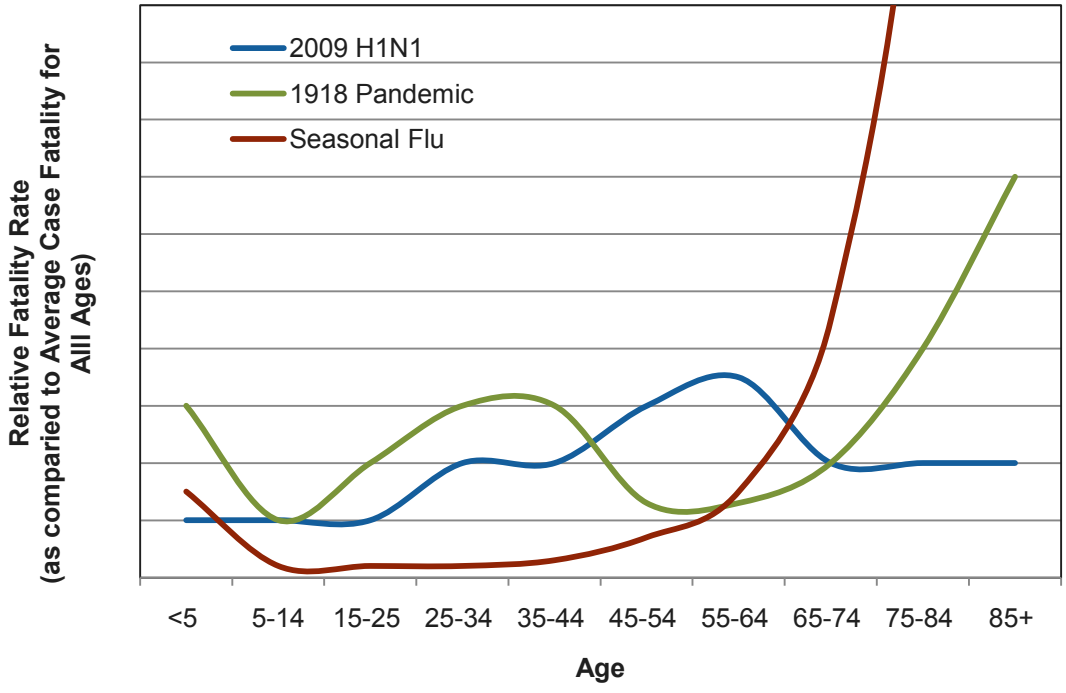

Figure 3: Age distribution of influenza mortality: comparing seasonal flu to the 1918 and 2009 pandemics

A young age demographic of flu victims is a key indicator of a cytokine storm, where the immune system becomes over-stimulated and unregulated in response to a novel strain of flu; as a result, the symptoms are most severe in young adults with strong immune systems. Other contributing factors to a mortality spike in younger populations include a highly virulent virus, overwhelmed health systems, limited supplies of antiviral drugs, or a slower time to widespread vaccine availability.

This phenomenon of greater disease burden on younger people has been observed in the 2009 pandemic, as indicated in Figures 4 and 5, which show the age breakdown of early novel H1N1 cases and deaths in the U.S. Moreover, researchers studying the 2009 H1N1 virus have found evidence to indicate that older people had pre-existing or residual immunity, resulting from contact with a previous virus that was genetically similar to the 2009 H1N1 virus (Xing, 2009).

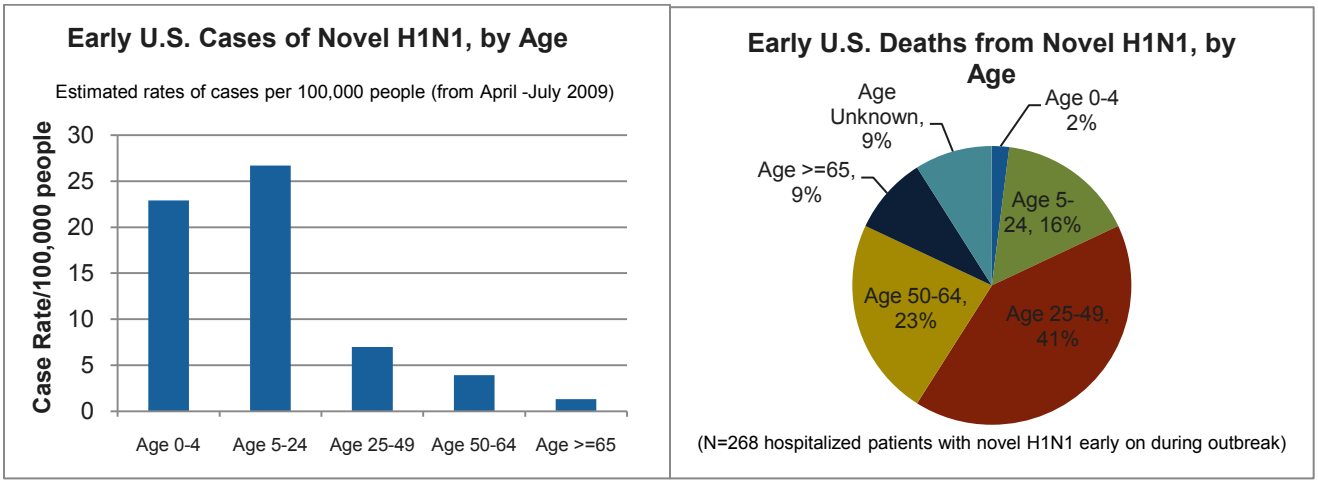

Figure 4: Early outbreak characteristics of the 2009 H1N1 virus in the U.S.: number of cases per 100,000 people from April to July 2009 (left) and early fatalities by age (right) (Date Source: CDC, 2009).

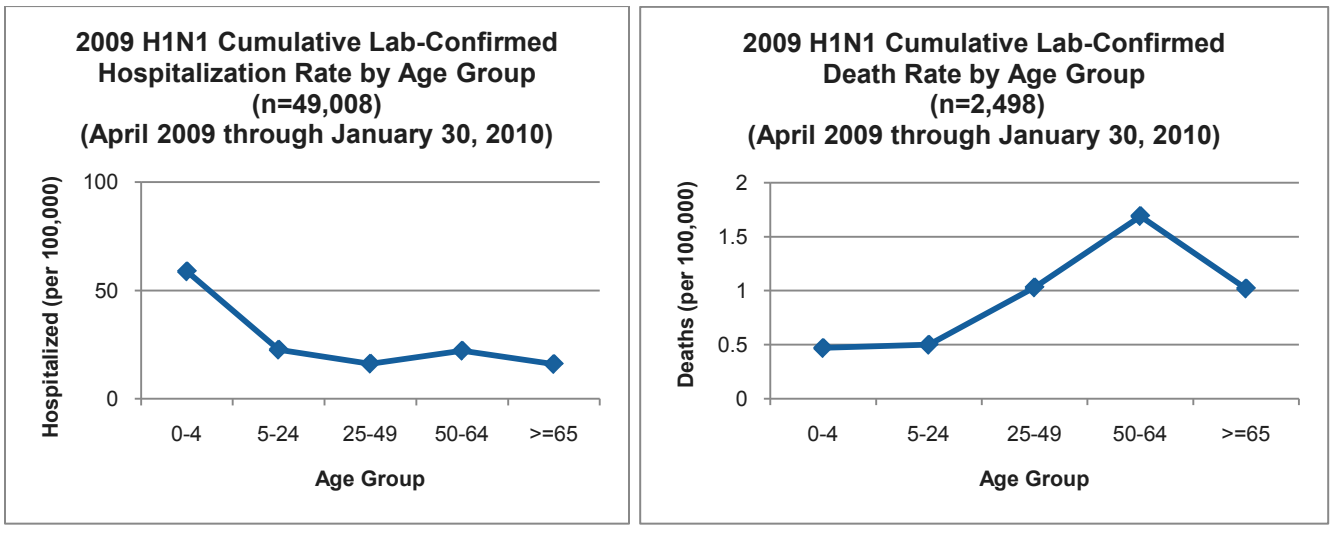

Figure 5: Age distribution of U.S. hospitalized cases (left) and fatalities (right) from the 2009 H1N1 pandemic from April 2009 through January 30, 2010 (Data Source: CDC, 2010c)

The Use of Antiviral Drugs

Antiviral medications, most effective in reducing the severity and duration of flu symptoms when started within 48 hours of the first sign of symptoms, played a key role in moderating the impact of the 2009 influenza virus. The vast majority of cases of the 2009 H1N1 infection have been treatable with Tamiflu (oseltamivir) and Relenza (zanamivir). Tamiflu, an oral antiviral drug for the treatment of uncomplicated influenza, works by blocking the final stage of the flu virus life cycle. Trials by the drug's manufacturer, Roche, show it can reduce mortality by up to 70% when administered correctly within 48 hours. However, during the course of an infection, treatment with Tamiflu may lead to the selection of novel viruses harboring mutant proteins that no longer bind to oseltamivir. No longer vulnerable to its inhibition, Tamiflu-resistant strains can emerge that circulate in the population and compete with the Tamiflu-sensitive strains.

Tamiflu-resistant cases have been reported sporadically around the world since the start of the 2009 pandemic, although evidence of the ongoing transmission of resistant strains is lacking. To date, Tamiflu-resistant pandemic H1N1 viruses have been susceptible to Relenza.

Experts worry the pandemic strain of H1N1 will reassort with Tamiflu-resistant seasonal H1N1 viruses. If the pandemic strain picked up the neuraminidase (NA) gene from the seasonal H1N1, it would likely acquire similar resistance to treatment from antiviral drugs. More specifically, if viral strains become moderately or severely resistant, the overall mortality among all age groups will increase.

The Availability of Vaccine

Pandemics occur worldwide, striking multiple locations at the same time. Therefore, local and state governments are best able to respond to the event, providing for the safety of their communities. For example, to facilitate planning in the U.S., $350 million in grants were allocated to states and territories in July of 2009 for seasonal and pandemic H1N1 preparedness. Furthermore, the U.S. Department of Health and Human Services provided online resources, such as listings of locations providing vaccinations, recommendations for individuals, families and professionals to stay healthy, and state-specific pandemic influenza preparedness response plans.

Vaccination is currently the most effective tool in the prevention of both the spread of influenza and serious illness from influenza. After sequencing the novel H1N1 virus, the WHO coordinated the collaboration of laboratories and companies around the world on the development of a vaccine. In late May 2009, the CDC shipped the seed strain to vaccine manufacturers to be grown in bulk. A rigorous process of purification, testing and quality control was implemented before shipments began in October 2009. Pandemic H1N1 flu vaccine products are manufactured in inactivated (i.e., killed virus) or LAIV (i.e., live attenuated intranasal vaccine) form. The inactivated vaccine is given by injection into the muscle, while the LAIV formulation is administered via nasal spray.

Vaccine experts provided an early production estimate of approximately 4.9 billion doses per year—a best case scenario. These estimates were later scaled back, due to production delays and vaccine yield issues. Most H1N1 vaccine manufacturers still rely on the traditional egg-based method for producing vaccines, where the virus for the vaccine is grown in chicken eggs, a laborious process that can take months.

Pandemic H1N1 vaccinations began in October 2009, when the first doses of the new vaccine were shipped, and continued throughout the flu season and beyond. In the U.S., priority was given to pregnant women, infants, healthcare personnel, individuals with pre-existing health conditions, caregivers of immunocompromised persons or infants. Despite early manufacturing and distribution setbacks, as of the end of January 2010, around 120 million doses were shipped in the U.S. (shown in green in Figure 6).

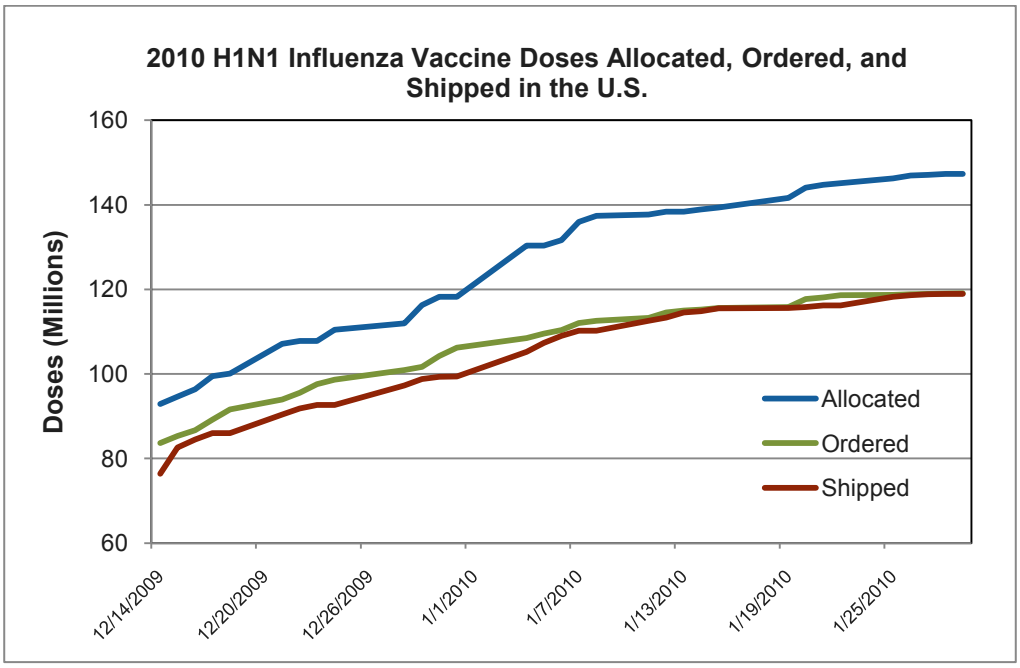

Figure 6: H1N1 vaccine availability in the U.S. from mid-December 2009 to end of January 2010 (Data Source: CDC, 2010)

Global health officials stated that the new vaccine works much better than expected, in that a single dose appears to protect people against pandemic H1N1 flu. Moreover, early experience in several countries shows the new vaccine to be as safe as seasonal flu vaccines, with little evidence of serious side effects from or due to the vaccine.

A recent CDC survey (CDC, 2010b) estimates that more than 72 million people (24% of the population) have received the pandemic H1N1 vaccine in the U.S. Children (ages 18 and under), have the highest reported vaccination rate, with about 37% H1N1-vaccinated. One reason for this success is the implementation of school-based vaccinations. In contrast, only 20% of adults (i.e., over 18 years of age) in the U.S. received the pandemic H1N1 vaccine.

In the U.S., there is currently a countrywide surplus of vaccines, possibly due to diminished perception of risk or concerns about vaccine safety (Blendon, 2010). In Europe, several countries are negotiating with vaccine suppliers to cancel their orders. The U.K. has used only 24 million doses of the 90 million ordered doses and is now negotiating with GlaxoSmithKline and Baxter to stop delivery and dispose of unwanted supplies. Similarly, the French Government is cancelling 50 million of the 94 million ordered doses, Germany is canceling half of the 50 million ordered doses, and Spain, the Netherlands, and Switzerland are looking to return unused vaccine or ship it to countries with a shortage. While the potential threat of the pandemic in June 2009, when these orders had to be placed, merited the strongest possible public health measures, officials in these countries are now dealing with public criticism that they overreacted to the pandemic.

The 2010 Seasonal Flu Vaccine

The WHO makes yearly recommendations for the seasonal flu vaccine, which is comprised of two influenza type A (one H1N1 subtype and one H3N2 subtype) and one influenza type B flu strain. The recommendations for the northern hemisphere's 2010–2011 seasonal flu vaccine, based on the current circulating strains, are to include the pandemic H1N1 virus rather than a current seasonal H1N1 strain, indicating that the pandemic strain is expected to remain more common than the seasonal strain.

While next year's seasonal vaccine could offer protection against pandemic H1N1, the U.S. CDC continues to encourage people to receive an H1N1 vaccine in the spring of 2010, as some states are still reporting flu activity. This

recommendation is based on past experience. For example, in the 1957–1958 pandemic, people stopped getting vaccinations after flu activity decreased in December and January, resulting in higher hospitalizations and deaths when flu activity began increasing again in February 1958.

MODELING THE 2009 H1N1 INFLUENZA PANDEMIC

Modeling Results

Utilizing the framework of the RMS® Pandemic Influenza Model, in July 2009, researchers at RMS began investigating over 2,000 different pandemic events that represented the range of potential impacts of the H1N1 virus as the pandemic was developing. The 2009 pandemic H1N1 virus represents a small portion of the solution space for infectious disease pandemics, and an H1N1-specific model provided real-time probabilistic insights into the impact of pandemic. The impact of each pandemic event, defined in terms of its mortality and morbidity rates for different age groups, resulted from a specific combination of pandemic characteristics and estimated using a susceptible, infected, and recovered (SIR) model. Thousands of pandemic events were simulated using this epidemiological approach and the effects of variables, such as vaccines, countermeasures, changes in virulence and transmissibility, as well as antiviral effectiveness, were quantified.

The RMS modeled results were compared to CDC estimates of fatalities and hospitalizations, as well as the expected number of cases, to assess the H1N1 modeled results against reported mortality and morbidity estimates. In Table 2, the modeled outputs are compared to the February 2010 CDC estimates, cumulative from April 2009 through February 2010.

Table 2: Estimates of fatalities, hospitalizations, and cases for the 2009 H1N1 influenza pandemic, as modeled by RMS and estimated by the CDC as of February 13, 2010 (Data source: CDC, 2010a). Note: The CDC estimates are preliminary and do not represent the entire H1N1 pandemic. These numbers are expected to increase as more data becomes available.

Age	RMS Modeled Expected Value	CDC Lower Bound	CDC Upper Bound
		Fatalities	
0-17 yrs	6,000	890	1,840
18-64 yrs	13,500	6,530	13,500
over 65 yrs	8,500	1,100	2,280
Total	**28,000**	**8,520**	**17,620**
		Hospitalizations	
0-17 yrs	71,660	60,000	125,000
18-64 yrs	155,646	109,000	226,000
over 65 yrs	102,280	19,000	38,000
Total	**329,586**	**188,000**	**389,000**
		Cases	
0-17 yrs	25,000,000	14,000,000	28,000,000
18-64 yrs	37,000,000	24,000,000	50,000,000
over 65 yrs	3,000,000	4,000,000	8,000,000
Total	**65,000,000**	**42,000,000**	**86,000,000**

The RMS modeled values are currently higher than the preliminary CDC estimates. This is due, in part, to the fact that the CDC estimates do not represent the entire duration of the pandemic; these numbers are expected to increase. The CDC estimation methodology assumes a uniform rate for all adults. Typically, the influenza mortality rate increases exponentially with age, especially over age 65. As death certificates are reviewed and deaths are counted, the H1N1

fatality rate can be expected to increase in the elderly. It is likely that fatality rates among the elderly will be lower than a normal flu season, because initial reports indicated that approximately one-third of the elderly population had some residual immunity and were less susceptible to the virus. Figure 7 illustrates the RMS modeled exceedance probability (EP) curves of fatalities from the H1N1 pandemic (classified by age), as they compare to the CDC projected fatality ranges (i.e., upper and lower bounds shown in Table 2) through February 2010. The RMS model is consistent with the CDC estimates with modeled probabilities of exceedance between 30%-65% depending on the demographics.

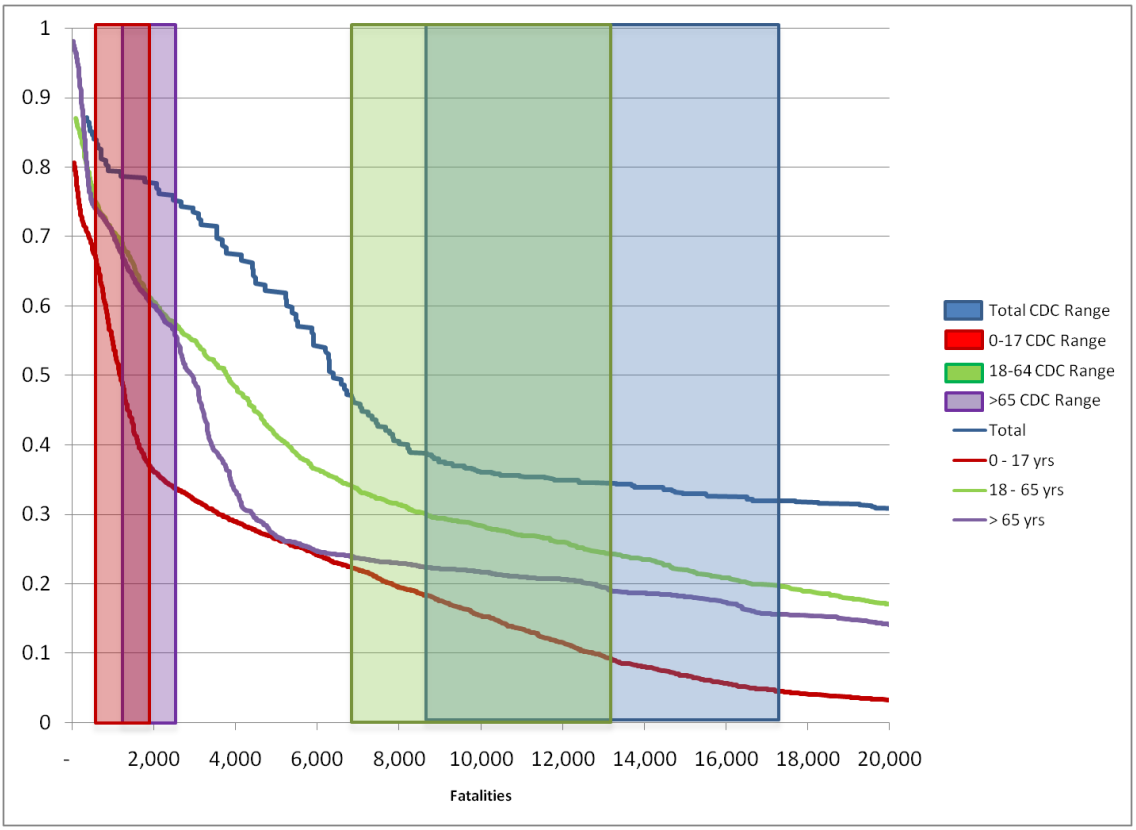

Figure 7: Estimates of fatalities from the 2009 H1N1 influenza pandemic, as modeled probabilistically by RMS (via exceedance probability curves) and estimated by the CDC (via upper and lower bounds); data based on numbers provided in Table 2.

Benefits of Modeling

These illustrations show how analytical and statistical models are critical in understanding and managing infectious disease risk. Using historical data, as well as established principles of epidemiology, virology, mathematical analysis and modeling, the range of possible pandemic events and their impacts can be quantified. Models provide a quantitative framework to evaluate risk and make informed decisions.

Models also provide a framework to explore the sensitivity of outcomes, based on various assumptions. To be most useful, models must capture underlying trends, reflect accurate data, and be able to explore uncertainty. Modeling requires a balance between data and assumptions; a model must capture enough data to adequately explain the observed trends and make appropriate selections for parameters with limited data sources. Robust modeling further involves an understanding of which variables are the key drivers of outcomes, and the uncertainty and interaction between variables.

The Future of the 2009 Pandemic

All three 20th century pandemic events leading up to the 2009 H1N1 pandemic were characterized by successive waves of infection, causing increased mortality for as many as five years after the initial pandemic. For example, the 1918 flu pandemic spread in three distinct waves during a 12-month period between 1918 and 1919 in North America, Europe, and Asia. The first wave occurred during the spring and was characterized by higher incidence rates, but relatively mild clinical disease. In contrast, the second wave during the fall of 1918 was highly fatal. Whether successive waves of pandemic 2009 H1N1 will cause higher fatality rates is unpredictable. Flu experts believe that the novel H1N1 pandemic strain will be around for many years to come and its descendents will become seasonal flu strains.

As is typical with pandemic mortality, the pandemic H1N1 virus affected a disproportionate number of people under age 65. But surprisingly, the elderly were not impacted nearly as severely as they were by the 1918 pandemic. One explanation is that older people have some residual immunity resulting from a genetically similar virus that circulated in the past, making them resistant to the dominant strain of the 2009 season. The next non-pandemic season in which multiple viruses are active may see a spike in influenza mortality in the elderly.

The presence of this current pandemic does not decrease the probability of another pandemic occurring in the near future. Viral reassortment can occur at any time, and flu viruses will continue to constantly evolve. Pandemics are inevitable and their impacts can be significant. Historically, there has been an average of three pandemics per century, but much shorter recurrence rates have been documented—in the 18th century, an interval of three years between pandemics was recorded.

Many consider the 1918 pandemic to be the worst-case flu scenario. However, it is prudent to consider a range of scenarios, as the 1918 pandemic may not be the worst case possible. There are other diseases that have shown higher case mortality rates than the 1918 flu virus—on the order of 2% to 5%. SARS had close to a 10% case fatality rate, and the avian influenza H5N1 virus continues to remain a threat. The highly pathogenic H5N1 avian flu virus was first reported in humans in Hong Kong in 1997. By the end of 2009, Egypt confirmed its 90th case of avian flu in humans, and several countries are still reporting avian outbreaks. Since the WHO began tracking cases in 2003, there have been over 260 deaths and over 440 confirmed cases globally, with case fatality as high as 60%. H5N1 has shown significant mutations since it first emerged, and it is continuing to evolve. Researchers have successfully constructed a reassorted strain between the H5N1 and seasonal flu variants (including H3N2) in animals, but fortunately this strain exhibited poor transmissibility (Li, 2010).

The possibility of the emergence of a deadly and highly contagious strain of influenza resulting from reassortment between the highly transmissible pandemic H1N1 flu virus and the highly virulent H5N1 avian influenza virus concerns many scientists. As long as human and avian influenza viruses are co-circulating, the possibility for an exchange of genetic material exists. Avian H5N1 cases have been detected throughout the world, indicating a continuing increase of human exposure to the H5N1 virus. There is currently intense surveillance to detect new influenza strains, which will assist the CDC and other worldwide health organizations to respond, as well as assist in RMS is refining its modeling of the 2009 pandemic.

While the U.S. CDC never declared an assessment of the H1N1 virus in terms of its Pandemic Severity Index (PSI), the pandemic H1N1 mortality rate has been estimated by others to range from 0.007% to 0.048% (Presanis, 2009). This rate is comparable to that of a moderate seasonal flu or a Category 1 pandemic on the CDC's PSI scale of 1 to 5. A seasonal flu epidemic would kill 250,000 to 500,000 globally, while a Category 5 would result in the death of tens of millions.

The final duration and severity of the 2009 pandemic is highly uncertain, and its analysis is based on the monitoring of a set of non-fixed factors. Accordingly, mortality from unprecedented events, such as new disease outbreaks, is difficult to estimate using traditional statistical analysis techniques. Event-based modeling, such as the RMS® Pandemic Influenza

Model, enables companies to assess their capital needs and to manage their risk, through reinsurance, portfolio diversification, and mortality bonds for the capital markets.

IMPLICATIONS OF THE 2009 H1N1 INFLUENZA PANDEMIC

Impacts on the Insurance Industry

In addition to the mortality and morbidity caused by the current H1N1 pandemic, there are economic consequences that insurers must consider. In a pandemic of even moderate severity, there are likely to significant, although perhaps temporary, impacts on financial markets, as individuals cut back on travel and recreation, and impose their own forms of social distancing. Some industries may fare well, but it is most likely that consumer behavior will negatively impact markets. For insurers, any declines in investment income will only deepen the losses experienced directly through claims. The impact of insurance claims will depend on the type of insurance.

Life Insurance

Fatalities depend on the lethality of the pandemic, as discussed in the previous section. Losses may not represent market share, as some insurers may have higher penetrations in markets susceptible to higher regional severity. Group life writers could see multiple claims from the same accounts, particularly if the virus is easily transmissible and goes undetected in a workplace.

Health Insurance

The load on the healthcare infrastructure may be very large, and health insurers are likely to bear the brunt of the cost. Concerns about infection, even if not associated with influenza, increase the utilization of medical facilities and resources. Tamiflu and vaccines will also drive up costs, as people try to protect themselves from contracting the virus. Health insurers with healthcare operations are further exposed, as their own employees, who are some of the first responders, may themselves be at higher risk.

Business Interruption

The threat from infectious disease is not limited to direct insured losses. A pandemic event can also cause severe losses due to business interruption (BI). For example, sickness in the workforce translates into lost productivity and thus business operations. It is estimated that as much as one fifth of the U.S. working-age population was infected with pandemic H1N1. Absenteeism from members of the workforce who may stay home to care for ill dependents or stay home out of fear of contracting the virus increases the impact. Losses can result from mandatory suspension due to governmental interventions as well. For example, the suspension of public activities and school closures was mandated in order to combat the pandemic.

Other BI-related losses include supply chain interruption, costs of preventative supplies and vaccine clinics, and the decrease in demand in the early stages of the pandemic, when significant fear caused individuals to restrict their activities. Industries relying on significant face-to-face interaction, such as travel companies or the entertainment industry, are most impacted. Cancellations of travel plans were common during the H1N1 pandemic, affecting the hospitality and airline industries disproportionately.

Other Impacts

Excess mortality risk from an infectious disease pandemic should not be considered in isolation. In 2010, the world has already experienced significant catastrophic loss following the destructive earthquakes in Haiti, Chile, Turkey, and China; terrorism risk also remains high in many parts of the world. Therefore, insurers may experience high claims from an influenza pandemic in the same year that there are unexpected claims from other disasters. The market mechanisms to transfer excess mortality risk are limited and expensive, although some activity in the capital markets—most recently with the issuance of Vita IV—suggests there is an appetite for this risk outside of the insurance industry. Still, many insurers will retain the risk.

Summary of Implications

Life and health insurance companies define their risk capital needs in terms of extreme events, and a major epidemic of infectious disease is a significant driver of extreme loss. This pandemic has a number of implications for the risk management of life and health insurance portfolios, as well as useful lessons to aid insurers understand and manage infectious disease risk.

Modern diseases spread too quickly to contain

The 2009 influenza pandemic was an object lesson in how fast a new infectious disease can spread through modern society. The speed at which the H1N1 virus spread to infect many millions of people was unprecedented for a disease of this scale. In contrast, the previous pandemics of the 20th century took many months to disseminate internationally. The 2009 H1N1 influenza virus became well-established in large numbers of countries within a few weeks. The volume of passenger air traffic ensured that there were infected people in many parts of the world before health authorities recognized the threat. Initial efforts to contain the disease were ineffective. It is clear that any future infectious disease will spread internationally very rapidly, and that public health containment strategies based on 20th century experience will be ineffective in the 21st century.

The low mortality of the 2009 H1N1 influenza pandemic was due to the low virulence of the virus

It was fortunate that the 2009 H1N1 influenza pandemic was mild, resulting in a low number of fatalities and hospitalizations. Although pharmaceutical advances, efficient public health measures, and pre-existing levels of immunity reduced the impacts from this event, the outcome was predominantly the result of the genetic characteristics of the virus. The 2009 pandemic influenza H1N1 virus had, in fact, the lowest virulence characteristics of any pandemic influenza virus that medical science has so far measured. It was within the range of the distribution of virulence characteristics, but precedents and early evidence suggested that the virulence would be higher. The speed at which the virulence reduced, as the pandemic unfolded, was faster than previously seen and led to overestimates of the likely overall mortality.

Severe pandemics are still possible

The 2009 H1N1 influenza pandemic is only the fourth pandemic in history for which the virulence and infectiousness parameters can be assessed with any confidence. So, this most recent pandemic, at the bottom end of the virulence scale, may imply that low virulence events are more likely in the future. However, this is not necessarily the case, as the potential for higher virulence viruses remains. Genetic permutations of viruses are random. The virulence distribution for future influenza virus mutations is derived from genetic considerations, and this pandemic will provide important, new data for research into the mutation mechanisms of influenza and the chances of more virulent viruses in the future.

There is no evidence of increased pandemic frequency

The 2009 pandemic occurred 41 years after the last pandemic in 1968, and is consistent with the historical frequency of pandemics of approximately three per century. The frequency of pandemics relies on the random mutation of a new influenza virus finding an infection route that evades the existing immunity mechanisms of human populations. Mathematical biologists have speculated that, although the evolutionary genetic mutation process of viruses is random, the fact that human populations have soared—growing from 1.6 billion in 1918 to 6.8 billion in 2010—provides a greater pool of hosts (both humans and their domesticated animals) in which the virus could potentially mutate. However, there is no evidence from the 2009 pandemic to suggest that greater human populations has shortened the period between pandemics or that frequency estimates should be increased.

The 2009 H1N1 influenza pandemic caused high mortality rates in insured populations

The 2009 pandemic had an age mortality distribution that was significantly different from expected patterns of death from influenza. Influenza predominantly causes complications and death in the old and the very young—neither of which are

strongly represented in insured portfolios. This pandemic had its highest mortality rates in the young adult and middle-aged population, which constitute the bulk of life insurance exposure. While above-average deaths occurred in young adults in the 1918 pandemic (as a result of cytokine storm processes), this impact on young adults has been assumed to be chiefly confined to the more virulent viruses. The 2009 pandemic demonstrated that low-virulence viruses can still impact healthy populations, which represent the greatest sums at risk in an insurance portfolio. The age mortality distributions that can occur in mild and moderate pandemics will most likely be reassessed, increasing the probability that an insurer will suffer losses in future pandemics.

The modeling of infectious disease risk is valuable

Overall, the 2009 pandemic progressed as expected from the scientific understanding of epidemiology and virology. The virus's transmission validated the approach of susceptible-infected-recovered (SIR) modeling, and the various interventions and attempts to combat the disease had the expected impacts. In other words, the RMS modeling of the actions taken by various national governments, as laid out in their pandemic response plans, was validated; the impacts of response plans matched closely with modeled best estimates. Forecasts of the timescale for producing a seed culture for the vaccine, the ramp-up and production volume for the vaccine, and the efficacy of the vaccine all fell within the best estimate or optimistic bracketing of the vaccine forecasts. Moreover, the modeling framework for understanding and projecting infectious disease was a useful guide to understanding the progression of this disease.

The 2009 pandemic has been a near-miss for the world. There is a danger in assuming that future influenza pandemics will be as benign as the 2009 pandemic. As the science illustrates, it is possible, and in fact probable, that the next pandemic strain of influenza will be significantly more virulent. Moreover, the time until the next pandemic is uncertain; another pandemic could happen relatively soon. In addition to influenza, there are other emerging infectious diseases that can occur, and the past few decades have seen outbreaks of diseases previously unknown to medical science, such as SARS, HIV, and Ebola hemorrhagic fever. The insurance industry needs to monitor and assess the threat of these infectious diseases in its overall management of life risk.

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