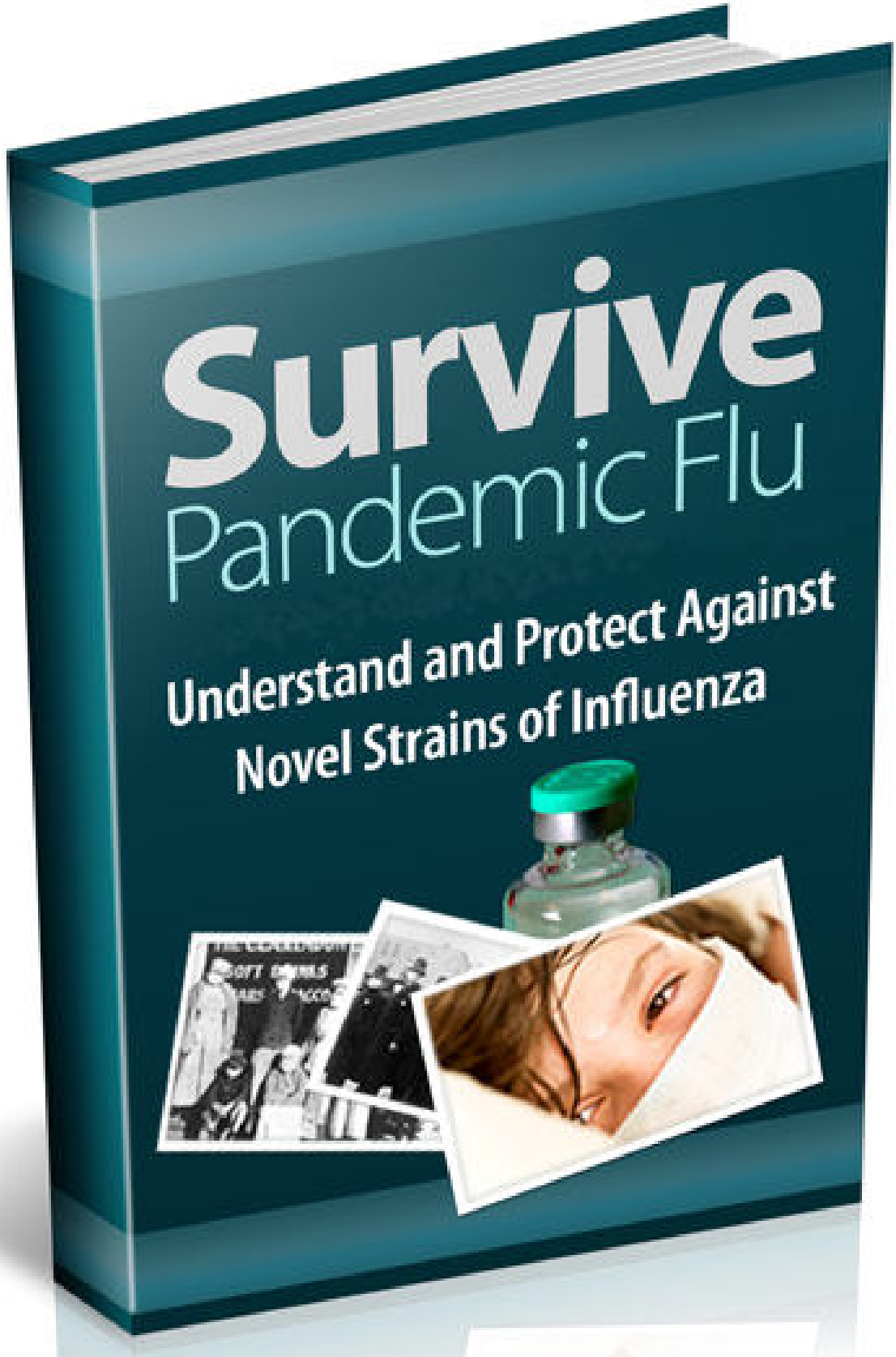
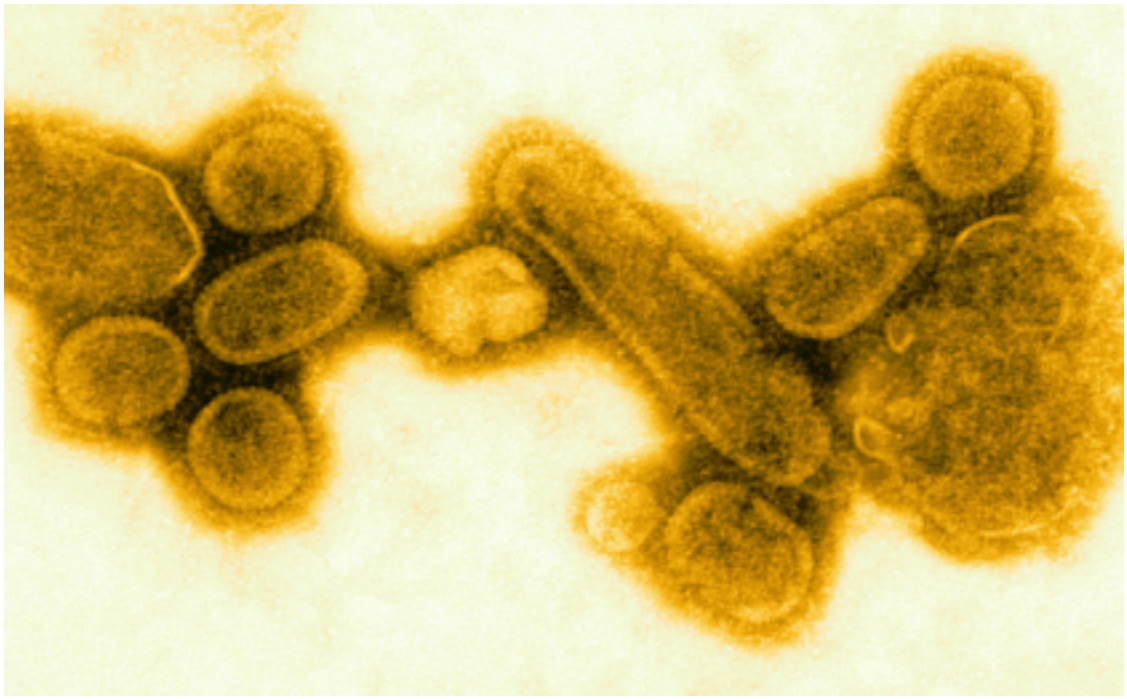

Survive
Pandemic Flu
Understand and Protect Against
Novel Strains of Influenza

Here Is A Closeup Of The Most Deadly Virus Known To Mankind



It's not Ebola, Marburg, or the Plague.

It is Influzena A - and in the fall of 1918 a strain emerged that would go on to kill an estimated 40 million people. It came to be known as Swine Flu...

This is the story of pandemic flu, past and present.

- Survive Pandemic Flu -

A Preview

Understanding And Protecting Against
Novel Strains Of Influenza

Author: Stephen Carter

http://www.survivepandemicflu.com/

- A note about this document from the author -

The **purpose** of this document is to provide a preview of the ebook Survive Pandemic Flu, which is a resource designed to background the story of pandemic influenza, and recommend best practices in order to reduce the likelihood that you or your family will come in contact with them. The book was written in response to the emerging H1N1 swine flu pandemic and should be regarded as a work of opinion.

Disclaimers

This guide contains information about pandemic strains of influenza, as well as advice on how to reduce your chances of being infected by such strains. This advice may or may not help you achieve that goal. I make no warranties or representations, express or implied, about the completeness or accuracy of the information found in this guide, and specifically disclaim any notion that by following the advice found here you are guaranteed to avoid pandemic strains (that would simply be unrealistic).

Despite the fact that organizations or websites may be referred to in this publication as a further source of information, I make no endorsement of the information the organization or website may provide, or recommendations it may make. Further, readers should be aware that websites mentioned in this work may have changed or disappeared between the time this work was written and when you read it.

Table Of Contents

About The Author

Feel free to jump ahead to the next section if you aren't particularly interested in WHY exactly I decided to write this little book about bird and swine flu viruses which are the cause of pandemic influenza in humans.

The reason I have added this section is so that you will be better able to put what I tell you into context, but it is otherwise completely ancillary material.

For several years the subject of the most notorious instance of pandemic flu occupied my thinking night and day. I am of course talking about the great Spanish Flu pandemic of 1918-1919. You could almost say that I was obsessed with it.

But not because I was a medical researcher trying to better understand the nature of a virus that took so many lives over nine decades ago. Nor was it because I was a historical researcher in any real sense.

I thought a lot about influenza A, and the so called swine flu strain of 1918 in particular, because it was the central component of a novel that I was writing - a science thriller. That book ultimately turned out to be very long, with lots of complex story lines, and a great deal of real science.

Now, the reason I emphasize the "real science" aspect is because I was trained as a scientist (I have a doctorate in the physical sciences). So I was a scientist turned novelist, and not a novelist writing about a science he barely understood. For three years I threw myself into the challenge of writing my novel, and I drew upon all of my intellect to get the science correct. The end result was that I got a thorough education in the biology of viruses, and a fairly scholarly understanding of the molecular biology of influenza A.

Without a doubt, I learned more during that three-year period about the influenza A virus than 99 percent of medical doctors ever will - and that is my ONLY qualification for writing the book you are reading now. Well, that, and the fact that I care enough about this subject to spend the time to assemble this information for you.

Also, as a novelist, it does help somewhat that I know how to string a few words together and make the journey relatively entertaining for you. Yes, it is a serious subject, but unless I entertain you just a little as I write I fear you may not reach the end of the book. I certainly do not want that to happen, as the subject is not just serious, it is very important - and the threat of deadly pandemic influenza is not going away any time soon, so the more people that understand the dimensions of this problem, and how to deal with it, the better.

As for that novel, agents and publishers thought my story line was too involved, and my work just a bit too ambitious for a first-time novelist. In the end I published the book myself as Ninth Day of Creation and turned my attention to things other than trying to write novels that nobody wants to read. But while I have largely forgotten the exact details of my novel's story line, it is much harder to forget the remarkable story that surrounds this particular virus, and the impact it has had on our approach to the problem of sustained contagious infections in the modern era.

One last thing I should probably mention is that I have deliberately refrained from trying to back up all my assertions with references to obscure and arcane papers that, while nonetheless critical to figuring out what is important and what is not on the subject of pandemic influenza, I know that you are not likely to spend any time trying to read yourself. I will simply be boiling down the essence of what I have uncovered.

So, I will be doing my best to keep the dialog light, swift, and entertaining, while I educate you on the nature of the most virulent killer microbe to emerge during the course of the 20th century, and how you can protect yourself from coming in contact with similar modern day strains.

Cheers,
Stephen Carter, Ph.D.

About The Book

This **preview** of *Survive Pandemic Flu* consists of the first part of the book, which backgrounds the nature of pandemics by looking at the history of them. The remainder of the book is outlined at the end of the preview, on page 46, and hints at the exploration of the biology of influenza, and the means by which we can protect ourselves against the threat of pandemic influenza. Now that I have set the scene, beginning from the paragraph that follows this one, everything else you read is cut directly from *Survive Pandemic Flu*...

When I set out to write this book I did so in part because I felt that I knew enough about influenza A and the story behind it that I could convey in a straight forward, but accurate, manner the important things I think everyone should know about pandemic influenza - the stuff you *need* to know because it might just save your life one day, or the life of someone close to you. Yes, that may sound a little melodramatic, but influenza has a rich history of striking at us in ways that are characteristic of anything but melodrama. The short and simple truth is that, because of its capacity to spontaneously produce shockingly lethal strains, it is currently the most dreaded microbe we face today.

Yes, the plain old flu. Well, the old part turns out to be correct. Plain - not so much.

As I got deeper into the side issues that surround this menace, such as disaster preparedness, and the woolly topic of trying to predict what might happen during the aftermath of a serious pandemic, it dawned on me that the whole subject is incredibly complex - more so than can be explored in a short ebook.

Having said that, my goal has always been to put together a **basic summary**

of all the things I consider to be important about pandemic influenza. My sense is that you probably do not want to be overwhelmed with information about a topic that most people do their best to ignore anyway.

So you should consider the contents of this book to be a **primer** on the subject of pandemic influenza. I actually believe it to be more than just that, but it is a lot less than encyclopedic. Hopefully you will find that it has about the right balance of material, and it will give you ideas about things you might want to go off and explore on your own when you have reached the end of the book. If not, at the very least, I hope you will feel comfortable grabbing this book again in the future when you feel the need to refresh your memory about one aspect or another of this subject.

The first part of the book gives you the background for this subject, which I consider important, because I do not really think you can protect yourself against a threat unless you have a real appreciation for what makes it a danger to you. The second half of the book offers suggestions on things you can do to minimize the chance that you or your family will become victims of pandemic influenza. If this book spurs you to do nothing else but follow the suggestions in the concluding Action Plan, then I feel you will have got more than a little from reading it.

Finally, a note about the references.

As I pointed out earlier, I have tried not to litter the text of this book with too many references and hyperlinks because I suspect few readers will feel the need to follow them. Another good reason not to add hyperlinks has to do with the ephemeral nature of the internet: link today, gone tomorrow. It is frustrating to attempt to follow a link to what appears to be an interesting supporting reference, only to discover that it is no longer functional. Someone has moved the page, or renamed the file, and it cannot be accessed.

Another reason not to pursue hyperlinking in a work that people might refer to during a pandemic is that there might actually not be an internet to access. A simulation of an influenza pandemic carried out by members of the World Economic Forum showed that the internet could shut down during a severe pandemic in a period as short as two days. This is why I have included several freely available public PDF documents as resources rather than link out to them at their home sites. For example, you can find the results of that WEF simulation in *resource-Influenza-Pandemic-Simulation.pdf*, one of the enclosed resources.

Having made the argument for not hyperlinking, you will nonetheless find that I have hyperlinked some references that I feel are worth checking out, and which do not lend themselves to reproduction as a resource document. These are supplementary references. If you discover that they are not available when you go in search of them, then their absence should not significantly disrupt the flow of information as you read this book.

Is This For Real?

It would be a huge mistake to think that just because you are reading this book and have some appreciation of the menace that bird flu and swine flu represents to us, that everyone shares your unease. This is not the case at all.

In April of 2009 I was in my local DVD rental store in Pasadena, California, looking for a movie to share with my son, when a customer to the side of me struck up a conversation with the store owner. He could not believe all the hoo-haa the government and the media were making over this thing they were calling the swine flu.

The day before this, the World Health Organization had raised the flu pandemic warning level from 4 to 5, meaning that governments should immediately prepare for the possible emergence of a global pandemic threat. Infections had been reported in many countries around the world, and the spectre of a long forgotten episode which had taken place ninety years earlier loomed in the minds of those health officials who made it their job not to ignore the lessons of history.

But at the time, the new strain that had the government and health officials so worried was thought to have caused the death of only one person in the United States.

"Swine flu," said the customer, mocking the name. "North American H1N1... Well, I guess they had to give the thing a name, didn't they?" He shook his head in disbelief. "You been *listening* to this stuff?"

"Oh, it's unbelievable," said the store owner, dismissively. "It's these 24-hour

news cycles, see. They gotta run with *something*. Apparently this is the best they can come up with right now."

The conversation quickly ran its course and the two went back to what they had been doing.

I have been frequenting this store for years and I had taken the store owner for a fairly smart guy, but it was clear that on this particular matter he had no clue. Nor did the customer. And that got me thinking. Despite all the news coverage surrounding the swine flu story, how many other people were simply blowing off the health warnings as yet another hyped-up news story?

As it turns out, most people share the view of the store owner and the customer. At least, if online discussion threads reflect the reality of the situation, which I suspect is the case. This should probably not surprise anyone, as most people just do not care about matters that do not *immediately* affect them. And they will maintain this attitude right up until the time the local school closes its doors and sends all the children home for "early vacation", at which point the penny drops and they finally say:

"Wait. Is this for real?"

Just How Scared Should We Be Of Bird Flu Or Swine Flu?

Then again, it very well might *not* be real. What appears to be a potentially very harmful bout of influenza at the national or global level might simply peter out before it can do much harm.

Sometimes the warnings are genuine false alarms issued when government officials misread the cautionings of their scientific advisors and go public

because they feel it is "better to be safe than sorry". As we will see later in this book, this has happened before (notably in a case involving the threat of swine flu in 1976), and it leaves a very sour taste in the mouths of the public who end up footing the bill for preparations for a pandemic that never arrives.

So when you turn on your television and you see the anchor man on the evening news telling you about this new strain called "swine flu" which has health officials concerned, you do not need to feel stupid for asking the obvious question: "Just how scared should I be?"

As I write this, the strain of swine flu that is currently receiving world wide media attention is suspected of having had its origin in a pig farm located near La Gloria, in Vera Cruz, Mexico, and is thought to have first emerged in the early months of 2009 as an entirely new strain of influenza that we have not seen before in humans. In 1997 we saw a similar story with the emergence of a new strain of bird flu in Hong Kong which was capable of infecting human hosts and killing about fifty percent of its victims. Fortunately the bird flu strain has so far proven to be only weakly transmissible in humans.

But these kinds of incidents are not particularly surprising news to anyone with some understanding of the influenza virus. As we will see later in this book, when we go into the biology of the virus, new strains of influenza appear every year in different locations all around the world. Strains are as widespread, varied, and relatively-speaking scarcely more threatening than weather patterns for the average person (the elderly, with their weakened immune systems, being the chief exception here). But every once in a while that "perfect storm" arises that causes complete and utter havoc for human beings.

Swine flu is variation of influenza A which circulates in pigs and crosses over into the human population on occasion (though very rarely). The image shown

below demonstrates one mode of transmission that should obviously be avoided.

We will learn more about the "mechanics" of this species-jumping process later, but you can think of pig intestines as a natural incubator for a variation of influenza A which is known as H1N1 (I will detail the meaning of the H and the N designations in the next chapter).

To put this into perspective, there is another strain known as H5N1 that percolates in the intestinal tracts of ducks and gulls, and which sporadically gives rise to avian flu in other species of birds that do not ordinarily host the virus. Both of these strains of influenza have the potential to be lethal to human beings when they become infected by them.

The key word here is *potential*. Pigs are known to have been domesticated by us for at least 7,000 years, and for the most part we co-exist with them without a problem. We have also been cultivating ducks for about 4,500 years, and they do not seem to cause us much problem either. Ducks tend to experience very mild flu symptoms, and only once early in their lives, and while pigs do routinely get sick with their own flu strains, none of this normally affects us.

But every once in a while something dramatically different takes place. That perfect storm alluded to earlier crashes unannounced upon the shores of an ill-prepared population of human hosts, sometimes infecting up to one of every three people on the planet.

The last time this happened was in latter months of 1918 and the early months of 1919.

1918 - When Monsters Come Out To Play

At the time, the science of virology was just emerging. Nobody understood the cause of influenza, and, just as today, virtually no one had any appreciation that devastating bouts of the disease had struck before in recent recorded history. In 1580 a flu pandemic had spread across Europe, following world trade routes. It was said to be so bad that "some Spanish cities were said to be nearly depopulated."

More recently, in 1889, a strain had emerged from Bukhara, Russia, which claimed at least a quarter million lives in Europe alone.

The origins of the flu that caused these two pandemics, in 1889 and 1918, will never be known for sure. The standard assumption is that an animal strain, of avian or swine origin, managed to jump the species barrier and set off a chain reaction in the human population (although we will see later that this kind of event might be more subtle than has been believed to date). But it would not be until the passing of the 1918-1919 episode that evidence of this animal-to-human mode of transmission was finally demonstrated.

Today, based on the results of twenty-first century gene sequencing techniques that have been applied to remnants of the 1918 virus, as well as the genetic profiling of strains collected from many different species in the years that have

followed 1918, two competing theories have emerged.

In the first theory it is speculated that the pandemic was likely initiated by an instance of bird flu that leaped directly into the human population. That is, a sick bird somehow managed to transfer virus into the respiratory passages of a single human host, and the resulting infection spread from that one person to a large fraction of the rest of the human population. This has been the prevailing theory since about 1997 when we first got a close look at the genes involved in the 1918 episode - the backstory of which I will provide later in this book.

Prior to this, however, the first indications of animal involvement - revealed with the introduction of the new antibody science of the 1930s - pointed to swine as the source of the pandemic. Hence the name, *swine flu*. From the early 1930s, right on up until a sample of the 1918 virus finally became available for testing, it was believed that sometime in 1918 a strain of swine influenza made its way from a single pig into a single human host, and then proceeded to spread globally.

The scientists who sequenced the 1918 strain in its entirety over an eight-year period ending in 2005 provided compelling genetic evidence that the strain originated with birds (possibly a goose was involved), and was only passed into pigs *after* going through us. While the question of exactly what had happened was not by any means fully answered, there was little debating that the event had involved a non-human strain of flu that found a way to propagate itself by exploiting the cellular machinery of humans.

So that is the first theory - an avian flu strain jumped directly into the human line in 1918 and set off a chain reaction that proved devastating for us. The second theory is quite different, and is actually more worrisome in its implications. It was published in mid 2009 by infectious disease scientists Drs. Gavin J. D. Smith and Robert G. Webster, and their colleagues, from the

Laboratory of Emerging Infectious Diseases at The University of Hong Kong.

The theory put forward in this paper, included as the resource document *resource-1918-Pandemic-Origin-Smith.pdf*, is that the genes of the 1918 pandemic virus did *not* come directly from a bird, but had in fact been circulating in pigs, and perhaps humans, for many years prior to 1918 - possibly as far back as 1911.

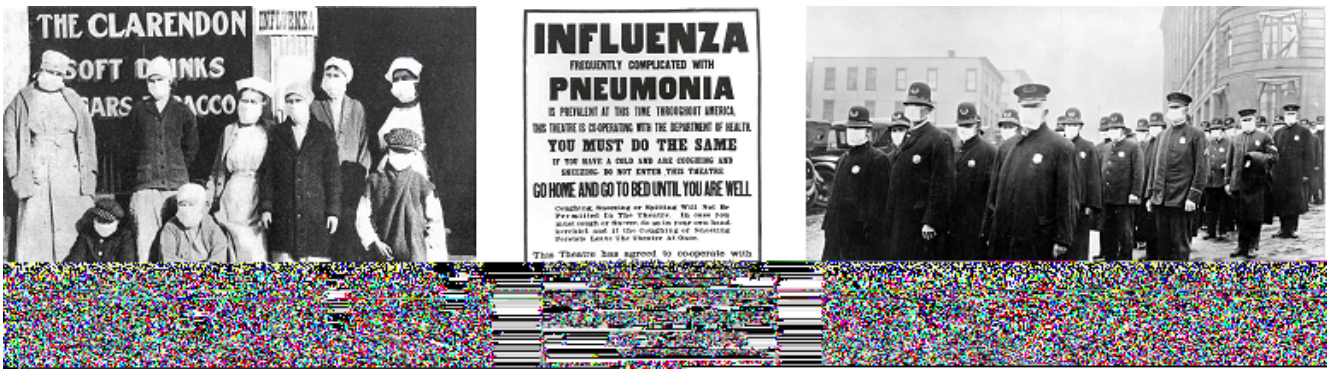
So we are back to the pig theory again, but with an important difference. Smith and his colleagues believe that the deadly cocktail of viral genes had been moving around in mammalian hosts for years, causing no problems while they were apart, but when they finally came together in 1918 the combination proved to be of just the right mix to initiate a pandemic. The evidence for this is based on a careful deconstruction of the genes responsible for all three pandemic influenza viruses of the twentieth century: the 1918 H1N1 strain, and the H2N2 and H3N2 strains of 1957 and 1968 respectively. The conclusion of this work is that pandemics *may* be seeded years in advance by avian flu gene crossover events that then percolate in mammals for some time before catching fire and "going viral" as a pandemic.

If Smith and his friends are right, then the implications for the 2009 strain of swine flu are clear: do not count this virus out simply because it appears so far to be a mild one. The spontaneous generation of a far more lethal strain could happens months, or even years, later. This is something to keep in mind as you read through this book. As in 1918, the current pandemic strain of 2009 is of the H1N1 variety (which I will elaborate upon in the next chapter) and its component genes may have been circulating in both humans and pigs for some time before going pandemic.

Let's take a closer look now at what we know about the pandemic of 1918.

One of the most striking characteristics of the 1918 strain was that it proved to be particularly transmissible and spread rapidly. Despite the leisurely transportation methods of the time (steam boats and locomotives) the virus managed to spread itself around the world in just six months.

It also came in waves. At first, the severity of illness was not so great as to make people believe that something new and alarming was taking place, and this likely helped the new strain to spread across the globe.

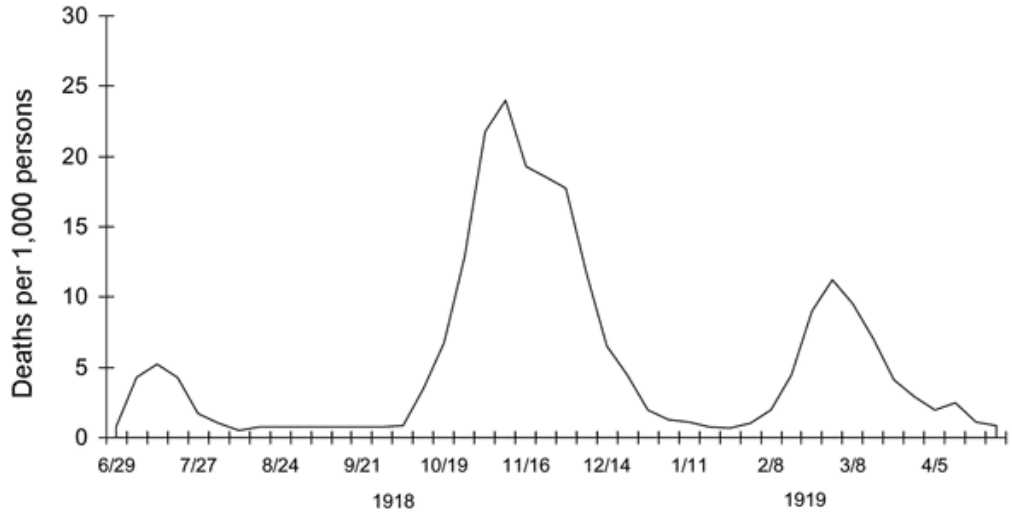


But then something about the virus changed, and in the course of the second wave it revealed itself to be a true monster capable of infecting and killing its human host within a period of about a day. In many cases secondary infections, like bacterial pneumonia, which account for many influenza-related deaths, did not even have a chance to arise.

In these atypical, but notable instances, the virus killed its host directly. Although the mechanism by which this took place was not understood until just a few years ago, victims of this rapid onset of "viral pneumonia", as it was called, saw their lower respiratory tract fill with blood and they literally drowned in their own body fluids while in bed. Some never even made it that far, and died on route to their workplace, evidently feeling well enough a couple of hours earlier to leave their house.

If you look at the graph below, which records the mortality rate at a U.K.

location during the nine month period that spanned the three waves, you can see the death rate rising and falling, with peak mortality occurring at intervals of approximately four months.



The reason I am showing you this graph is that it demonstrates the completely unpredictable course of a pandemic driven by a virus that can mutate and become even more virulent over time.

Most deaths took place during the months of September through December 1918, the time period that corresponds to the fall months in the Northern Hemisphere.

From a layman's point of view we could be forgiven for expecting to see just a single rise and fall in mortality rate over the course of a pandemic as the virus spreads through the population and kills off the portion that is susceptible to it, leaving alive the hosts whose immune systems can fight off the infection.

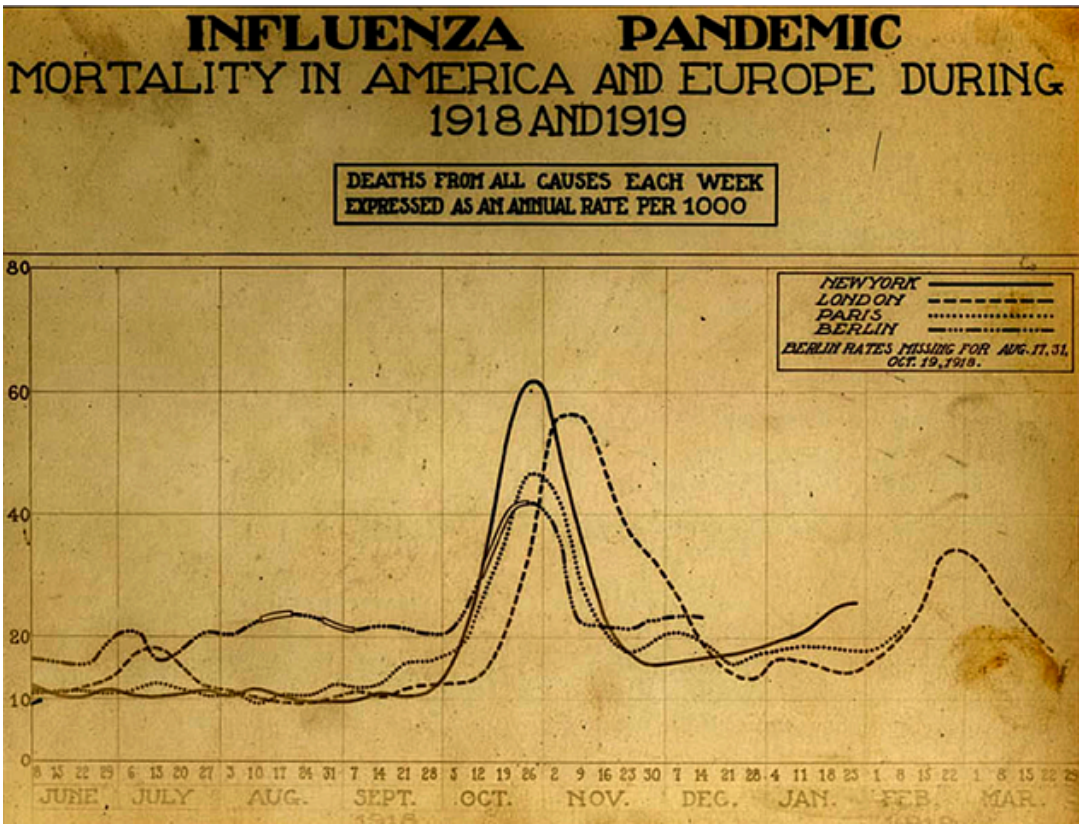
But in the case of the 1918-1919 strain something happens to restart the escalation of deaths twice more after the first episode. Because of this, it is important that when any modern flu pandemic is set in motion we do not automatically assume the worse is past when the mortality rate initially

appears to crest and fall away. This may prove to be merely the precursor to the main event to follow.

This non-predictability in the progression of an influenza pandemic is particularly unnerving, since the general public is almost guaranteed to let down its guard after the first spike in the number of deaths, and then go about their business and assume all the health warnings they heard about were just another false alarm.

This is the time they toss out the safeguards and unknowingly invite the mutating monster back into their lives.

Something else about that three-humped graph is particularly unnerving, but it only reveals itself when you look at the mortality curves as a function of time for widely separated locations, as in this chart:



You can see here that the second wave struck virtually simultaneously in all major cities, despite the fact that transportation lines back then were limited by the speed at which ships could cross an ocean. So how did the virus explode in severity everywhere at the same time?

Clearly the virus was already in place and spreading throughout these cities by the beginning of October 1918. It is quite possible that when the virus began spreading pandemically earlier in the year that it contained a gene only a little different in form from one that would ultimately be responsible for the uncharacteristically high death toll seen in the winter months, and that it was only a matter of time before that gene evolved to its deadlier form.

This genetic mutation could have happened everywhere in early October, but it is more likely that it happened in different locations at different times toward the end of summer, and that the arrival of winter helped fan the flames of the second wave. The influenza A virus seems to relish the cold low humidity air of winter.

When the pandemic struck in 1918, particularly during the second and third waves of the illness, the immune systems of many victims were simply unable to mount an offense to the new virus quickly enough, and it decimated communities everywhere. Fully twenty-two percent of the population of Western Samoa died. Some Alaskan Eskimo and South African villages were entirely wiped out. In the United States an estimated 675,000 people died. Across the globe, the total number of deaths is thought to have been somewhere in the region of 40 to 50 million, and may have even been twice that number.

No other recorded plague, or natural disaster, is known to have resulted in the loss of so many human lives in so short a period of time.

So - should we be scared when swine flu or avian flu outbreaks make for the lead story on the evening news every day for a week or two? My answer is yes, *absolutely*. When that happens we should be very worried indeed. I have known about the very real threat of another influenza pandemic since 1995 when I first began studying it, and it has bothered me ever since, as it should bother all of us.

How Likely Is A Repeat Of The 1918 Episode?

This is the $64,000 question. As you will see in this section, most virologists who study influenza pathways are not at all hopeful that we will *not* see something like 1918 take place again.

In order to get a handle on the answer to the question we really need to understand a little bit about how influenza goes from being a relatively familiar illness that makes us feel miserable, to something which exhibits a much higher death toll than usual when it sweeps through the human population in pandemic fashion.

For perspective, it is important to understand that in the United States about 36,000 deaths, on average, are attributed to seasonal (regular) influenza infections every year. But most of these deaths involve aged patients whose immune systems are weakened, and who cannot put up a very good natural defense. Pandemic flu, which occurs when an entirely new strain is introduced into the human ecosystem, can also take the lives of those who seem to be in the prime of health. The Spanish Flu, as the pandemic came to be known, was notable for taking the lives of people between 20 and 40 years of age.

In order to get some idea of how likely it is that a pandemic event will occur in the near future (ignoring for the moment that we are already headed down that road in 2009-2010), and that when it does it will be a bad one (which is all we

really care about), I will break the discussion down into a few short sections.

First up, the cause of all the problems...

Blame It On The Birds

By the 1980s it had been established that influenza A is fairly widespread in the animal world. In addition to being a human pathogen, it was seen to take up residence in pigs, horses, seals, and a variety of birds. There are two other (less serious) forms of influenza, referred to as types B and C, though only influenza A in known to infect non-human hosts.

But it is the aquatic birds of the world that seem to be the true reservoir for all strains of influenza A. Robert Webster, a virologist specializing in influenza A, put forward this idea in 1980. If the name seems familiar it is because he is one of the authors of that 2009 paper which proposed the idea that novel influenza genes may float around in pig and human populations for years before initiating a pandemic.

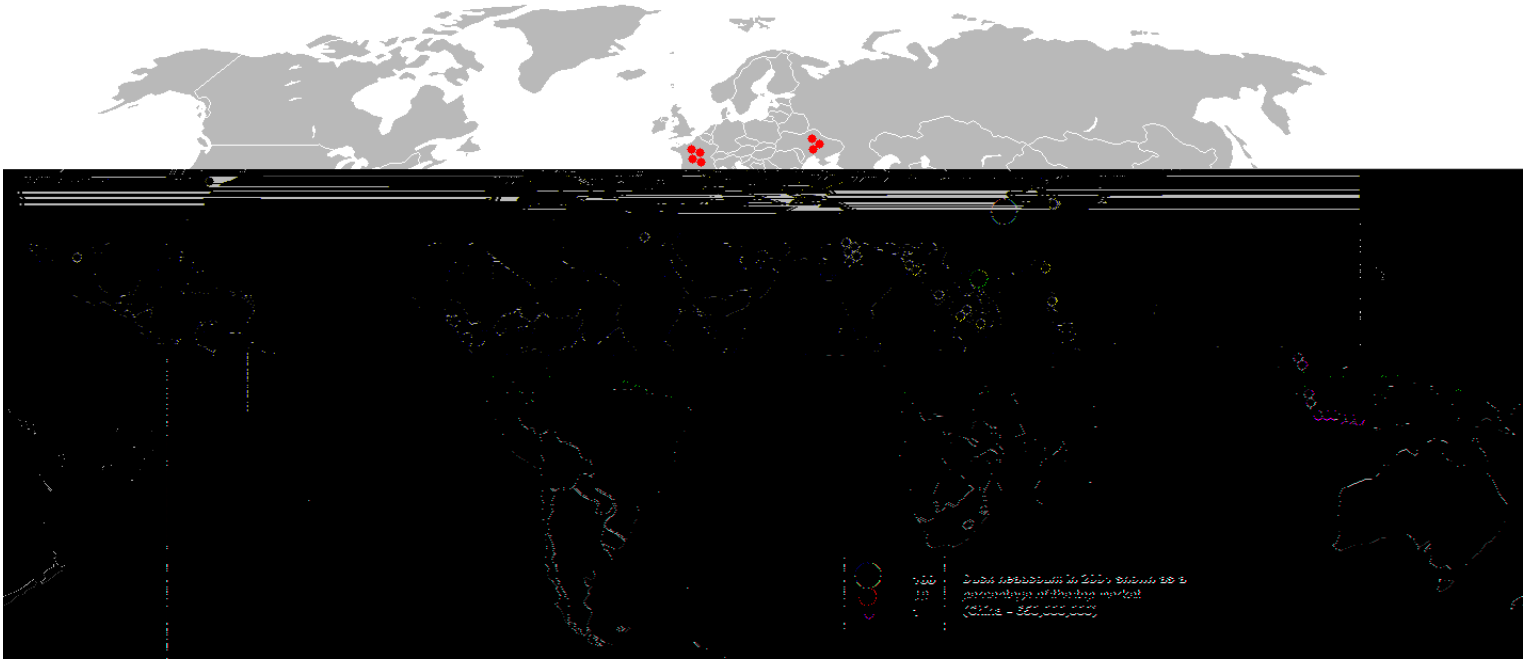
Back in 1980 Webster pointed out that wild ducks, and other water fowl, had achieved perfect co-adaptation with the influenza A virus, and rarely ever showed any symptoms of distress.

Influenza A reproduces benignly in the intestines of these birds, who then

spread their feces throughout the waterways of the world. This provides for a perfect reproduction strategy as the influenza virus can survive for days, or even weeks, in a body of water. Ducks and other water fowl then drink from their own self-contaminated waterways and give the virus plenty of opportunity to move from one bird to another.

So wherever large bodies of fresh water are to be found in climates conducive to the congregation of aquatic birds, there is influenza virus far and wide. Naturally enough, in areas where humans take advantage of the abundance of these birds to hunt them, farm them, or otherwise interact with them, there is the potential for an avian strain of the virus to make its way into humans.

Take a look at the image below, which shows the distribution of domesticated ducks world wide in 2004.



The large green dot in this image represents 650 million ducks located in Northern China, while the yellow dot shows about one tenth that number, and elsewhere the red dots represent about a hundredth the number. It is this huge reservoir of domesticated aquatic birds in China that points to it as the most

likely place to generate novel strains of influenza.

Fortunately bird-to-human transmission of influenza virus turns out to be quite difficult to achieve. Bird flu virus just does not like us that much. This should not be too surprising, since the virus has adapted so well to the cellular environment of its native non-mammalian host - so much so that taking up residence inside a human cell requires some serious adaptations.

This is where pigs are believed to provide an unfortunate bridging station for those finely tuned avian strains of flu. Nobody needs to be reminded about the filthy habits of pigs, who will eat whatever is put in front of them. They also enjoy raking about in whatever muck they can find. When you introduce ducks into the vicinity, as may happen in farming environments, it does not require too much imagination to see how the virus-laden fecal matter of ducks can end up in the belly of a pig. In fact, the Chinese practice of "aqua culture" involves feeding bird feces to pigs, then pig feces to pond life, so as to streamline the farming process toward greater cost-effectiveness. Of course, these "efficiency measures" virtually ensure that animal flu strains get plenty of opportunity to mix.

Despite the observation by many researchers that birds seem not to be able to pass on flu very easily to humans, this nonetheless may have been what happened in 1918, or in the years prior to it, and it has since been observed to happen on about a half dozen occasions in the last decade or so. Fortunately no sustained human-to-human transmission has been observed. If it had, said one notable scientist in this field, reflecting on the extreme lethality of bird flu infections in humans, "We'd be screwed".

We may never know for sure whether the 1918 event involved a direct to human transfer of avian influenza genes, or whether pigs provided the intermediate stepping stones, as the most recent research suggests. What we

do know is that the 1918 virus, with its high concentration of novel flu genes, was *extremely* contagious. This is why news publications today seem fixated on the idea that a human version of bird flu could decimate us. If bird flu jumped into the human line in 1918 and went airborne (meaning that infections are triggered by merely exchanging the air we breathe), what is to stop it from happening again?

This begs a very important, and so far unanswered, question. Is there any *fundamental biological impediment* which ensures that a bird flu strain that kills fifty percent of its victims *cannot* arise in the human population? This question really scares influenza scientists, because so far they have not been able to convince themselves that there is such an impediment. In fact, the question they really seem to be preoccupied with is this one: Is luck all that separates us from coming face to face with a nasty biological event similar to the one that occurred in 1918?

Because if it is, then it is just a matter of time.

Careful monitoring of small-scale influenza outbreaks suggests very strongly that chance plays a big part in the initiation of a pandemic, and this has likely always been true.

Linus Pauling, the Nobel Prize winning chemist, has pointed out that we know influenza is an old disease, because in 400 B.C. Hippocrates wrote in his book *Epidemics* about a respiratory disease that ravaged the inhabitants of Perinthos in Crete. The ancient physician's account of the symptoms experienced by the afflicted people of Crete have convinced many that influenza was indeed the cause of the epidemic.

So serious influenza epidemics have been with us for at least 2,500 years. Influenza specialists think the actual length of time is more like 4,500 years,

because this is roughly how long we have been farming ducks. They believe it is entirely possible that before this time influenza was essentially an avian disease walled off from humanity, and perhaps even from pigs.

Other empirical studies have shown that for pandemic events to arise in the human population, a certain minimum population size is required in order for the pandemic to take root, and that the minimum size for this to happen is around half a million people. Such population sizes are not thought to have occurred earlier than perhaps 10,000 years ago. So there seems to be a population size factor to the likelihood that a newly-emerged novel strain of influenza will give rise to a full-blown pandemic. A contagious virus that arises in the rural setting has the odds stacked against it, since it is likely to be suppressed by the collective immune systems of the hosts it infects *before* it can spread at a geometric rate. But you put one of those sick people into a city of one million people, or ten million people, and it blows up. It goes everywhere.

In the modern world, it does not matter that a virus may start spreading within a small community, because all small communities are now connected by cars to large towns, or cities, and those in turn are connected by airports to other cities around the world. Today, the epidemiological population size of the community we live in is six and a half billion - no matter where we live.

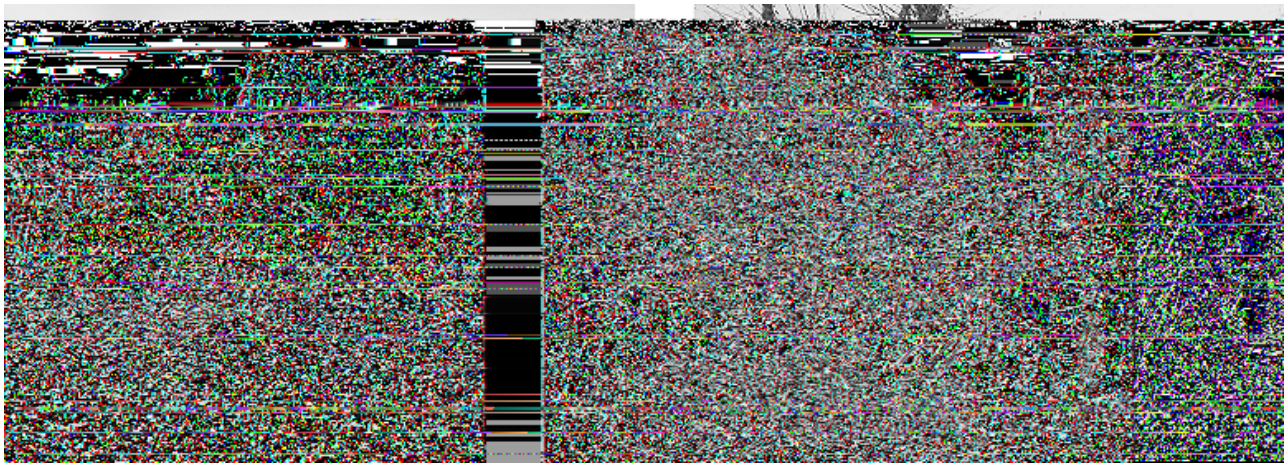
The Broiler House Theory Of Pandemics

Many observers have wondered over the years about the coincidence that allowed the Spanish Flu pandemic to take hold in Europe at the very time that World War I was raging. Conditions certainly seemed ripe for spreading a human pathogen in the overcrowded, disease-ridden, and stress-filled trenches of the battlefields in France. Nor could the packed cattle cars of soldiers moving into and out of the region have helped matters any.

But what if it was *not* a coincidence? What if the extreme lethality of the 1918 strain was a *consequence* of the similarly-extreme battlefield environment of the day?

Military medical historian Carol Byerly has pointed out in her book *Fever of War: The Influenza Epidemic in the U.S. Army During World War I* that the trench warfare conditions experienced by soldiers during the four long years of WWI were unimaginably hard on the men. Certainly no one who signed up to fight for the honor of their country had any idea about what they were in for. Soldiers found themselves in muddy, water-soaked, and corpse-filled trenches from which they could not escape, while above their heads an endless rain of bullets and the constant threat of chemical attack left no doubt in anyone's mind that death might be just a few inches or a few seconds away.

The war was a kind of grinder, into which healthy soldiers were fed, only to be quickly converted into spent shells of men who bore neither hope nor the ability to adequately fight off infection when sickness came. Today we would call them immuno-compromised, and on the battlefields of 1917 and 1918 millions of these men crammed together in the trenches, and when off the battlefield they crammed into farmyards converted to army camps and other rural properties converted to makeshift hospitals.



In doing so the pathway for animal to human transmission was set up. BUT the important point is not that animal infections were made more probable, but that the close-packed disease-sharing environment the men found themselves in may have *coaxed* the killer microbe that would ultimately emerge into revealing itself.

How? The explanation runs something like this. Bird flu is transmitted from one bird to another through contact. A chick sticks its beak in that of the parent. Two birds at different times drink from the same puddle of water (if we are talking about ducks, then they practically live in water). If bird flu gets into a human, the transmission route is again through contact. But under normal circumstances human beings do not exchange virus through contact so easily. Especially if they live on farms ten miles apart, as farmers tend to do. So the infection cannot travel that widely in the population.

Everything changes when you huddle those infected people into a trench during war time and force them to share what limited supplies are made available to them. Infections that can only propagate via contact, and which might normally burn out very quickly, are instead sustained.

What does this mean? It means that any fairly innocuous bird (or pig) flu virus that manages to find a way to infect human cells in just one person, now has a way to keep experimenting as it reproduces in that host, and any other host that it manages to latch onto via shared body fluids. One soldier sneezes in the face of another, or shares a drinking cup, or cigarette, and so on.

The infection is not only sustained because of the unnaturally close contact that the hosts are forced to endure, but because the virus never gets a chance to saturate the host population and fizzle out. New soldiers were being continually fed into the trenches to replace those lost to the ravages of the battlefield.

The virus has gained a foothold, and now has time, and bodies, to burn through as it mutates and eventually gets more and more effective in its ability to propagate from one soldier to the next. The closer it gets to efficient airborne transmission, the more lethal it can afford to be. But genetic mutations that favor transmission are a lot more beneficial to a virus than mutations that kill its host, and in this case the virus was able to find its way out of the trenches and spread around the entire world before turning into the superbug that began dropping people in their tracks in the fall of 1918.

This interesting, and tidy, little "theory" for what might have happened in 1918 could easily be dismissed as yet another academic packaging of historical events if not for the fact, as pointed out by Michael Greger in his book Bird Flu: A Virus Of Our Own Hatching, that it mirrors almost perfectly what we are seeing happen in outbreaks of bird flu in overcrowded chicken "broiler houses" in recent years.

Instead of millions of immune-compromised soldiers packed into earthen bunkers we have millions of immune-compromised chickens packed into the poultry mega-farms that service our insatiable and ever-growing appetite for chicken flesh. Birds that ranged free for millions of years, and which could not have sustained accidental influenza infections from ducks, are now packed beak to beak and able to transmit an ill-adapted virus through the population until it picks up a mutation that turns it into a killer.

This is actually to be expected. The influenza virus has had millions of years to fine-tune itself to the ecology of ducks and other water fowl. When a strain like H5N1 accidentally makes its way into a foreign host like a chicken, it is only a matter of time before it makes a serious change for the worse and begins wiping out its host in large numbers.

This is what happened in Hong Kong in 1997. If it had stopped with chickens that would have been one thing, but it managed to make its way into a three-year-old boy who later died of his respiratory infection, and that sent shock waves first through the epidemiological community, and then throughout the world.

When it was first determined that an H5 version of influenza had been detected in the boy it was suspected to have been a mistake, a result of some kind of laboratory contamination. For an H5-type virus to reproduce inside a human host - well, that just did not seem possible to the first scientists who got a look at this strange newcomer from the microbial world. Now we know better.

The "bird flu" as it is called, kills about **6 out of 10** people who are unlucky enough to come down with it. It is just as lethal as the Ebola virus which takes human lives in Africa by liquifying the internal organs and causing patients to bleed out through every orifice. In fact, bird flu is often called "chicken ebola" by those who witness its effects because of the way it is said to turn the insides of chickens into "Bloody Jell-O". By all accounts it treats its human hosts in much the same way.

The only good news in the bird flu story is that the virus has not yet managed to figure out how to spread efficiently when it hits the human host line. Transmission appears to occur only through direct contact - by touching an infected bird or person, and then ferrying the virus into the nose, eyes, or mouth. The worry, of course, is that one day H5N1 will find a way to adapt, become airborne, and go from being a nasty virus that can infect and kill millions of chickens, to one that can spread through *billions* of humans and do who knows how much damage.

Today research on the H5N1 strain is done in Biosafety Level 3-enhanced laboratories like the one shown below, and stories about it run in national

publications whenever a new cluster of human cases emerges.

For all of the reasons stated above, whenever *any* form of pandemic influenza is seen in humans, we get very worried. The less familiar the virus appears to us, the more uncertain we become about what it might do next. When the H1N1 swine flu virus of April 2009 began spreading around the world, people took notice. We had not seen this pattern of behavior from an H1N1 influenza strain since 1918. But then nothing really bad happened even as it continued, and still continues, to spread globally.

But what happens as it makes its way into the "broiler" populations of the world? The crowded slums of India, and the immuno-compromised nations of Africa which are already struggling with widespread HIV infections? These are places where H1N1 can easily gain a foothold and fester.

Where Did The 1918 Strain Come From?

Did the pandemic that swept the world in 1918 really enter the human population for the first time in Europe, or did it start from some other place?

It is unlikely that the true answer will ever be known, but if you do a little research you will discover that most historians believe the Spanish flu actually originated in the United States sometime in the beginning of 1918. In March of that year, *The Brainerd Daily Dispatch* published an account of an unusal

event that seemed to result shortly thereafter in the deaths of forty eight soldiers.

It took place at Fort Riley in Kansas, where soldiers had been burning tons of manure, when: *"a gale kicked up. A choking dust storm swept over the land... a stinging, stinking yellow haze. The sun went dead black in Kansas."* Two days later the first soldiers reported feeling sick, and soon pneumonia would be listed as the cause of the deaths to follow.

Many people believe that Fort Riley (then Camp Funston) was the initiating event, and that it was soldiers leaving the camp for Europe who took the seeds of the pandemic with them as they filed onto the battlefields of France. But at least one researcher seems to be able to point to an earlier period of infection that seems likely to have been the cause of the Fort Riley infections.

John M. Barry, the author of *The Great Influenza: The Epic Story Of The Deadliest Plague In History*, wrote in 2004 about his research into the initiating event. His account of his findings is provided as the resource file *resouce-1918-Pandemic-Origin.pdf*

Barry believes that the place from which the pandemic began was isolated and sparsely populated Haskell County, not 300 miles west of Fort Riley.

It was in late January of 1918 that the local medical practitioner, a Dr. Loring Miner, found himself faced with an epidemic of influenza of a type that he had never seen before. Despite the fact that most members of this hardy farming community were extraordinarily healthy, many of them were being struck down by this new flu. People began to die - and then, suddenly, it was gone.

Loring considered the episode so alarming that he wrote it up in *Public Health Reports*, which later became *Morbidity and Mortality Weekly Report*. It

appears to be the first record of the emergence of the virus responsible for the great pandemic which would explode upon the world nine months later.

What The Mathematical Models Say

If the epidemiologists truly believe that the emergence of a highly-virulent influenza virus in the next decade is not only possible, but very likely, then you would expect them to be doing their best not only to ward it off, but also to quantify the risks involved.

Not every piece of scientific work makes its way out into the world for inspection by the public, but the results of some attempts to mathematically model an influenza pandemic event are known, and are instructive.

In 2005 the CDC published the results of a flu pandemic model that it had developed based on the premise that the next pandemic might be avian in origin (of H5N1 type) and exhibit a mortality rate of about three times that of the Hong Kong pandemic of 1968. At the time, several outbreaks of bird flu in Asian countries had health officials and epidemiologists brainstorming the possibilities. One of the outcomes was *FluAid 2.0*, an improved pandemic projection model of expected fatalities and hospitalizations in the event of a "relatively mild" bird flu pandemic.

The model predicted a United States death toll of over half a million, another two million hospitalizations, and more than sixty-five million infected individuals. Of course, models like the one developed by the CDC depend on a number of assumed conditions, all of which are based on inspired guesswork. The actual numbers predicted by such models are highly variable. Tweak a couple of the variables and the number of estimated fatalities in the United States goes down to 200,000 or up to two million.

Tweak the model again by factoring in a "severe" bird flu pandemic, rather

than a mild one, and suddenly we are talking about perhaps 50 million deaths, just in the United States. The problem with numbers like this is that they are just too big to wrap your head around them.

Despite this, the important conclusion to draw here is that a *modern* pandemic model, put together by some very smart scientists who do not like to be caught publishing shoddy work, still predicts the possibility of a pandemic death toll that goes into the millions. The public is never told about these model predictions, but the results nonetheless trickle into the ears of policy makers and are not soon forgotten. In April 2005, then Senator Barack Obama introduced the AVIAN Act of 2005, which proposed a mandate to stockpile Tamiflu in preparation for the emergence of a future bird flu pandemic. So President Barack Obama is well aware of the potential damage that a pandemic could visit upon the world, even if he has been known to get in front of a podium and declare that "there is no reason for alarm".

Replikins - Did They Really Predict The 2009 Pandemic?

Pandemics have always been a little like large earthquakes in the manner in which they arrive. Usually, in hindsight, we can see that there were warning signs that an event of magnitude was about to happen, but we are always unable to interpret them correctly to benefit from the advance notice.

If one carefully studies the medical records from 1918 it is possible to see a killer lurking in the mild influenza season that marked the early months of that year. Some of the first victims of the Spanish Flu showed the characteristic pathology of the illness that would be seen later in the year. But there were just too few cases of this nature to warrant any real cause for alarm. The rumblings would go unnoticed right up until the time that the virus returned in full swing four months later to claim its place in history.

We still have no reliable way to forecast large scale earthquakes. But a small

biotechnology startup in Boston, Massachusetts, thinks it might be able to help us out with those pandemic predictions. In fact, it even claims it saw the 2009 H1N1 pandemic coming, or at least heard the rumblings in early 2008.

Replikins, as the company calls itself, was founded in 2006 by 81-year-old Dr. Samuel Bogoch. Years earlier, Bogoch and his wife Dr. Elenore Bogoch, had been researching the factors responsible for the rapid replication rate of tomato gemini virus, which causes massive losses in the tomato crop industry. What they found was a correlation between certain short protein sequences (or peptides) in the virus, and periods of heightened replication, or plant infectivity. This class of proteins, says the company, "contains high concentrations of amino acids lysine and histidine that have been previously determined to be related to rapid replication and virulence."

They called these special peptides **replikins**, and used the name to launch their company, which would focus on a more pressing matter of the day - influenza pandemics, and the then-pressing matter of a possible bird flu pandemic. They have since expanded their work to cover other rapidly replicating diseases like malaria, and HIV. Their business model is based upon developing and marketing forecasting tools and synthetic vaccines which are tailored to these diseases.

The sound bite used by the company to describe its technology to the press in 2006 was short and to the point: *"Replikins, Ltd. has discovered a group of virus peptides that predict whether a virus is rapidly replicating and whether it is likely to spread."*

Sam Bogoch claims that Replikins is also able to predict the degree of lethality of the viruses his company monitors. If he is correct, the work is of immense importance both to the science of epidemiology, and to matters of public health which relate to epidemics. But can the company's work be taken seriously?

Apparently, not many people seem to have thought so. While it has plodded on with its work, the company and its technology have largely been ignored. In fact, if it was not for a press release issued by Replikins in April of 2008, that went unnoticed, Sam Bogoch would still be trying to get the attention of pharmaceuticals with the big money needed to bring his company's synthetic vaccine technology into commercial application.

A year after the April 2008 press release by Replikins, people started to take notice of the company. ABC News in Boston ran a news story on it. So did CNN. Suddenly that once-passe press release was hot news. Here is what Replikins said in that 2008 publicity statement:

> ----------
> H1N1 Influenza Virus with Highest Replikin Count(TM) Since the 1918 Pandemic Identified in the U.S. and Austria
>
> Boston, MA (PRWeb) April 7, 2008 -- Replikins, Ltd. has found that the Replikin Count(TM) of the H1N1 strain of influenza virus has recently increased to 7.6 (plus/minus 1.4), its highest level since the 1918 H1N1 pandemic (p value less than 0.001). A rising Replikin Count of a particular influenza strain, indicating rapid replication of the virus, is an early warning which has been followed consistently by an outbreak of the specific strain. The current increase appears to be specific to H1N1; there was a concurrent 80% decline in the Replikin Count of H3N2, for instance.
>
> The current H1N1 appears to be rapidly replicating simultaneously in the U.S. and Austria. It may succeed H5N1 as the leading candidate for the next expected overdue pandemic. However, the same virus replikin structures detected by FluForecast(R) software in all three previous pandemics, namely 1918 H1N1, 1957 H2N2, and 1968 H3N2, as well as in H5N1, have not yet been

detected in the currently evolving H1N1.

There is evidence that many factors, including virus structure, host receptivity, and the environment, together with infectivity and rapid replication, need to converge for a pandemic to occur. For H5N1, the high human mortality rate, which peaked at over 80% in 2006-07 in Indonesia, as well as current low infectivity, both appear to limit H5N1's ability to produce a pandemic. Furthermore, the H5N1 rapid replication cycle which began in 1996 now appears to be over. The H5N1 virus produced less than 300 World Health Organization confirmed deaths over the past 10 years.

On the other hand, H1N1, with an estimated human mortality rate of only 2.5 to 10%, but with much higher infectivity, produced an estimated 50 million deaths in the 1918 pandemic. A number of countermeasures exist today which did not exist in 1918, however. Among these is Replikins' ability to manufacture synthetic vaccines based on current sequences, with a seven day production turnaround.

So why did I think it important enough to reproduce every word of that press release for you? Because if Replikins did accurately predict the emergence of H1N1 as the pandemic we now see in 2009, then maybe we should give whatever else they say about this subject just a little extra consideration.

If you are wondering about the term "Replikin Count", which litters the company's literature, it is just the number of those special viral peptides (per 100 amino acids) that they have identified in the viral protein sequences that have been published in the online PubMed database. The higher the count, say the Bogoch's, the more infectious the virus.

From the press release we can see that Replikins was doing its counting on

H1N1 seasonal flu strains and noticed that the last time the number of *infectivity Replikins* was this high was during the 1918 pandemic. Now, if it dawns on you that it is just a little strange to hear that the company is making their predictions on the basis of the genetic profile of the 1918 flu strain, which was long thought to be lost to history, do not worry - you will discover later in the book just how they were able to get their hands on that elusive genetic data (a fascinating story in itself).

Replikins' confidence for issuing their press statement was based on the strong correlation that the company had observed between increased Replikin Count and the emergence of a pandemic shortly thereafter. The software seemed to be able to find the correlation in the data for the pandemics of 1918, 1957, and 1968. It also found correlations in the data for the bird flu outbreaks seen in the years following 1997.

In fact, until 2008, virtually all of the company's focus had been on the H5N1 bird flu strain. So this press release was really a case of the company doing a double-take while looking at auxiliary data, and saying to itself "Hmmm. What have we here? We need to make a note of this in case it turns out to be something..."

Of course, that is exactly how it has turned out. One year after Replikins noted that H1N1 appeared to be priming itself for a pandemic, the first swine flu cases began appearing in Mexico, in April of 2009.

FluForecast is the name of the software application that was developed by Replikins to scan the latest viral protein sequences posted to public protein databases, and keep a track on the "replikin count" for both the *infectivity* and *lethality* peptides. The higher the count for each of these, the more infectious and lethal a virus is expected to be. It is upon these observation that their influenza forecasts are based - predictions of heightened infectivity and

lethality one to three years in advance of the main event.

If we look through the press releases that Replikins has issued, we find that "The Replikin Count in the 1918 H1N1 influenza pandemic was 7.0" which is comparable to the value of 7.6 quoted in the press release of April 2008 for H1N1 strains. Replikins contends that pandemics are marked by run up periods where the Replikin Count goes above 4.0 or so. This is the reason the company felt comfortable waving a red flag at H1N1.

Interestingly, this idea that a pandemic undergoes a period of simmering that is open to observation squares with the new theory of Smith and Webster, which says that novel genes may float for a time before acquiring their pandemic potential.

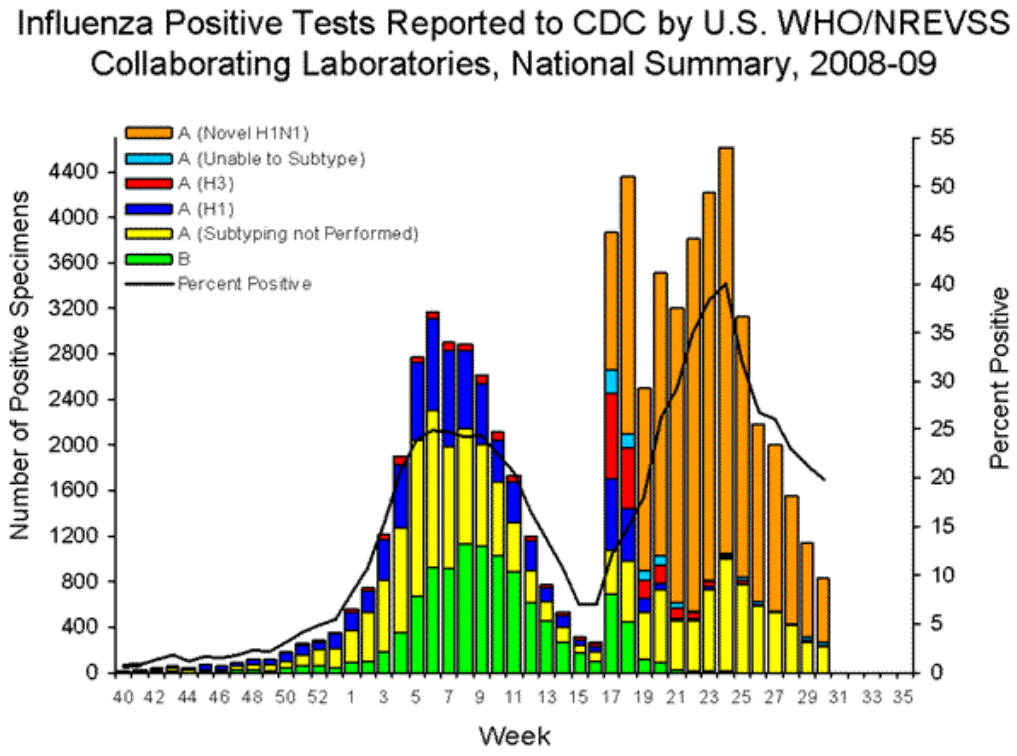
Now, does this mean we should all start panicking? Good question. The truth is, it is just too difficult to interpret the results that this company is alluding to in its press releases. I have read all of them, looking for clues, and the essence of the science seems to be that they are monitoring the Replikin Count in two distinct proteins in influenza - one controls the degree of infectivity, and the other the degree of lethality. In April of 2008 Replikins was able to point to the infectivity Replikin Count and note a marked increase. The forecast was for a *possible* emerging H1N1 pandemic.

As of June 2009 they have been able to report, in press release #29, an increase in the lethality Replikin Count. But the analysis was carried out on only 144 samples of the novel H1N1 swine flu virus. As we get closer to the fall of 2009 we are sure to see updates in this *prediction* of forthcoming higher mortality rates on the Replikins News page.

Replikins has also predicted that the rate of spread of the virus would not dampen out substantially over the summer months in the Northern

Hemisphere, but the virus would continue to spread unabated. This is in contrast to the expected behavior of flu viruses, and also seems to accord with the observed behavior (as of early August 2009). Despite the fact that the number of infections in North America does appear to have peaked for the first wave, the virus was still spreading well after the July 4th weekend.

You can check whether the number of cases is subsiding by visiting the FluView page maintained by the Centers for Disease Control and Prevention. A graph of the number of documented influenza A cases shows both the seasonal and the novel strains of influenza. The graph for the week ending August 1 appears below.



The first rise and fall of cases in this graph (devoid of the orange bars that come after week 16) shows the normal behavior of seasonal flu - both types A and B. The blue bars represent seasonal H1N1 influenza A. If not for the emergence of the new strain, the number of influenza A cases would probably have tapered off completely by week 23 (mid June).

The orange bars appearing after week 16 (the end of April) represent cases of the new H1N1 swine flu strain of influenza A. It was not until that week that health professionals knew what to look for. As you can see, at week 25, about a month before this graph was generated, the number of new H1N1 infections had just begun to taper off. To see an updated graph for the current week, click on the image above and scroll down the CDC FluView page.

The CDC itself has been quoted in news stories saying that they believe they are "seeing something different" with this new strain and the way in which it is continuing to spread even during the summer months. If, in fact, we do see continuing numbers of cases in North America in the coming summer months it will add credence to the prediction by Replikins of a more deadly version of the virus arising later in 2009 and 2010.

The bottom line is that here we have a *scientific prediction* that points towards a worsening swine flu situation, not one that is going to get better in the foreseeable future.

From Nuisance To Killer Flu

I have read quite a few accounts of the threat of pandemic influenza over the years and I know that often the author has blunted their message by insisting that people need to get prepared immediately for what is coming because (a) it is going to be bad, and (b) it is just around the corner. The reality is that we cannot know that either of those things is true, and so far the second point has proved incorrect every time. Again, *so far*.

On the other hand, it surely does not pay to be the know-it-all who rolls his eyes at the expert's warnings of the brewing threat to the human population of a devastating bird flu or swine flu pandemic. If the expert proves to be correct, the know-it-all is going to be one of the least likely to survive the event.

Here is a little story about why you do not want to underestimate the threat of a new strain of influenza virus. In this case the incident involves an H5N1 strain that never left the chicken population into which it jumped from its (unknown) native host, the most about which we can say is that it was another species of bird. Keep in mind though, that what is true about this case, which deals with chickens, could easily be translated into a story about a completely new strain of flu (like the H1N1 strain of April 2009) that had crossed over into humans. This is the reason this story keeps the virologists up at night.

In April of 1983 birds began getting sick on the chicken farms of Pennsylvania. They were coming down with a case of bird flu. Nothing major at first, some of the chickens died, and the egg production slumped, but nobody got too alarmed about the event. The virus behaved like bird flu viruses normally do and infected the lungs and guts of the birds.

But six months later the situation changed dramatically and the chickens began dying in massive numbers. This time the virus went at every organ in the body of the chicken. The virus even assaulted the brains of the birds and their innards literally dissolved into a bloody mush. It was not long before the U.S. Department of Agriculture ordered the slaughter of all chickens on the three affected farms - all 20 million birds.

Dr. Robert Webster and his apprentice bird flu sleuth Yoshihiro Kawaoka were tasked to find out what had happened. How did the relatively mild nuisance strain transform itself into a supremely efficient killer, almost overnight? Kawaoka set about comparing the genes of the two strains and discovered something remarkable.

The only difference between the earlier and the later strains was for the gene that coded for the hemagglutinin protein used by the virus to penetrate the

cells of the chicken's lungs and other organs. Kawaoka was stunned to find that, of the 1700 letters that made up the genetic code for that particular protein, the only difference between the mild form of the virus, and the killer strain, was a single letter. The change of just *one* nucleic acid had set the fate of all those birds. It was as if a gigantic dark hand had passed over the Pennsylvania chickens and begun laying them down.

The news set Webster to thinking about the origin of the 1918 strain, which he already suspected may have been due to a reassortment in pigs, or humans, of an avian form of influenza - a bird flu that had escaped its confinement in birds and gone on to infect about a third of the human population, and kill upwards of 100 million. Webster had no proof at the time, but every time he thought about what had happened in those Pennsylvania chicken houses it was as though the ghost of 1918 was trying to tell him something - trying to warn him of unfinished business.

It is a feeling of foreboding that has never left him. In the next section we will take a closer look at the ABCs of influenza and learn a little bit about why the emergence of new pandemic strains like the H1N1 strain of April 2009 so unnerves Webster and the other virologists who make it their business to know what influenza is doing.

Learn More

I hoped that you enjoyed reading the first part of Survive Pandemic Flu. If you would like to read the remaining 130 odd pages you can find the book at my Survive Pandemic Flu website. I have taken the remaining part of the table of contents for the complete book and added it to the next two pages so that you can see what has yet to be covered.

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Final Note

By now you have probably seen a hint of the enormously interesting biological side of influenza A, which in part was what attracted me to the study of this virus more than a decade ago. It is, of course, a virus that carries within it a propensity for devastating illness, and that is the other very good reason for not being complacent about the threat of influenza, and learning all you can about it, which I hope you will do with Survive Pandemic Flu.

Cheers,
Stephen Carter, Ph.D.

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