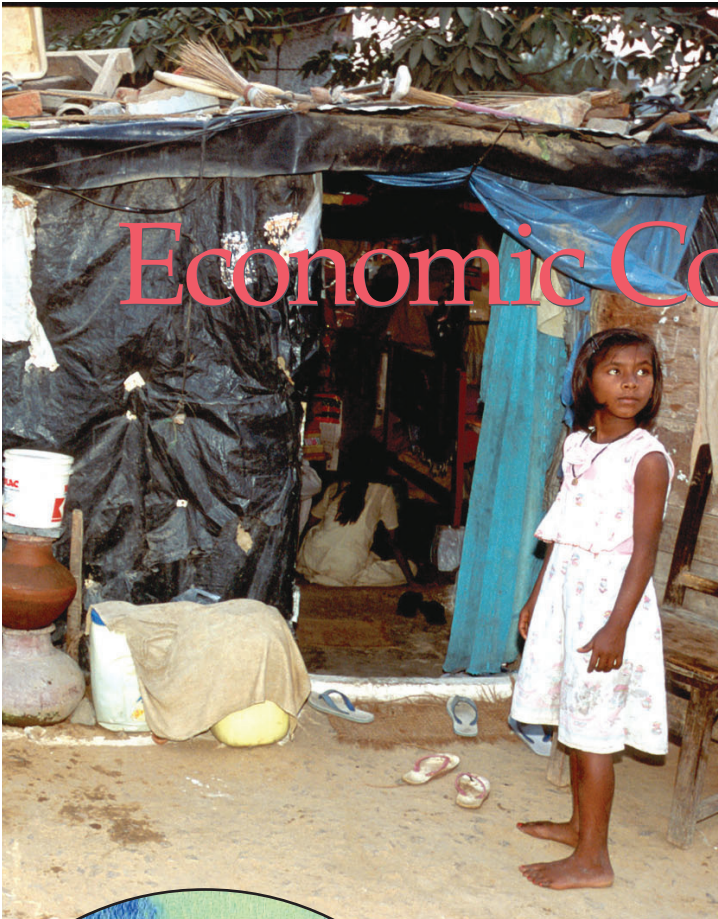


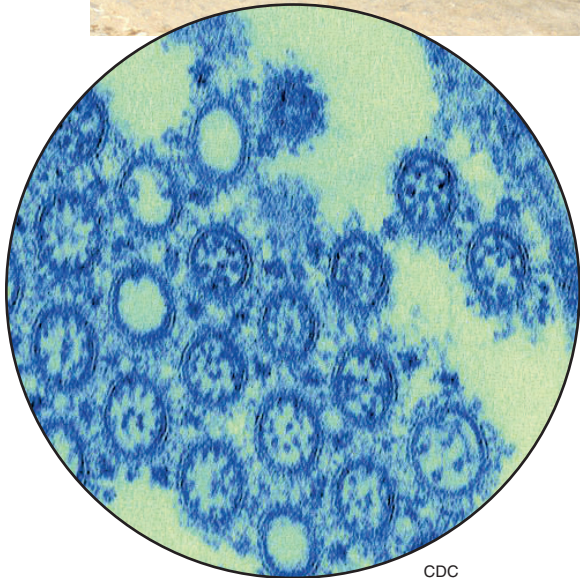
FLU DYNAMICS IN THE NOÖSPHERE

Economic Collapse Breeds Pandemics

by Christine Craig



WHO



CDC

Globalization and its destruction of the physical economy have created perfect conditions for pandemics to kill millions. Above an urban slum dwelling in India. At left is a transmission electron micrograph of the current H1N1 virus virions in a tissue sample.

To stop oncoming pandemics we must reverse the policies responsible for the current economic collapse.

Whether today's swine flu becomes apocalyptic in outcome is not so much a matter of how a virus works, but of the economic conditions that enable it to thrive. The poverty, hunger, disease, and wars raging in the wake of globalization and the collapse of physical economies worldwide create perfect conditions for a new and terrible flu pandemic. How successful this pandemic will be in killing millions depends on whether we reverse the policies sending civilization into a Dark Age, and mobilize our scientific and public health resources to use the anti-flu tools we have, and to develop new ones.

We can get a glimmer of the tragedy ahead by looking at history. World War I was the first pandemic war; it was a war of catastrophic proportions and import, and it hosted a pestilence of kindred nature. The Great War spawned the Great Pandemic.

More than 50,000 American troops died in combat in World War I, while more than 60,000 died of disease, primarily influenza and its complications. Almost 100 percent of those flu deaths were young and healthy adult males, who had been screened several times to be the best stock in the country.

The flu worked with the flower of youth on the battlefield. There were no old people or babies to kill in the filthy, rat-infested trenches, packed with one live soldier per 4 inches of trench length (the estimate for 1915 Western Front). The U.S. Army mobilized more than 4 million soldiers and transported 2 million of them across the Atlantic by boat to crush the "Hun," only to be immobilized on the Western Front by an unseen enemy travelling among them. Medical officers at the time estimated that almost 9 million soldier-days were lost to the influenza—and the United States only engaged in the fighting for six months before peace was declared on Nov. 11, 1918!¹

1. Carol R. Byerly, *Fever of War*, New York University Press, New York (2005). This book is one of the few that address the interaction between the Great War and the Great Influenza Pandemic of 1918.

This is an aspect of the natural history of the influenza A virus that you don't read about in the virology and medical journals, or even, I suspect, in epidemiology books and journals, although epidemiologists, more than most groups, should understand the import to disease spread of the policies and practices of war.

How It Began

One of the first recorded cases in 1918 of an outbreak of a highly infectious form of influenza came in the Spring of 1918, from an Army camp in Kansas. When the U.S. Army got to the front lines in France, a highly infectious form of influenza began spreading among the soldiers there as well. Then came word of a much more virulent form spreading in southern France and Spain.

By August of 1918, the influenza spreading widely in Europe was not only highly infectious, but it had turned uniformly deadly as well. Almost simultaneously, it manifested itself in many parts of the globe.

The influenza outbreak was seen at the time by military doctors as a phenomenon of the military mobilization. There were warnings and fears that it would get out into the civilian population, but the war effort took precedence. Now, almost 100 years later, although the mention of the 1918 flu causes fear, few people make the connection between the pestilence and the war.

But the 1918 flu was not forgotten. In January 1976, when the H1N1 swine flu showed up at Fort Dix, an army camp in New Jersey, it initiated an immune reaction in the Federal bureaucracy that continued to rage until the next Winter, and resulted in the mass production of a swine flu vaccine which was given to 40 million people, many months after it was clear that there was no swine flu spreading among humans anywhere in the world. The damage to the prestige of the Federal health authorities still persists today.

It was the specter of the 1918 virus's ravages, coupled with lack of knowledge about what drives pandemics, plus bureaucratic inertia, which led to the debacle. One Army recruit died of the flu after a 5-mile forced march while ill. Several others became acutely ill and were hospitalized. Probably hundreds were infected by the virus, but the flu never got out of that camp.

What was the difference between this incident and the first spread of the Spanish flu in the Kansas Army camp, full of recruits in the Spring of 1918? Some say it was a difference in the innate qualities of the viruses. But what would have happened if Fort Dix had been processing hundreds of recruits daily, pack-



Fresh young troops, the flower of youth, en route from New Zealand to the killing fields of World War I in Europe.



Today's public admonishments about the swine flu, as World War I's slogan, "coughs and sneezes spread diseases," ignore the conditions of poverty, crowding, and war that proliferate the spread of killer disease.

ing them like sardines, and sending them off to a war fought by tens of millions in close-quarter combat? Would that virus have gained the tools necessary to have equalled or

bested its ancestor, the 1918 Spanish Flu?²

Why did the 1918 flu kill so many young and healthy people? Was it because it held that potential uniquely within its genes? Or was it also because it was given such unique opportunities to do so, with (1) the economic and social devastation of four years of the most horrific and widespread war up to that time; (2) incessant close packing of millions of young men during the war mobilization and warfare; (3) all the sick soldiers crowded together on troop ships and headed home with their diseases to re-infect their countries all over again after Armistice was declared.

Virologists Taubenberger et al. marveled at the dynamics of the 1918 Spanish flu spread:

But 3 extensive pandemic waves of influenza within 1 year, occurring in rapid succession, with only the briefest

2. Justin Lessler, et al., 2007. "Transmissibility of swine flu at Fort Dix, 1976," *J. R. Soc. Interface*, Vol. 4, No. 15, pp. 755-762.



Photo by Harris & Ewing, courtesy of the Library of Congress

A flu ward at Walter Reed Hospital in Washington, D.C., during the Influenza Pandemic of 1918-1919, where beds are lined up, separated by sheets, on a porch.

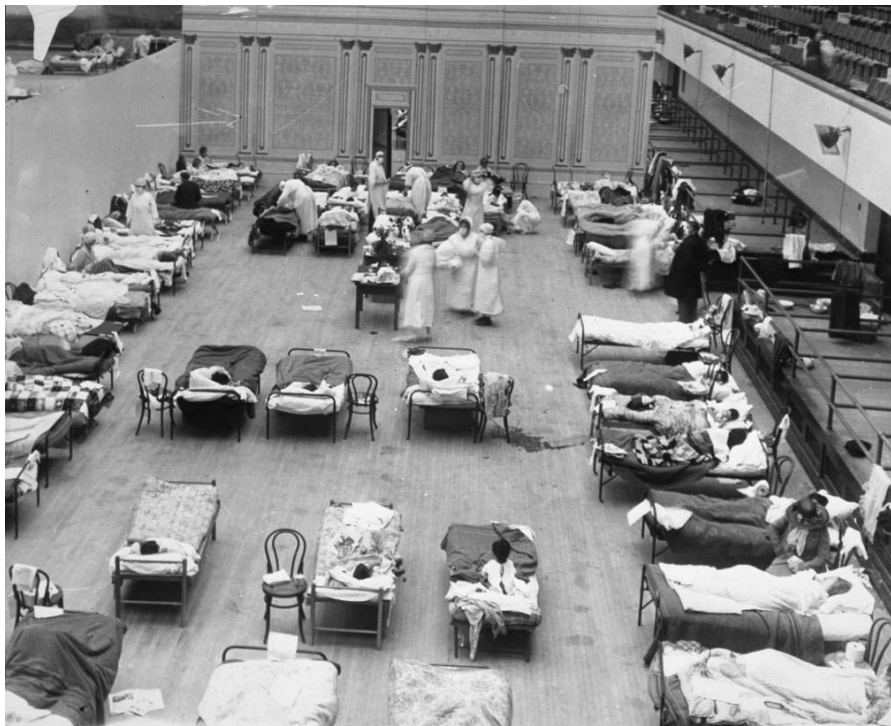


Photo by Edward A. "Doc" Rogers; from the Joseph R. Knowland collection, Oakland Public Library

The 1918 flu swept the country from coast to coast. Here an emergency hospital set up in the Oakland Municipal Auditorium in California, with volunteer nurses from the American Red Cross.

of quiescent intervals between them, was unprecedented. The occurrence, and to some extent the severity, of recurrent annual outbreaks, are driven by viral antigenic drift, with an antigenic variant virus emerging to become dominant approximately every 2 to 3 years. Without such drift, circulating human influenza viruses would presumably disappear once herd immunity had reached a critical threshold at which further virus spread was sufficiently limited. The timing and spacing of influenza epidemics in interpan- demic years have been subjects of speculation for decades. Factors believed to be respon- sible include partial herd immunity limiting virus spread in all but the most favorable circumstances, which include lower environmental tempera- tures and human nasal tempera- tures (beneficial to thermolabile viruses such as influenza), optimal humidity, increased crowding indoors, and imperfect ventilation due to closed windows and suboptimal airflow.

However, such factors cannot explain the 3 pandemic waves of 1918-1919, which occurred in the spring-summer, summer-fall, and winter (of the Northern Hemisphere), respec- tively. The first 2 waves occurred at a time of year normally unfavorable to influenza virus spread. The second wave caused simultaneous outbreaks in the Northern and Southern Hemi- spheres from September to November. Furthermore, the interwave periods were so brief as to be almost undetectable in some locales. Reconciling epidemiologically the steep drop in cases in the first and second waves with the sharp rises in cases of the second and third waves is difficult.

Assuming even transient postinfection immunity, how could susceptible persons be too few to

sustain transmission at 1 point, and yet enough to start a new explosive pandemic wave a few weeks later? Could the virus have mutated profoundly and almost simultaneously around the world, in the short periods between the successive waves? Acquiring viral drift sufficient to produce new influenza strains capable of escaping population immunity is believed to take years of global circulation, not weeks of local circulation. And having occurred, such mutated viruses normally take months to spread around the world.³

Obviously Taubenberger et al., although part of the Biosphere and Noösphere, know little about the interconnections between life, the Biosphere, and the Noösphere. Although these authors raised some pertinent questions, they were apparently not aware of the work of the great Russian biogeochemist Vladimir Vernadsky.

Vernadsky and the Noösphere

In 1918, Vernadsky was a mature scientist observing and pondering such ideas. He experienced not only the devastation of the Great War and the Great Influenza, but the many years of violence, unrest, famine, disease, and social upheaval of the Bolshevik Revolution as well.

In 1945, at the end of World War II, Vernadsky expressed his thoughts in his essay, "Some Words About the Noösphere":⁴

In the thick of life today, intense and complex as it is, a person practically forgets that he, and all of mankind, from which he is inseparable, are inseparably connected with the Biosphere—with that specific part of the planet, where they live. It is customary to talk about man as an individual who moves freely about our planet, and freely constructs his own history. Hitherto, neither historians, scientists in the humanities, nor, to a certain extent, even biologists, have consciously taken into account the laws of the nature of the Biosphere the envelope of Earth, which is the only place where life can exist. Man is elementally indivisible from the Biosphere. And this inseparability is only now beginning to become precisely clear to us. In reality, no living organism exists in a free state on Earth. All of these organisms are inseparably and



Vladimir I. Vernadsky (1863-1945) located man within the Biosphere, inseparable from its laws. Here, Vernadsky on a geological expedition.

continuously connected—first and foremost by feeding and breathing—with their material-energetic environment.

Let's look further at these inseparable connections.

Influenza A is the classic zoonosis. It doesn't need humans. It can survive and thrive quite well within its primary natural host and reservoir—aquatic birds. Within its natural habitat, the bird gut, it is spread by the fecal/oral route. Ducks and geese defecate the virus into the waters of lakes and ponds and other wetlands, where their neighbors, who feed on animals and plants of the same waters, pick up the virus and start the cycle over again. At certain times of the year, some lakes and ponds are bursting with flu viruses of many subtypes. All known flu subtypes can be isolated from bird habitats (though not all can be found at any one habitat). Young waterfowl are invariably infected before they migrate. Few, if any, waterfowl show any symptoms of illness. The host and disease seem

totally adapted to each other.⁵

But viruses are by nature opportunists, and the flu virus, by nature, is one of the more opportunistic of viruses. If a flu virus lodges in a human passing through its domain, and finds the human terrain conducive to replication and spread, it will readily add that human being to its list of travelling companions, along with dogs, horses, pigs, seals, whales, ferrets, and chickens, just to name a few.

The possibilities for such encounters are endless: duck hunters gathered in a duck blind on a duck-filled lake; soldiers tramping through a wetland; farmers growing ducks or geese for eggs and food; farmers using duck feces to fertilize fish ponds; children plucking goose feathers from geese just shot by hunters.

Similarly, pigs and chickens living openly on farms, as was the case in the United States at the beginning of the 20th Century, could be easily infected by sharing ponds with domestic or wild waterfowl.

It has always been popular to attribute all new flu outbreaks ultimately to some mixing bowl in Asia, because Asia was the hypothetical origin of the 1957, 1968, and 1977 pandemics. The first solid information about the 1918 virus, however, came from an outbreak at Camp Funston, Kansas, in March of 1918. Could the proximate progenitor of the 1918 pandemic virus have been a locally grown virus?

3. Jeffery K. Taubenberger and David M. Morens, 2006. "1918 Influenza: The Mother of All Pandemics," *Emerging Infectious Diseases*, Vol. 12, No. 1, pp. 15-22.

4. V.I. Vernadsky, 2005. "Some Words About the Noösphere," *21st Century* (Spring), pp. 16-21.

5. Robert G. Webster, et al., 1992. "Evolution and Ecology of Influenza A Viruses," *Microbiol. Rev.*, pp. 152-179.

According to the Kansas State Historical Society, in 1885 there were 3 million pigs in Kansas—two for every human being in the state. Many farms of that era contained ponds or reservoirs on the premises, accessible in many cases by pigs and domestic fowl, which could have hosted local species of waterfowl as well as the multitude of migrating ducks and geese in Spring and Fall. At the beginning of the Fall migration, at least 20 percent of ducks are still shedding influenza virus, and could easily infect the pond water of farms along the migration route. In fact, in lakes and ponds where such ducks spend their summers, live virus can be isolated from unconcentrated water.

Pigs have been shown over the years to be able both to give their flus to humans and to get them from humans. They have surface receptors on the cells of their upper respiratory tract, which recognize and bind both avian- and human-adapted flu viruses. It is not much of a stretch to imagine among the 4 million soldiers recruited and drafted, that many were rural farm boys who were in close contact with pigs, just days before reaching their training camps. We know that something like this must have happened at Fort Dix in 1976.

A Convergence of the Right Circumstances

We don't know how long influenza A has been with humans in a consistent and global way, nor do we know how many times bird flu has independently jumped to humans in our long history, either directly, or indirectly, through the mediation of another species like the pig or chicken. Probably this has happened countless times locally, without fixing itself among humans. But, once in a while, when the right circumstances have converged at the right place and time, the flu has made the transition on a more permanent and widespread basis. It seems that 1918 was such a time.

Fortunately, the state of the medical and biological sciences had advanced enough by 1918 to recognize the nature of the disease, as well as understand some of the measures which should be taken to blunt its assault. Unfortunately, the Spanish Flu was spawned during one of the most savage wars in recorded history, where its primary victims—the young—were gathered in unprecedented numbers under the worst possible sanitary, nutritional, and crowding conditions.

The migrations of the soldiers from countless countries, by ship, train, or on foot, carried the virus to all parts of the globe, where it tore through the civilian population with similar force and speed. Recent tallies of the carnage caused by the Spanish Flu virus estimate that perhaps 30 percent of humanity was infected, and more than 50 million people may have died from it. The death toll from the influenza dwarfed the official death toll from World War I—8.5 million.

Modern genetic analysis of samples of the Spanish Flu taken



Army Corps of Engineers

Aquatic birds are a primary reservoir for viruses to spread to other animals and human beings. These waterfowl inhabit a wetland in Kansas.

from flu victims in 1918 by Taubenberger et al.⁶ have given strong evidence that the 1918 Spanish Flu was a fully avian-type flu in terms of amino acids coded for by the genes. It has recently been hypothesized that it jumped directly into humans from birds. Humans then spread the disease, not only throughout humanity, but into pigs as well, where it has become enscathed as the classic H1N1 swine flu.

More recently, Webster et al., following on years of phylogenetic analysis of flu family trees, countered that the progenitor viruses to the 1918 flu had been co-circulating for years in human and probably swine populations, and that several reassortment events were necessary to finally produce the killer of Fall 1918.⁷

There is obviously not enough evidence to prove either claim, although the Webster et al. hypothesis is a better fit with the historical evidence. However, trying to build a theory of the evolution of the 1918 virus from a few “fossils” and a lot of speculation has some resemblance to building a theory of man's evolution based on a study of the “fossils” known as Piltown Man.

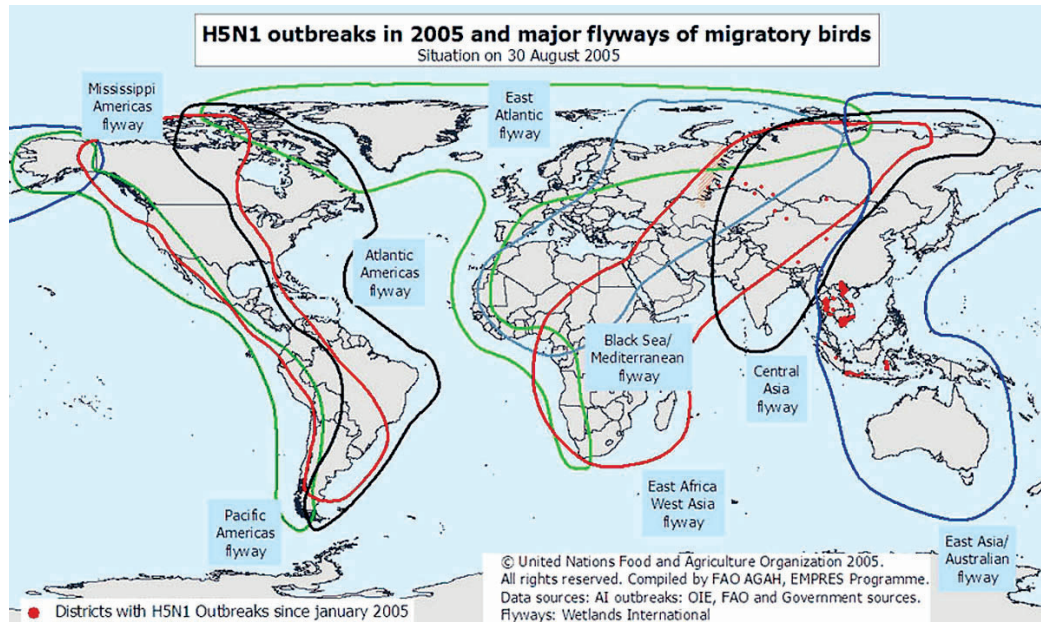
In any case, the 1918 flu started out as a mild but very contagious disease, and within six months had become a monster. Somewhere on the Western Front a change occurred in the virus that sealed the fate of humanity. Thinking back on the hypothesis of an Asian origin for all pandemic flu, I was intrigued to read in a soldier's account of the 1918 influenza in Europe, that among the ambulance drivers used to ferry the sick and wounded back to hospitals were many whom he called

6. Jeffery K. Taubenberger and David M. Morens, 2006. “1918 Influenza: The Mother of All Pandemics,” *Emerging Infectious Diseases*, Vol. 12, No. 1, pp. 15-22.

7. Gavin J.D. Smith et al., 2009. “Dating the emergence of pandemic influenza viruses,” *PNAS*, Vol. 106, No. 28, pp. 11709-11712.

**Figure 1
GLOBAL
WATERFOWL
FLYWAYS AND
H5N1**

Waterfowl are efficient conveyors of viruses around the world.



Source: FAO

“chinks.” I had never really thought about the colonial nature of World War I until I read that.

It turns out that many thousands of Vietnamese and North Africans served on the Western Front in support and fighting positions for France. Apparently, many thousands of Chinese were contracted out to the British by China, and colonial Africans participated as well, in support work, such as digging the endless trenches.

Here we have a potent situation of East meets West on the battlefields of France. Could some unique reassortments or recombinations have taken place in France between Asian and North American flu strains that Summer in 1918? Ambulance drivers would certainly get more than their fair share of exposure to the flu viruses going around. And while we are speculating about origins, we can hardly forget that the main residents of the trenches were the rats. There were many millions of rats living in the trenches with the humans. The rats fed on anything they could sink a tooth into, including rotting casualties of war. They grew as large as cats. And then there were the lice and fleas.

What happens when a flu with novel antigenicity gains the ability to effectively infect human tissues, especially the tissues of the lower respiratory tract? We can read the horrific medical accounts from 1918 to find out.⁸ And we can look at the much more recent medical history of the H5N1 avian flu virus, which periodically sweeps through Asian poultry farms, concomitantly causing occasional human disease—a virus causing human symptoms almost identical to those of the Spanish Flu, but with a kill rate of about 50 percent rather than the 2-5 percent of the version of the Spanish Flu which swept the world in 1918.

8. <http://web.uct.ac.za/depts/mmi/jmoodie/influen2.html#Pandemic%20influenza>

The difference between the H1N1 Spanish Flu pandemic strain, and the H5N1 avian flu pandemic strain, is that the H1N1 pandemic strain somewhere, somehow, gained the genetic tools to spread easily among human beings—something the H5N1 strain has not yet attained. Then, apparently, the H1N1 flu gained the ability to strongly infect the lower respiratory tract, specifically the lungs. The H5N1, however, remains a bird flu pandemic, and more specifically, a poultry pandemic, where it is a very efficient spreader and killer.⁹

The Sepsis Danger

Sepsis is the medical term given to a systemic inflammatory response by the innate immune system of the body to a microbial infection, whether bacterial, viral, or other. Sepsis can lead to rapid multi-organ failure and death in severe cases, and often the patient requires a ventilator for breathing assistance. Lungs, kidneys, brains, and livers all can be severely damaged through actions by cells and chemicals of the innate immune system. It is not so much the virus directly causing fatal harm to the tissues, as it is the immune system’s exuberant response in trying to eliminate the virus from the tissues, which causes the damage. It has been suggested that sepsis, in the form of cytokine storm, was the mechanism of many of the excess deaths in the 1918 virus—specifically among those who succumbed quickly to primary viral pneumonia. These included many of the young

9. The descriptions of the symptoms of many early victims of the main wave of the Spanish Flu in 1918 are eerily similar to descriptions of symptoms in chickens infected with the highly pathogenic H5N1 bird flu when it first appeared in Hong Kong’s live markets in 1997:

“One moment, birds happily pecked their grain, he recalls, the next, they fell sideways in slow motion, gasping for breath with blood slowly oozing from their guts. On necropsy, pathologists found that the virus had reduced the bird’s internal organs to a bloody pulp. We were looking at a chicken Ebola, Shortridge recalls. I had never seen anything like it.”

In Michael Greger, “Bird Flu: A Virus of Our Own Hatching.”

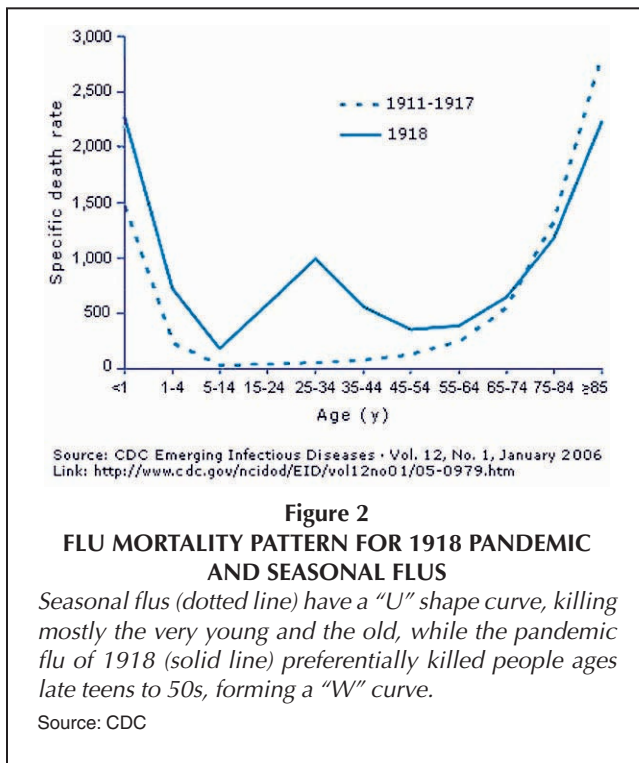


Figure 2
FLU MORTALITY PATTERN FOR 1918 PANDEMIC AND SEASONAL FLU

Seasonal flu (dotted line) have a “U” shape curve, killing mostly the very young and the old, while the pandemic flu of 1918 (solid line) preferentially killed people ages late teens to 50s, forming a “W” curve.

Source: CDC

and previously healthy adults who proved so susceptible to the virus. This same mechanism seems to play a role in the high fatality rate in chickens and human beings infected with the H5N1 highly pathogenic bird flu.

It is this sepsis, causing fulminant and fatal primary pneumonia in the young and healthy, that strikes fear into the heart of flu-watchers every time a flu acts a little differently. The seasonal flu is “comfortable.” It kills only the old, the extremely young, and those whose underlying diseases make them vulnerable.

Almost 90 percent of seasonal flu deaths at the beginning of the 21st Century have been in the very old and frail. Similarly, at the beginning of the 20th Century, the infants and elderly shared equally in flu deaths, giving a “U” shape to flu mortality charts.

But in the 1918 Spanish Flu, a terrible thing happened. The chart changed into a “W,” with a death spike peaking around the age of 25. The very young and the very old still died in similar numbers, but the young adults, who barely registered on the chart during the seasonal flu, died in very large numbers, accounting for most of the excess deaths over those for seasonal flu. Because similar, but much smaller spikes of fatal infection in young people showed up in the other main flu

pandemics of the 20th Century—1957 and 1968—the ideas of the 1918 Spanish Flu, of flu pandemics in general, and of excess mortality in the young and healthy, have fused into one idea in the population.

What is worse, that idea takes us back to the fearful times we have barely emerged from even now—a time when mankind lay helpless before the onslaught of infectious disease, where smallpox, polio, tuberculosis, bubonic plague, cholera, typhoid, typhus, yellow fever, puerperal fever, and malaria stalked young people in the United States just as most of them still do in many underdeveloped countries. Barely under the surface of rational Western man hunkers an inchoate horror in the face of microbial disease, born of millennia of ignorance and helplessness to stop the periodic dying, and more dying, particularly among the cherished young, whose lives were cut short in their prime productive years.

The Fear Today

Looking out at the new AH1N1 swine flu through the lens of this shared psychology, the 2009 swine flu seems very fearsome. It touches that sense of horror just below the surface, as it strikes in school classrooms and sickens the young people preferentially, killing a few. It travels the airlines and spreads almost visibly (given the plethora of internet maps and charts tracking the disease) from country to country, preferentially seeking out the young people to infect. It is clearly not the 1918 Spanish Flu yet, but is it the precursor to a flu of that viciousness and virulence—a flu that could pop up at any time in its killer mode and mow down the youth like before?

Are we still helpless primitives in the maw of the marauding

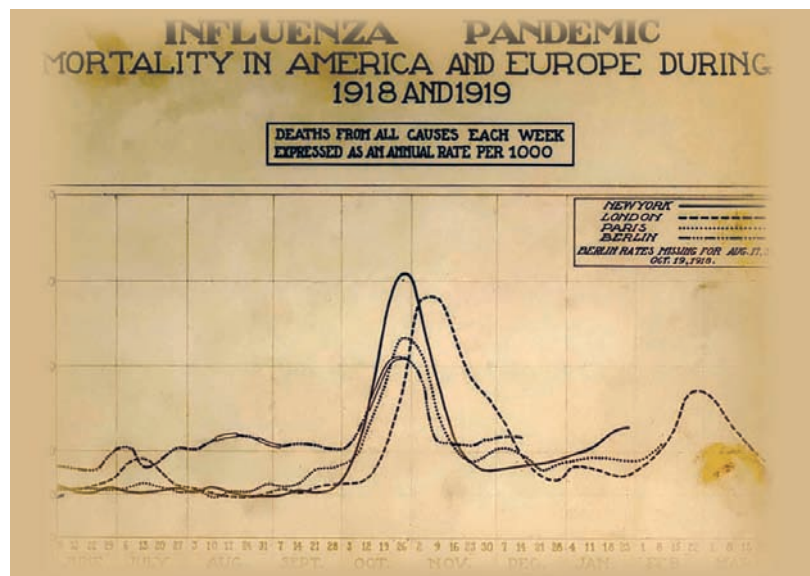


Figure 3
FLU MORTALITY IN 1918 PANDEMIC

The 1918 flu pandemic killed more than 1 million people in the United States, and about 50 million worldwide. This illustration charts the deaths in major cities, 1918-1919.

Figure 4
HOW THE FLU VIRUS INVADES THE CELL

1. The flu virus uses special molecules (the glycoproteins hemagglutinin and neuraminidase) projecting from its surface envelope which have been derived from host cell material, to bind to complementary receptor molecules on the host cell membrane.

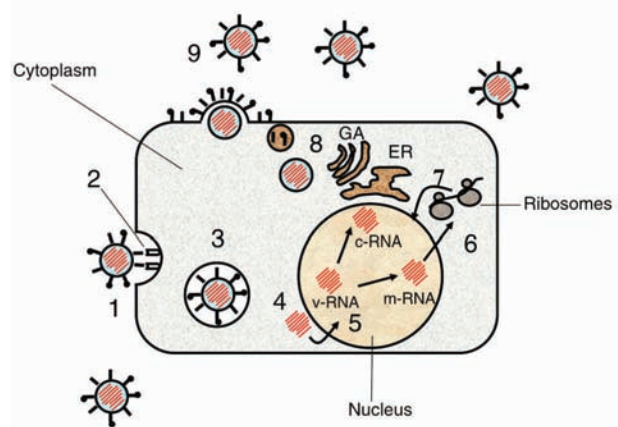
2. This handshake of recognition at the cell membrane sets into motion a process called endocytosis, the same which cells use to bring external substances into the cell for nutritional or other purposes. The cell membrane surrounds the virion and fuses around it.

3. Now the virus is within the cell, in a spherical vesicle surrounded by a membrane. But it does not yet have access to the rest of the cell.

4. Next, the aqueous environment within the vesicle acidifies, which sets into motion a cascade of events, resulting in the release (decoating) of the virus RNA strands and related proteins into the cytoplasm of the cell. The virus now has free access to hijack the cellular machinery required for its replication and the eventual release of its progeny from the cell.

5. The viral RNA (v-RNA) is transported into the nucleus, along with the four viral proteins essential for processing of the viral RNA. Here are found the host cell chromosomes and the required apparatus for DNA and RNA synthesis and processing. Using its own nuclear enzymes and those of the host cell, the viral RNA is transcribed into messenger RNA (m-RNA, the code for protein translation) and complementary RNA (c-RNA). The c-RNA will eventually produce all the copies of the eight viral RNA strands necessary for the hundreds of infectious progeny which a single infected cell can produce.

6. Meanwhile, in the cell cytoplasm (the aqueous milieu outside the nucleus), the cell's protein-manufacturing equipment, its ribosomes, have been conscripted to pro-



Christine Craig

duce the protein products necessary for the assembly of new virions.

7. The protein products destined for packaging within the viral envelope are now transported into the nucleus, where they are assembled in the proper proportions and configuration with a complete set of eight v-RNA strands. Then they are exported into the cytoplasm and migrate toward the inside of the outer cell membrane.

8. While this is occurring, two viral components take a different route. Hemagglutinin and neuraminidase, the two glycoproteins which will eventually stud the outside of the viral envelope, are transported to the outer cell membrane via the endoplasmic reticulum (ER) to the Golgi apparatus (GA) route.

9. The viral glycoproteins are duly incorporated into the cell membrane, whereupon the virions exit the cell in a reverse of the process by which they entered—exocytosis.

—Christine Craig

pestilence? Not unless we choose to be.

The 1918 Spanish flu was a terrible pandemic, brought on by the economic, environmental, and cultural devastation of a worldwide war of imperial powers. But even before the war broke out, the seasonal flu was a terrible killer. Before the United States went to war in 1918, its average death rate for influenza/pneumonia over the preceding three years was around 650,000, compared with 1,000,000 for the 1918 pandemic year. That is, 650,000 deaths in, say, 1915 (with a population of 100 million) compared with a mere 35,000 deaths for seasonal flu in recent years in the United States (with a population of 350 million). That is, .65 percent versus .01 percent deaths per total population.

Obviously, something has changed. We don't die of the flu as we used to. And we don't get pandemics as we used to. The 1957 and 1968 pandemics were pale imitations of the Mother of All Pandemics. And the seasonal flus of recent eras were pale imitations of the seasonal flus in the years before 1918.

Of course, flu commentators, including some who should know better, say that a 1918-like pandemic is a once-in-a-

century phenomenon, while the other pandemics of the 20th Century are once-in-30-years phenomena. They can't be citing statistics, because there are too few instances to make statistics useful. They must be consulting the Delphic Oracle. In any case, such predictions put flu snugly in the category of unstoppable acts of nature.

While Vladimir Vernadsky is clear about the inescapable connections between man and the Biosphere, he is also clear that the Noösphere, the phase-space of man's reason and its products, is a powerful and growing force, able to change the aspect of the Biosphere in remarkably short time periods, compared with even life's powers of change:

Mankind taken as a whole is becoming a mighty geological force. There arises the problem of the reconstruction of the Biosphere in the interests of freely thinking humanity as a single totality. This new state of the Biosphere, which we approach without our noticing, is the Noösphere. The Noösphere is a new geological phenomenon on our planet. In it, for the first time, man

becomes a large-scale geological force. He can, and must, rebuild the province of his life by his work and thought, rebuild it radically in comparison with the past. Wider and wider creative possibilities open before him. It may be that the generation of our grandchildren will approach their blossoming.¹⁰

How Viruses Work

A virus can be likened to an idea. The British zoologist Sir Peter Medawar once commented that a virus was “simply a piece bad news wrapped up in protein.” Someone throws a stone through your living room window. It has a note attached. You can’t help yourself; you open it up, and it reads: “If you are reading this, you will be dead in 24 hours. Have a nice day.”

Like an idea, if a virus lodges in fertile ground, much work can be produced out of something too small to even detect with the senses. If it lodges in sterile ground, no work can be accomplished, although the idea can be passively transmitted to another without the middleman being affected by the idea. That would be the case if a message were sent by semaphore or telegraph: “Enemy aircraft 10 minutes away.” The person receiving and retransmitting the code might not understand the code, and so could be caught unaware by the attack, whereas the person to whom the message was sent might understand and take defensive action.

Think of the influenza virus as an idea in the realm of life. When implanted in a receptive organism, it can cause the organism to perform work and produce things it otherwise would not be able to produce. It can cause the organism to amplify the successful viral idea manifold, then spread it to everyone it meets. As is common with ideas, in the process of reproduction and reassembly of the viral idea within the host, the product is constantly changing slightly. And as with many ideas, the products of the viral idea within the organism can be very harmful or fatal at times.

So, we have three interwoven qualities of this viral idea: *infectivity*: the ability to implant itself and perform work once within a receptive host; *contagion*: the ability to transmit the idea from one host to another. And to these we must add a quality which we will call *virulence* or *pathogenicity*: the ability to produce morbidity and mortality in the host organism while performing work.

Viruses are very simple ideas, for the most part. They cannot build the factory necessary to carry out the processes they need.



CDC

The gold standard public health response to killer viral diseases is vaccination, priming the human or animal host with disease antigens to promote an adaptive immune system response. Here are smallpox vaccinations, which in the past decades have resulted in stopping smallpox worldwide.



WHO

They rely on the infrastructure already present in the host cells. They simply alter the line of products that the cell produces, using their own code to hijack the assembly line.

The influenza A virus, for instance, contains 8 separate strands of RNA (ribonucleic acid) coding for 11 genetic products necessary for the efficient production and release of many new viral particles by the infected host cell. These products, when transcribed and translated by the cell, are able to direct the machinery of the animal cell to produce many copies of the viral genes and proteins, which are assembled, then packaged in the host membrane as the viral particles move out of the cell.

While in the cell, the original viral RNA strands are copied many times for repackaging in the new viral particles. This process is extremely prone to errors, leading to mutations in each generation.

Furthermore, if more than one flu virus type is within the body in the same tissues, the RNA segments will be assembled without regard to which flu virus the genes came from. This is termed *reassortment*, and is a primary way that flus with new qualities of infectivity, virulence, and contagion are generated. That is how one gets RNA segments from bird viruses, swine

10. V.I. Vernadsky, 2005. “Some Words About the Noösphere,” *21st Century* (Spring), pp. 16-21.

viruses, and human viruses into one new virus.

A third and very potent form of genetic change can occur among the viral RNA strands as well. Two homologous RNA strands with differing information can undergo recombination, whereby they effectively switch homologous sections of the RNA strand. Non-homologous switching could also occur, but would be much less likely to lead to a viable viral particle. But that is all right. In such a large factory, with so much product, a few of these new ideas will be viable ones, leading to potent new flu types to plague humanity.

The host response to a flu attack is to mount an immune counter-offensive to drive out the invader. If the host recognizes the flu type, it sends out cells that produce antibodies to antigens remembered from the previous attack. The flu attack is driven off rapidly with little host damage.

However, if it is a sufficiently new virus antigenically, the body sends out raw and untrained but enthusiastic recruits called the innate immune system. These agents attack the virally infected tissues much like the troops fighting in World War I. But this battlefield is not the farmers' fields and lush vineyards of France; this devastated battlefield is your lungs. After the initial bloody battle has already severely damaged your lungs, the adaptive immune system kicks in, producing specific antibodies to the viral invader. If you survive, the next time you meet the same flu, the adaptive immune system will drive the invader off with little damage.

The Public Health Response: Vaccination

The gold-standard public health response to viral diseases which pose a widespread threat to humans and domestic animals is vaccination, to prime the host body with disease antigens so that the adaptive immune system can kick in at the least sign of the flu virus. Vaccines have proven themselves extremely valuable for two centuries in saving the lives of young people from virulent contagious viruses such as smallpox, and more recently, polio, measles, chickenpox, and the like, which act as if they were unstoppable acts of nature. For smallpox, vaccination combined with other public health measures like surveillance and quarantine, drove the virus to extinction in the world-at-large (it still exists in a few laboratories). With a concerted effort, polio might also be eliminated by such means.

But Influenza A is a different story. Influenza A is not a disease of humans only. It is, as mentioned above, a zoonosis originating in birds. It has also shown itself to be shared easily among man and the domestic pig, and there could be other vectors as well. Further, it changes antigenically each season as new mutations or reassortments find more fertile ground for infection. Such diseases cannot be stamped out by vaccination, at least in the form we now know it.

For influenza, vaccination only blunts the effect of the virus on the population. Seasonal flu kills around 35,000 people (90 percent of them old people) every year in the United States, despite a vaccination rate in old people of more than 60 percent. Until the 2008-2009 flu season, national flu policy did not encourage vaccination among children (ages 5-18) unless there were underlying health problems. During the 2007-2008 flu season, fewer than 25 percent of young people were vaccinated.

From a rational public health standpoint, it might be better if the young were required to be mass vaccinated, despite the fact that few die from seasonal flu. Recent research has given evidence that the death rate of the very elderly is not strongly affected by their vaccination against the seasonal flu, because they often mount such a feeble immune response to flu vaccine challenge.¹¹ Society might be better served by requiring vaccination among the main spreaders of the flu—children in school and daycare—even though they are the least likely to die from it. We can see by the behavior of the novel H1N1 flu now in circulation, that with an antigenically new flu type, the group with the most cases also becomes the group with the greatest morbidity and mortality, especially because many of the young these days may never have had exposure to any flu virus in their young lives.

Because vaccination as now practiced only blunts the seasonal flu attack, antiviral drugs such as oseltamivir, zanamivir, amantadine, and rimantadine have been developed to prevent or treat the flu among those still susceptible each season. These drugs can either prevent the disease process, or decrease symptoms and viral shedding, depending on how they are prescribed. Until the 2008-2009 flu season, recent recommendations from the Centers for Disease Control advised using only the neuraminidase inhibitors, oseltamivir and zanamivir for seasonal flu, because of widespread resistance of the H3N2 seasonal flu to the older drugs amantadine and rimantadine. However, this season, resistance to oseltamivir in the prevailing H1N1 seasonal flu strain reached 98 percent, leading to recommendations for use of zanamivir alone, or in combination with rimantadine for confirmed cases of seasonal H1N1.

A handful of known cases of the new H1N1 swine flu of 2009 has now been shown to be resistant to oseltamivir as well (the strain was already resistant to amantadine and rimantadine). If the resistance factor spreads as rapidly in the 2009 H1N1 pandemic strain as it did in the seasonal H1N1 strain, by Fall, only one antiviral option might remain: zanamivir.

New Treatments Needed

Some flu experts have questioned why, with all the pandemic planning and funds poured into flu research since the H5N1 avian flu reappeared in 2003, little has been done to identify and mobilize for use, treatments aimed at quelling the sepsis (including the so-called cytokine storm) which leads to so many flu deaths, especially among the young and healthy. Not only is sepsis a prominent feature of fatal flu, it is a major cause of deaths from infections such as MRSA (methicillin resistant staphylococcus aureus) and other hospital-acquired diseases. In many of these cases, the basic innate immune mechanism is the same.

In fact, the recent outbreaks of community-acquired MRSA in the United States seemed to be concentrated in the same young and healthy population as the H1N1 swine flu. Even though MRSA is a bacterial invader, and influenza is a viral invader, the innate immune response of the host to the invader has many similarities. If drugs were found and developed which

11. Lone Simonsen, et al., 2007. "Mortality benefits of influenza vaccination in elderly people: An ongoing controversy," *The Lancet Infectious Diseases*, Vol. 7, No. 10, pp. 658-666.



Millions of people now live in urban slums, like this favela (shanty town) in Rio de Janeiro—or worse. The poverty, lack of basic water and sanitation infrastructure, and crowded conditions, exacerbated by the current economic breakdown, are perfect breeding grounds for the spread of diseases.

could modulate the overexuberant parts of the innate response to infection, without significantly weakening the overall immune response to the disease, many sepsis deaths could be avoided every year.

Such a group of drugs would be especially welcome at the beginning of a novel pandemic flu, before closely matched vaccines could be manufactured and distributed. Those showing signs of sepsis, or those in the most vulnerable groups for that particular flu, could be given such drugs to minimize the impact of the flu infection on their bodies.

This is not just a pipe dream, although few such drugs are now in the pharmaceutical pipeline. The eminent flu expert Dr. Yoshihiro Kawaoka, of the University of Wisconsin, has been pursuing such drugs with many collaborators, both in Japan and the United States. Several such drugs have been found that show promise in animals infected with the flu.¹² Dr. Robert Webster, another eminent flu expert, has also been working on this angle.¹³

Dr. David Fedson, a prominent flu researcher, now retired from the vaccine manufacturer Sanofi Pasteur, suggested as early as 2006 that the same statins prescribed to millions of middle-age and elderly people with high cholesterol could provide protection from the fatal sepsis common in infection with the H5N1 flu.¹⁴ Studies of hospital cases of pneumonia have shown that statins can reduce hospital deaths from pneumonia

12. David Marsolais, et al., 2009. "A critical role for the sphingosine analog AAL-R in dampening the cytokine response during influenza virus infection," *PNAS*, Vol. 106, No. 5, pp. 1560-1565 (Feb. 3).

13. Jerry R. Aldridge, Jr., et al., 2009. "TNF/iNOS-producing dendritic cells are the necessary evil of lethal influenza virus infection." *PNAS*, Vol. 106, No. 13, pp. 5306-5311 (March 31).

14. David S. Fedson, 2006. "Pandemic Influenza: A Potential Role for Statins in Treatment and Prophylaxis." (June 6)

by half. Fedson has recently written several papers promoting such agents as a first line of defense against a pandemic flu—before vaccines and antivirals become available for the masses.¹⁵

Many of these agents would be drugs already readily available in all countries, including the developing countries (which would be the last to secure sufficient vaccines and antivirals in a pandemic). Such drugs could be much cheaper than the vaccines and antivirals hoarded by the rich industrial nations; and they would be available even at the very beginning of the pandemic, for the first casualties, even in flus resistant to antivirals. As Fedson et al. stated in a 2007 paper on treatment alternatives for a pandemic flu:¹⁶

If, however, statins are shown to be of benefit, the public health argument for their use in a pandemic would be hugely compelling. Currently, a five-day course of

treatment with a neuraminidase inhibitor costs \$60 to \$90 in the US, and the global availability of these agents is limited. By contrast, generic statins are available worldwide and are inexpensive. In the US, a five-day course of treatment would cost approximately \$1.75, whereas in a developing country such as India, it would probably cost less than \$1.00. Moreover, unlike vaccines and antivirals, statins would be available in almost all countries on the first day of the pandemic.

Fedson has proposed an international effort to find many such drugs among the vast library of known drugs, herbs, or other substances. These candidates would be tested and developed for the purpose of standing as a first line of defense against our dreaded nemesis, pandemic flu—not to prevent it, but to turn a dragon of mythical proportions into an everyday pussycat.

Man's Relationship to the Biosphere

While these lines of inquiry are laudable and necessary, it may be that we already have a well-characterized and safe first line of defense against the flu at our fingertips. We just are not using it wittingly to our benefit. And this points up the noöspheric aspect of the flu idea. Man is not just a passive factory for the flu; like all life, man has tools at his disposal to fight the flu—physiological and behavioral tools to keep the flu at bay. But man has other tools not available to mere life: tools crafted through reason, or the lack thereof.

15. David S. Fedson, 2009. "Meeting the Challenge of Influenza Pandemic Preparedness in Developing Countries," *Emerging Infectious Diseases*, Vol. 15, No. 3, pp. 365-371.

16. David S. Fedson and Peter Dunnill, 2007. "New Approaches to Confronting an Imminent Influenza Pandemic," *The Permanente Journal*, Vol. 11, No. 3, pp. 63-69.



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Dr. Terrence Tumpey, a microbiologist at the Centers for Disease Control and Prevention, holding a reconstructed 1918 pandemic flu virus, inside a specimen vial containing an orange-colored culture medium. The virus was re-created in order to examine what made it so virulent.

Our modern civilization has, in a geological and evolutionary blink-of-the-eye, set man up in a new relationship with the Biosphere. With this new relationship have come inevitable unintended consequences—details still to be worked out by man's reason. The creation of cities with large populations of humans in close proximity is one great change, a change made possible by advances in agriculture over millennia, creating enough food for more people to exist, and for many to turn to other productive and creative labor.

However, the creation of cities and towns has had the unintended consequence of providing many diseases, such as tuberculosis, smallpox, cholera, and influenza, with more opportunities for sustained spread. Furthermore, within the cities and towns much of the population has often been made more susceptible to disease by their separation from the fruits of agriculture, and from nature in general. Within cities, the need for water and sanitary infrastructure becomes crucial, but the maturation of that technology has long lagged the development of cities. Even today, in the huge human agglomerations within the developing world euphemistically called cities, a crush of millions of the poor subsist with no such infrastructure.

Cities and towns are the first to suffer famine and then pestilence when war or nature disrupts the life-sustaining agricultural output upon which the whole edifice of civilization depends. One only needs to look back to the plague which struck Athens while it was under siege during the second year of the Peloponnesian War to realize the importance of proper food, water, and sanitation infrastructure for a civilization to survive.

Even during times of peace and plenty, the labor required of many in the cities and towns, especially since the industrial revolution, has necessitated working indoors during the hours of sunlight, leaving a majority of the population, especially school children, deficient in the direct harvest of the Sun's life-giving rays through the transformation of a certain portion of the ultraviolet spectrum's energy into a potent immunomodulating molecule known as Vitamin D. Especially in Fall and Winter, and in the temperate latitudes, there is a pandemic of Vitamin D insufficiency in the developed world.

This lack is especially prevalent among dark-skinned city residents in temperate climates, and among those who wear the most clothes, slather on the most sunscreen, and spend the most time indoors. After the Winter Solstice, the shallow angle of incidence of the Sun's rays through the atmosphere in temperate zones means that the important UV light is used up in chemical reactions with ozone, leaving little at the surface of the Earth's crust for transforming the pre-Vitamin D stored in the skin into the active precursor of the molecule. Then man must either live on the Vitamin D stored during Summer, move south like the birds, or ingest Vitamin D from dietary sources.

Rickets among children has been the noöspheric fossil of gross Vitamin D deficiency in temperate zones of the planet: soft bones, bowed legs—osteomalacia. It was so rampant in the 19th and early 20th centuries, that a public health measure was instituted in the 1930s, fortifying certain foods in the United States with Vitamin D, notably milk. However, the amount of Vitamin D in milk is small, and many of those most in need of the vitamin cannot tolerate ingesting dairy products. Public health measures have made rickets less prevalent (although it is on the upswing again), but they have not banished Vitamin D deficiency in its more subtle forms.¹⁷

There have been many studies in recent years pointing to a link between Vitamin D deficiency and several prevalent diseases associated with modern civilization: colon cancer, diabetes, heart disease, obesity, and multiple sclerosis, just to name a few. There has also been some mention of Vitamin D status and resistance to influenza and other infectious diseases, notably tuberculosis.¹⁸⁻¹⁹

Vitamin D Essential

Back in 1981, a British amateur scientist, R. Edgar Hope-Simpson, pondered the strong positive correlation in temperate latitudes between seasonal influenza and the Fall/Winter sea-

17. [New Roles of Vitamin D](#), 2006

18. John J. Cannell, et al., 2008. "[On the epidemiology of influenza](#)," *Virology Journal*, Vol. 5, No. 29.

19. John J. Cannell, et al., 2006. "[Epidemic influenza and Vitamin D](#)," *Epidemiol. Infect.*

son when the Sun was weakest. He spent the rest of his life unsuccessfully looking for the physical cause of the “seasonal stimulus” for the seasonal flu. It is a remarkable testament to the bankruptcy of much of molecular biology that we still, even today, know so little about such a powerful immunomodulatory molecule as Vitamin D. Because it is so cheap, being free to those who care to sunbathe, science has, until recently, been indifferent to it except where clinical manifestations of its lack are undeniable. Where deficiencies occur, cheap remedies of cod liver oil can solve the problem. In the Winter, brief exposure to UV lamps has been shown to work. There is absolutely no reason for anyone to be short on Vitamin D except:

1. People spend their time indoors working and playing. Many work at night and sleep during the day, as well.
2. Dark-skinned peoples have moved into temperate latitudes, where their highly pigmented skin blocks much of the incident UV light.
3. Women (and often men) in many cultures are encouraged to cover themselves completely while outside.
4. Fear of skin cancer and wrinkles.
5. Old people get less Sun exposure and are less efficient in using the UV light to make Vitamin D.

6. Breast-fed infants get no Vitamin D; their mothers often had little Vitamin D during gestation, and infants are often covered to protect them from the Sun.
7. Nutritionists (and the public), have underestimated the requirements of Vitamin D, and failure to routinely test blood levels has hidden the extent of the silent pandemic of deficiency. Most people think the Vitamin D problem was solved long ago by public health, and is not a problem for them.

Ninety percent of black children in a recent study had insufficient Vitamin D (by public health standards) during the late Winter. More than 50 percent of children in the study had insufficient Vitamin D overall. A high percentage of pregnant women have been shown to have insufficient Vitamin D at temperate latitudes. Dark-skinned pregnant women are at much greater risk.²⁰

Pregnant women were shown to have insufficient Vitamin D even when taking multivitamins. Their neonates, having no source of Vitamin D but their mothers, are even more deficient. Starting out life deficient in Vitamin D can lead to life-long health problems we are only beginning to investigate, including type 1 diabetes, multiple sclerosis, and other autoimmune



Department of Health and Human Services

An American child in 1970 with symptoms of nutritional rickets (bowed legs and enlarged right wrist), in which children's bones are too soft and do not develop properly because of a lack of Vitamin D. Many adults and children today, especially those with darker skin, are deficient in Vitamin D, and Vitamin D supplements are a frontline defense against the pandemic flu.

diseases, plus schizophrenia, obesity, and other ills. Many of these are the special risk factors which are correlated with increased risk of complications or death from seasonal flu.

While all countries with financial means are spending billions to purchase vaccines to protect their populations from a novel influenza, and large amounts are spent each year for seasonal flu, almost nothing has been done to test the theory that increasing Vitamin D blood levels in the population can reduce morbidity, mortality, and spread of the seasonal, or any other flu. This is all the more surprising since a proper blood concentration of Vitamin D, unlike the flu vaccine, has a multitude of other beneficial effects. In many poor countries, especially in temperate climates, Vitamin D deficiency coupled with other nutritional factors might be contributing to the high death rates seen in certain of the young during the present flu pandemic.

At least one country, Canada, has begun studying the effect of Vitamin D status on influenza susceptibility as it collects flu data.²¹ More countries should do the same.

Universal availability of Vitamin D supplements (as in Vitamin D-calcium-fortified orange juice), and education

campaigns connecting Vitamin D sufficiency with immune function may provide tools available to all people to fight the flu, especially as the pandemic vaccine will not be available to all, or even most, people in these countries this Fall.

During the Fall, national, state, and local public health officials will bring people together for mass vaccinations against the novel and seasonal influenza, especially at schools where children congregate. This would be the perfect time to administer Vitamin D to a large group, which could be followed up at a later time. The children and their parents could be educated about the value of Vitamin D and the dangers of its lack.

Although the old are slated to be last in line for the novel flu vaccine, they should be high on the list of those receiving Vitamin D, as they are the most deficient, and the most susceptible to seasonal flu.

The Task Today

As we have seen, influenza is not a simple mathematical expression in one variable. It is an extremely complicated interaction of variables residing in three separate but finely intermeshed

20. Lisa M. Bodnar, et al., 2007. “High Prevalence of Vitamin D Insufficiency in Black and White Pregnant Women Residing in the Northern United States and Their Neonates,” *The Journal of Nutrition*, Vol. 137, No. 2 (Feb.).

21. http://www.privatemdlabs.com/news/Vitamin_D_Deficiency-Diagnosis_and_Treatment_/Canadian-government-using-vitamin-D-testing-to-determine-susceptibility-to-S19286038.php

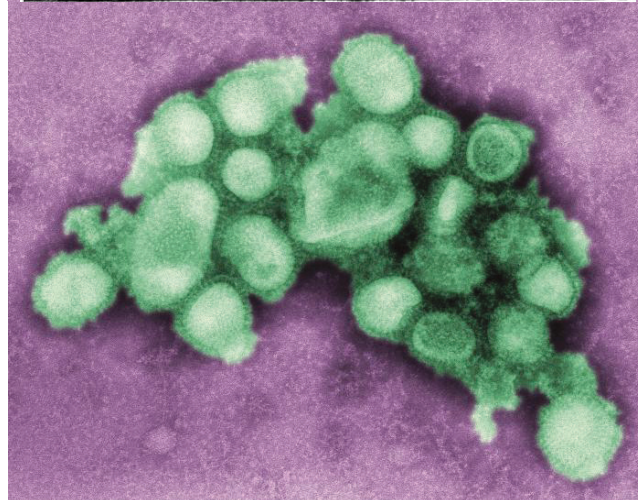
and multiply connected domains of existence, the nonliving, the living, and the Noösphere—a kind of three-body problem. The dynamics of influenza are astoundingly complex, and only an understanding of its dynamics within the Biosphere can give us long-term solutions to its yearly cycle of infection. These solutions must take into account genetic potentialities of both the virus and host, as well as the health and nutritional status of the host.

Furthermore, they must take into account the seasonal and climatic cycles of the planet. Finally, the solutions must take into account the health and nutritional status of the Noösphere itself—the scientific, technological, and cultural level of man within the Biosphere.

This last area is obviously the most important aspect for flu dynamics, because it is through the Noösphere that man gains the power to alter processes within the Biosphere, either for his benefit or his detriment. Without the Noösphere man is as helpless before disease as a beast. Today, 10 years into the 21st Century, we are still relying mainly on a 200-year-old idea—vaccination—to manage the flu.

Without knowledge of the nature of viruses, men like Louis Pasteur experimented to alter the human terrain to make it more resistant to the agents of disease by stimulating the unknown black box of the immune system with viral antigens. This was a very powerful intervention for clinical medicine and public health, and led to huge advances in biological and medical sciences. The modern medical search for antimicrobial drugs is more than a century old, spearheaded by searches for drugs that could treat syphilis and tuberculosis—two bacterial scourges of the 19th Century. We can add a third great idea tied up with these other discoveries of the dawn of microbiology: that

As the ignorance, poverty, rage, and fear expand, the Four Horsemen of the Apocalypse (Albrecht Dürer woodcut, 1498) will commence their ride, mowing down populations in their path like a sharpened scythe through ripe grain.



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of disinfection.

Since those discoveries, although medical and biological fields and subfields have proliferated like viruses, we have not really progressed much beyond these simple ideas, which came only with great difficulty to our ancestors. Although its effects are blunted by medical and other advances, influenza still has the upper hand. And whenever man's cultural and scientific progress halts or regresses, influenza and other infectious diseases roar back with a vengeance.

This is where we stand today. Not only have we not developed new technologies to deal with influenza, but we have spent 40 years dismantling the medical infrastructure that it took a century to develop.

Furthermore, today we are teetering over the abyss of a financial breakdown crisis that will bring on a profound noöspheric contraction of a magnitude not seen since the days of the Black Death in Europe. As the ignorance, poverty, rage, and fear expand, the Four Horsemen of the Apocalypse will commence their ride, mowing down populations in their path like a sharpened scythe through ripe grain. Billions could die of war, starvation, and disease in a Dark Age spanning generations.

To avoid this unthinkable calamity, we must stop the tumble into the abyss by reversing the recent ruinous economic/financial policies now dragging us over the edge. We must rebuild our ruined physical economy, and set our sights on a Noösphere-expanding mission to propel us into the future.

How about building a colony on Mars?²²

22. <http://www.larouchepac.com/webcasts/20090801.html>