

# Viral Panic, Vulnerability, and the Next Pandemic

*Ann Herring*

## **Introduction**

The problem of infectious disease in human societies, past and present, is an important site for anthropological theorizing because it sits at the juncture between the microcosmos, evolution, and human behavior. It forms a natural bridge between the nature/culture divide. In this essay, I discuss the intersection between the social and biological worlds through a consideration of the prospect of an avian influenza pandemic in the twenty-first century and its connections, real and constructed, to the 1918 influenza pandemic.

More specifically, I explore a narrative line that is embedded in the discourse on avian influenza. During the course of any epidemic, social responses surface in parallel to the challenge of the disease itself as the epidemic takes shape, becomes visible, and then is acknowledged by the people and societies vulnerable to it. Explanations emerge as a means of regaining control and asserting rationality over the crisis. Structures of blame inevitably arise through the process of explanation, and as managing the epidemic becomes a vehicle for social control. As disease and death subside, moral lessons are drawn (Rosenberg 1992).

Narratives have a powerful influence on public concern about health crises and may influence health policy. For this reason, it is important to identify and critique the narratives and moral lessons that run through scholarly and media discussions of epidemics, here exemplified by avian influenza. In the case of avian influenza, failure to identify the connections between “bird flu” and the social, economic, and political contexts that influence who is actually vulnerable to it, creates panic about an inevitable global pandemic that threatens everyone. It also masks who is likely to be at the greatest risk of acquiring and dying from avian influenza. This is dangerous from a

public health policy perspective. In this essay I propose that the concept of syndemic offers a useful way to consider both the terrain and the microbe together, and to develop a more textured analysis of who may be vulnerable to “the next pandemic.”

## Viral Panic

By the third quarter of the twentieth century, interest in infectious disease had waned—at least in a western medical context—and epidemiologic transition theory had relegated pestilence and famine to the past. Degenerative and human-induced diseases, such as cancer and cardiovascular diseases, predominated (Oman 1971). Frederick Cartwright’s leap of faith in 1983 nicely captures the conviction of the period: “It is my belief that, unless control breaks down through widespread famine or atomic warfare, both of which are possibilities, our world has seen the last of the great killing pandemics” (1983: 279). He was not alone. In 1967, William H. Stewart, surgeon-general of the United States, declared that “victory over infectious disease was imminent” (Armstrong 1998: 24).

Then HIV/AIDS emerged to shake the foundations of epidemiological thought. The worldwide pandemic demonstrated that infectious disease was not a vestige of the past but an inevitable aspect of living in the organic world (Lederberg 1988), even the affluent Western world (Morse 1991:387). In his book *History of AIDS: Emergence and Origin of a Modern Pandemic*, Mirko Grmek captures the complete reversal in thinking that accompanied the emergence of HIV/AIDS: “Influenza was the last of the classic pestilences; AIDS, both unpredicted and unpredictable within the framework of the old nosology, is the first of the postmodern plagues” (Grmek 1990: ix).

As the security of the age of degenerative and human-made diseases (Oman 1971) has given way to the age of emerging, re-emerging, and antimicrobial resistant diseases (Barrett et al. 1998), anxiety has grown as a breathtaking array of emerging and re-emerging diseases has been recognized, along with the many factors that contribute to their new visibility (Waltner-Toews 1995: 46).

We live in an era obsessed with killer germs, says Nancy Tomes, in an era of “viral panic” (2000: 194). A “post-AIDS, post-Cold War crisis of confidence” has emerged as the old twentieth century belief in the biomedical conquest of infection has faded in the face of insurmountable evidence to the contrary (Tomes 2000: 192). There is a new sense of vulnerability and uncertainty with respect to infectious disease, rekindling fears of mortality on the scale of historic plagues and spurring research into the origins and circumstances that allowed epidemics to erupt and flourish in the past. Since it came into view in 1981, HIV/AIDS has “stimulated more interest in history than any other disease of modern times” (Fox and Fee 1988: 1).

## Vulnerability to a Pandemic

A body of opinion now considers emerging infectious diseases and epidemics as inevitable (Klempner and Shapiro 2004: 2334), natural features of human life in

a dynamic ecosystem (Lederberg 1993), connected to human-induced changes in that ecosystem (Last 1999). Among the emerging diseases currently generating viral panic—and apocalyptic terror that it represents the seeds of “the next pandemic”—is avian influenza (H5N1).

As we wait for the next pandemic, discussions of viral evolution understandably have assumed enormous significance. There are three major forms of influenza (A, B and C), but only influenza A gives rise to pandemics. Influenza A, the 8-stranded RNA virus associated with human pandemics, has the capacity to evolve rapidly through genetic recombination with influenza strains from animal species (Palese 1993: 226), a process through which it can evolve suddenly and dramatically through genetic shift. These new combinations of genes, in turn, produce variation in the two antigens, hemagglutinin (H) and neuraminidase (N) that sprout from its surface coat. When this process of hybridization and genetic shift occurs, a new strain of influenza emerges. Ultimately, influenza is a zoonotic disease of avian origin; all known influenza A subtypes originated from the aquatic bird reservoir (Webster 1998). It spreads efficiently via droplet nuclei and has a short incubation period, which enhances its ability to spread rapidly from person to person. It “is probably one of the oldest emerging viruses” and may have been responsible for epidemics in ancient Greece and Rome (Webster 1993: 37).

The antiquity of influenza pandemics, their reservoir in aquatic birds, and the emergence of a new avian virus H5N1 leads to questions about how far away we are from the next pandemic. There are three steps in the process: 1) transmission of a new influenza viral subtype to humans; 2) viral replication that produces disease in humans; and 3) efficient human-to-human transmission of the virus. Since 1997, the first two conditions have been met on several occasions. As for the final condition, efficient human-to-human transmission, “The question ... is when such changes will happen” (Monto 2005: 324). “It could happen tonight, next year, or even ten years from now” (Osterholm 2005: 36). The last of the classical pestilences is the impending scourge of the twenty-first century.

When the H5N1 strain of avian influenza infected and killed six people in Hong Kong in 1997, the World Health Organization ordered the slaughter of all chickens to prevent the third step, efficient human-to-human transmission, and a worldwide pandemic of bird flu. The virus was not eradicated and avian influenza, endemic in poultry in many parts of Asia, continues to evolve. In 2003, the Z strain of H5N1 emerged. Pathogenic to a wider range of species compared to other strains, the new strain is also resistant to first-line antiviral drugs, such as amantadine and rimantadine (Monto 2005: 323).

The Z strain has widened its geographical range. In 2004 it had spread to nine countries in East and Southeast Asia (Li et al. 2004) and was identified in the Middle East, Africa, and Europe in 2006 (WHO 2006a). It is expected to infect poultry operations in North and South America (Butler and Ruttimann 2006), though this had not transpired at the time of writing (WHO 2008a). As more poultry are infected,

and as increasing numbers of people are exposed to H5N1, “all the prerequisites for the start of a pandemic have been met save one—namely, genetic changes in this virus that would allow it to achieve efficient human-to-human transmission” (Stöhr 2005: 4).

Person-to-person spread of avian influenza nevertheless has been documented. PCR analysis of a cluster of seven relatives who contracted H5N1 in a remote village in Sumatra, Indonesia, indicates that a father contracted the virus after prolonged, close contact with his ailing 10-year-old son whom he nursed in hospital (Rosenthal 2006). Studies of human cases of avian influenza show, however, that the virus does not spread easily between people. It tends to colonize the lower lung and favours cell receptors in the deepest branches of the respiratory tract. Its preference for deeply buried tissue has limited the ability of H5N1 to spread from person to person by coughs and sneezes (Shinya et al. 2006). This seems to have inhibited achievement of the final step along the road toward a pandemic: *efficient* human-to-human transmission.

### **Anchoring Avian Influenza to the 1918 Influenza Pandemic**

Fears that a killer bird flu is on the horizon—along with the massive damage that may accompany it—are anchored in the 1918 influenza pandemic and H1N1, the influenza A virus associated with it. *Anchoring* is a mechanism whereby the understanding of a new disease is linked and configured in terms of past epidemics (Joffe 1999). This is a process of representation through which a crisis is made understandable and less threatening by connecting it to familiar historical events, metaphors, or symbols.

Anchoring a potential H5N1 outbreak to the 1918 influenza pandemic serves to enhance the climate of viral panic. This also occurred when media representations of SARS linked it to the 1918 influenza pandemic and the Black Death (Washer 2004). In much the same way, anchoring the vCJD/BSE to HIV/AIDS in Britain in the late 1990s increased fear (Washer 2006). Discussions of avian influenza’s potential to produce an unimaginable death toll draw parallels to the 1918 outbreak in which some fifty to one hundred million people may have perished worldwide (Johnson and Mueller 2005). The rapid spread of the disease, sudden onset of symptoms among otherwise healthy people, and excess mortality among young adults in the prime of life are frequently reported. The symptoms and medical histories of people who died from H5N1 and from H1N1 in 1918 are described as “disturbingly similar” (Garrett 2005: 14) and H5N1 seems to have an affinity for previously healthy young adults and children.

There are other, less alarming and destructive pandemics that could be anchored to avian influenza, notably 1957 (“Asian influenza pandemic,” H2N2), 1968 (“Hong Kong pandemic,” H2N2), and 1977 (“Russian flu” or “Russian threat,” H1N1). They are not invoked in discussions of avian influenza or other frightening new diseases, such as SARS. This is because the 1918 pandemic is constructed as “the catastrophe against which all modern pandemics are measured” (Pandemics and Pandemic

Threats Since 1900: 1). It is “the mother of all pandemics” (Taubenberger and Morens 2006) and the gold standard for emerging and re-emerging disease.

The 1918 pandemic is anchored, in turn, to ancient plagues. Its devastating death toll, for example, is said to have outranked the Black Death and the Plague of Justinian (Walters 1978: 856). At the time of the 1918 pandemic, when no one knew what was causing healthy people to sicken and die with astonishing speed, and from frightening symptoms, some suggested that the Spanish Flu actually *was* the Black Death in new guise (MacDougall 1985: 2090–91). In this way, the discourse about avian influenza is connected, through the 1918 pandemic, to medieval plague—the classic image of pestilence and plague.

The connection between the H1N1 1918 virus and H5N1 avian influenza tightened in October 2005 with the publication of the genome for H1N1, an internationally newsworthy event (see Appendix 1 of this chapter for more details). Initial phylogenetic analysis had suggested that the 1918 variant of H1N1 was closely related to a classical swine flu strain (Reid et al. 1999). A later, more comprehensive analysis resulted in a different conclusion: the strain’s genome was primarily avian (Taubenberger et al. 2005). This heightened worries about the risks to global health from avian influenza. This anxiety was magnified in January 2006 when H5N1 virus samples taken from people in Turkey were discovered to carry mutations believed to have the potential to facilitate person-to-person spread. Later, this conclusion was judged “premature” and “overinterpreted” in light of the genetic complexity of the influenza virus and the fact that virulence and transmissibility are multigenic traits (Basu 2006: 258). But it is evident from the hasty conclusions that scientific researchers are not immune to the influence of viral panic.

Even *differences* between the two viruses provoke anxiety. A high case rate and low mortality rate are well known features of the 1918 outbreak; the vast majority of people who contracted influenza recovered from it. There was considerable variability in the death toll from influenza (see below), but influenza mortality averaged about 3 percent, exceeding the less than 0.1 percent mortality typical for other influenza epidemics (Dull and Dowdle 1980). This is much lower still than the case-fatality rates for the 1997 Hong Kong outbreak of avian influenza in which 18 percent of affected children and 57 percent of adults older than 17 years of age died (Snacken et al. 1999). WHO mortality rates for the 362 reported, laboratory-confirmed human cases of H5N1 average 63 percent (WHO 2008b), contributing to the fear that avian influenza is “far more dangerous” than the 1918 variant (Garrett 2005: 3).

### **Who is Vulnerable to Avian Influenza?**

According to classic epidemiological theory, virtually everyone is vulnerable to H5N1 avian influenza. This is because there are no antigens from previous exposures that would confer immunity to individuals, and herd immunity to communities. Still, human cases of H5N1 are not found in all age groups. Analysis of the forty-four cases

of H5N1 documented in 2004, for instance, shows that avian influenza was concentrated in previously healthy children and young adults for whom the case fatality rate was 73 percent (Stöhr 2005).

This nexus of illness and death among the young looks suspiciously like the behavior of a disease that has afflicted the population before and to which older adults may have already acquired immunity through exposure in childhood. Such an interpretation is consistent with the observation that H5 viruses have been present in human populations since the late 1950s (Wade 2006). In addition, immunity to the N1 antigen has been insufficiently studied (Kilbourne 2006: 13). To have a better sense of who is actually vulnerable to contracting and dying from avian influenza, we need to know more about the seroprevalence of H5N1 in communities that have been affected by it. Furthermore, there has been insufficient study of the social and economic context of vulnerability to infection. The higher risks of contracting avian influenza, especially among females in the 10–29 age category, may be linked to their roles in poultry farming, such as culling and de-feathering birds, or in food preparation (WHO 2006b: 256).

Human cases of avian influenza tend to cluster among relatively impoverished people, mostly rural farm families, in countries with developing economies in Southeast Asia (WHO 2007). Important social and economic factors, such as subsistence farming and poverty—that contribute to human vulnerability to all infectious diseases—are receiving little attention in the face of H5N1 viral panic in the West (Lockerbie and Herring in press). To whom is avian influenza actually “emerging” (Farmer 1999)?

### **Vulnerability to Stigma (Shame and Blame)**

Since the 1930s, all serious outbreaks of influenza have developed in Southeast Asia (Scholtissek 1992). The focus of blame for avian influenza, therefore, has centered on Asian countries, the geographical epicenter identified for most new variants of influenza (Scholtissek 1994) and, so far, the region hardest hit by H5N1 in poultry and humans (WHO 2007). It has been suggested that aquaculture, a common form of agriculture in this region, favors cross-species exchange of influenza genes. Aquaculture brings ducks, pigs, and humans together in close contact. Specific receptors in the pig’s throat allow both bird and mammalian influenza viruses to enter pig cells, intermingle, swap genes, and generate new variants of influenza virus (Ito et al. 1998). Swine therefore can act as “mixing vessels” for influenza strains, resulting in novel trans-specific strains (Scholtissek 1992, 1994; Hollenbeck 2006).

Implicated as the origin of new influenza viruses, Asian agricultural and health practices have consequently received extensive attention and censure. In discussing communities afflicted with avian influenza, images are offered up of filth and backwardness (lack of modernity), subsistence farmers living in close proximity to animals, densely packed open markets, and poverty (Figure 3.1).



**Figure 3.1.** “Live-poultry markets like this one in Hanoi speed the spread of the virus from farm to farm when vendors take leftover birds back home, along with any flu viruses they’ve picked up” (Appenzeller 2005:13).

Such images have a rapid and global stigmatizing impact (Lee et al. 2005: 2044). The message is that small-scale farming, aquaculture, and open-air markets common in Asia are dangerous to global health. By implication, Asian subsistence farmers and market vendors are not good citizens of the world and are threats to world health. Yet it is the large-scale, international poultry industry that creates conditions that favor the emergence of new avian influenzas, not the small-scale poultry farmers typically depicted in media and scientific accounts (GRAIN 2006). The virus spreads slowly among small village chicken flocks and has difficulty persisting under such low-density conditions; in contrast, it spreads and amplifies quickly in densely packed factory farms. Integrated trade networks offer efficient routes for the spread of infection; in most cases, trade has been the agent of viral diffusion (Butler 2006). The international trade in day-old chicks, eggs, live birds, meat, and secondary products, such as chicken manure, feathers, and animal feed, create the circumstances in which avian influenza can spread globally. In Laos, for example, 90 percent of chicken production comes from small-farm and backyard operations, yet the only outbreaks of H5N1 on these farms have come from those next to the country’s small number of factory farms (GRAIN 2006: 9).

Countries with avian influenza lose international markets for agricultural products and risk global censure, such as China faced in the wake of SARS (Washer 2004:

2565). Yet destroying infected poultry flocks literally wipes out the livelihoods and food security of small-scale farmers. By 2006, over 150 million birds had been culled in Asia, and avian influenza is estimated to have cost Asian economies in excess of \$15 billion dollars (Anand 2005–6: 18).

And while the focus of attention remains on a Southeast Asian epicenter for “the coming plague,” this was certainly not the source for the 1918 pandemic (Herring and Padiak 2008). The current debate about the 1918 pandemic locates its probable epicenter either in the USA (Barry 2004a, b; Burnet and Clark 1942; Crosby 1989; Jordan 1927) or in Western Europe (Oxford et al. 2002, 2005).

### **A Syndemic Approach to Vulnerability**

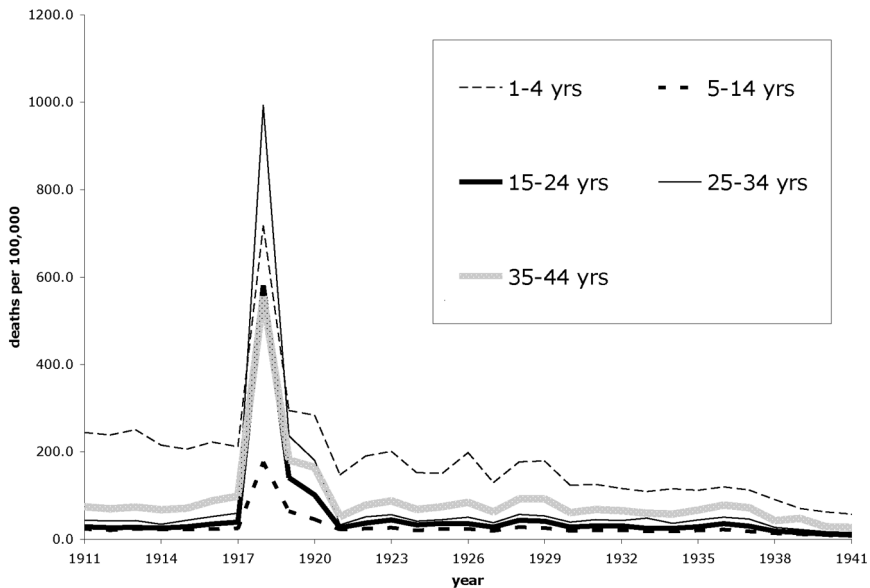
To recapitulate, current panic about the next pandemic focuses on the avian influenza virus, H5N1, whose origins are Asian. The mutability of influenza strains, their shifting antigenic coats, ability to infect human and animal species, to evolve and spread rapidly across boundaries, are elements of the “mutation-contagion package” of fear (Ungar 1998). Anchored to the 1918 influenza pandemic, H5N1 avian influenza contributes to the foreboding that a global cataclysm of unmatched dimensions lies just beyond the horizon.

In invoking the 1918 pandemic as the model for the coming pandemic, it is evident that some features of that outbreak have been stressed, such as the deaths of fifty to a hundred million people, while others, such as the extensive variation in death tolls, have received less attention. In presenting the 1918 outbreak in this way, what is highlighted and what is obscured? The wider terrain and the social context of the period also warrant careful scrutiny (Farmer 1999). To this end, the concept of *syndemic* provides a useful framework for exploring vulnerabilities to pandemics, past and present. A syndemic is a set of interactive and mutually enhancing epidemics involving disease interactions at the biological level that develop and are sustained in a community or population because of harmful social conditions and injurious social connections (Singer and Clair 2003: 429). The utility of the concept is well-illustrated by the example of a whooping cough epidemic in 1927 in the Canadian north (see Appendix 2 to this chapter).

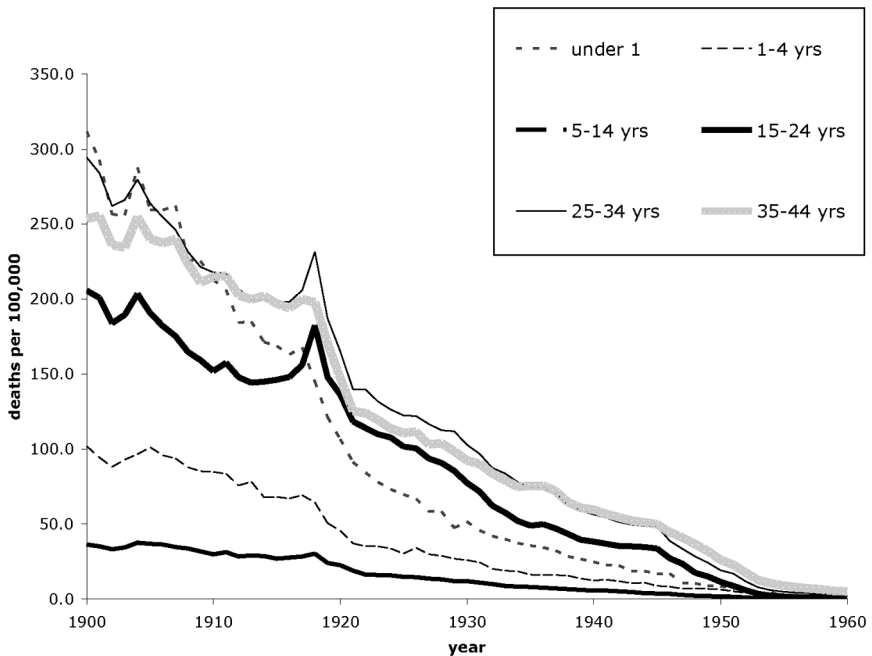
Let us consider the first facet of the syndemics concept: mutually enhancing epidemics involving disease interactions. Studies of the effects of the reconstructed H1N1 virus in *Macaca fascicularis* suggest that the 1918 virus provoked a severe respiratory infection and aberrant expression of the immune response that may help to explain its unusual virulence (Kobasa et al. 2007). That said, scholars have known since the 1918 outbreak that the majority of influenza sufferers recovered within about a week. About 20 percent developed severe secondary infections that gave rise to fatal pneumonia, sometimes within twenty-four hours. The deadly complication of influenza pneumonia killed 40 to 50 percent of people with secondary infections (Burnet and Clark 1942: 88).



Clearly, it is not sufficient to focus solely on the H1N1 virus; secondary infections played a significant role in the virulence of the 1918 pandemic (Crosby 1989; Kilbourne 2006). Tubercular infection, for example, may have contributed to its lethality. In the USA, tuberculosis mortality peaked along with influenza in 1918 (Figures 3.2 and 3.3). Excess mortality associated with the 1918 pandemic, it is argued, reflects interaction between the pathogens associated with two co-occurring epidemics: influenza and tuberculosis (Noymer and Garenne 2000, 2003; Noymer 2006). In other words, having tuberculosis increased the chances of dying from influenza. Analysis of Union Army veteran data reveals a statistically significant association between having tuberculosis and dying from influenza in 1918, as well as during inter-pandemic years, with tubercular individuals being four times more likely to die from influenza than those free of the disease (Noymer 2006). This selective mortality effect had long-term consequences for national patterns of mortality in the USA, resulting in a dramatic reduction in tuberculosis mortality in the aftermath of the pandemic (Noymer and Garenne 2000, 2003; Noymer 2006). In contrast, deaths from tuberculosis *decreased* in England and Wales during 1918–19 (Johnson 2003: 137). From these examples, it is evident that the expression of the 1918 pandemic differed, depending on local infectious disease ecologies and histories.



**Figure 3.2.** Influenza death rates under age 45 (without infants), 1911–1945, United States (Herring et al. 2006, drawn from data in Grove and Hetzel 1968; Linder and Grove 1947).



**Figure 3.3.** Tuberculosis death rates under age 45, 1900–1960, United States (Herring et al. 2006, drawn from data in Grove and Hetzel 1968; Linder and Grove 1947).

Let us now turn to the second facet of the syndemics concept: the maintenance of interacting epidemics because of harmful social conditions. The point is especially interesting in the context of the 1918 outbreak. This is because emerging diseases are usually understood to be “democratic” in the sense that everyone is theoretically vulnerable because no one has antigens that confer resistance to the new pathogen. In other words, there are not supposed to be health inequalities in the face of a newly emerging disease. The 1918 influenza pandemic, however, was anything but democratic. It took a disproportionate toll among young adults, pregnant women, tubercular individuals, immigrant and economically disadvantaged neighborhoods, and marginalized communities that lacked access to health care (Johnson 2003; Jones 2005; Lux 1997; Mamelund 2006; Noymer 2006; Noymer and Garenne 2000, 2003; Taubenberger and Morens 2006).

Some communities escaped infection altogether; others in the same region were devastated by it (Herring 1994; Herring and Sattenspiel 2003; Herring and Sattenspiel 2007). Recent recalculations of mortality on national and continental scales reveal how variable the death toll from the 1918 pandemic actually was (Johnson and Mueller 2002). African nations, for instance, show a range extending from 10.7 per

1,000 (Egypt) to an extraordinarily high or 445 per 1,000 (Cameroon), with most national estimates hovering between 20 and 50 per 1,000. Rates were lower in the Americas, ranging from 1.2 per 1,000 (Argentina) to 39.2 per 1,000 (Guatemala); the estimated rates for Canada and the US are about 6 per 1,000. In Europe, influenza mortality rates were even lower, ranging from 2.4 per 1,000 (Russia) to 12.7 per 1,000 (Hungary). The constellation of biosocial conditions that contributed to this diversity has barely been explored and warrants close scrutiny, as the implications are important for future pandemics.

### **Implications and Conclusions**

A syndemic approach—which considers biological synergies and their connections to harmful social circumstances—is a useful way to begin a discussion of inequalities in the experience of the 1918 pandemic, both locally and globally (Singer and Clair 2003). To develop local profiles of vulnerability, careful analysis of disease interactions and their distribution within and between socioeconomic groups needs to be conducted using historical mortality series for 1918 and beyond. The long-term impact of the pandemic on morbidity and mortality has scarcely been assessed beyond the suppression of life expectancy at birth that resulted from the deaths of so many young people. There are barely any national histories or systematic analyses of its connection to social conditions during World War I (Phillips 2004: 130–31). Until such studies are undertaken, the incorrect, stereotypical view of the H1N1 strain of influenza as a universal and relentless killer will continue to be communicated to the public.

This is not just a historical problem; it has important implications for public health policy. The “Spanish Flu” is the model against which catastrophic pandemics are compared; it is the “mother of all pandemics” (Taubenberger and Morens 2006). Fears about avian influenza have been linked to it in the scientific literature and media reports and it is a key element of popular narratives about “the next pandemic.” Anchored to the 1918 pandemic, and in the absence of analysis of the social, economic, and political circumstances that determine the virus’s distribution within and between societies, H5N1 will continue to generate viral panic about an inevitable global pandemic that threatens everyone. Viral panic will be fuelled as long as the complexities of the virus’s interactions with other pathogens, and their links to underlying social inequalities, remain superfluous and unexplored in comparison to the allure of the microbe itself. Failure to explore who is likely to be at greatest risk of acquiring and dying from avian influenza is dangerous from a public health policy perspective.

### **Appendix 1**

#### **Was the 1918 Pandemic Caused by a Bird Flu Virus?**

The development of sensitive PCR techniques, coupled with the successful search for tissue samples from individuals who died from influenza during 1918, allowed the

genome of the virus to be studied from a molecular perspective. A multidisciplinary team headed by Jeffrey Taubenberger of the Armed Forces Institute of Pathology (AFIP) located preserved tissue samples from autopsied individuals who died during the 1918 epidemic: a 21-year-old soldier from Fort Jackson, South Carolina, and a 30-year-old man stationed at Camp Upton, New York. Both men had died of influenza on September 26, 1918 (Taubenberger et al. 1997). The AFIP team later obtained biopsied lung tissue taken from the frozen body of an Inuit woman buried at the Teller Mission on the Seward Peninsula in Alaska.

Using the three sets of tissue samples, the team eventually sequenced the entire gene for hemagglutinin (H1), the surface antigen that allows influenza viruses to infect cells. The initial results seemed to support the idea that the closest relative of the 1918 sequences is the oldest classical swine flu strain, characterized as influenza A/Sw/Iowa/30 (Reid et al. 1999).

In the autumn of 2005, the team announced that it had completely decoded the 1918 influenza genome (H1N1). The new results contradicted their earlier interpretation. Rather than the product of reassortment with swine influenza, the new molecular research indicated that the 1918 virus had an almost entirely avian genome (Taubenberger et al. 2005). The results suggested that H1N1 most likely jumped from birds to humans shortly before the pandemic (Taubenberger and Morens 2006), a time frame that may have been as long as several years.

Publication of the findings electrified scientists and the popular press alike, fanning viral panic. Yet the plausibility of the avian-origins hypothesis of H1N1 in 1918 has been challenged on a number of fronts, before and after publication of the genome results. Hollenbeck (2005: 89), for instance, stresses the role of pigs as intermediate hosts necessary to convert avian strains to human strains, the lack of evidence that H5N1 has adapted to humans, and the rarity of avian influenza prior to the 1997 H5N1 outbreak in Hong Kong. Gibbs and Gibbs (2006) contend that errors were made in interpreting the virus's phylogenetic relationships, arguing that the 1918 virus is closer to mammalian than to avian viruses. Instead of leaping to humans shortly before the pandemic, they counter that it may have evolved in pigs or people for an unknown period of time prior to the pandemic. Antonovics and colleagues (2006) also disagree with the proposed avian derivation for the 1918 outbreak. Chastising the team for inflaming the public, they say: "This alarming implication, which is based on misinterpretation of the phylogenetic data, is completely unjustified and could seriously distort the public perception of disease risk, with grave economic and social consequences" (p. E9). Taubenberger and colleagues (2006: E10) responded that they "never maintained that the virus entered the human population in 1918...[and that] phylogenetic analysis on its own cannot definitively resolve this issue."

Evidently, there is still much to be learned about the origins of the 1918 influenza virus.

## Appendix 2

### Applying the Syndemic Approach: Whooping Cough at York Factory.

A seemingly obscure epidemic of whooping cough in 1927 at York Factory, Manitoba, Canada, illustrates the utility of a syndemic approach (Singer and Clair 2003). The severity of this particular epidemic cannot be understood as a singular event in isolation from concurrent and preceding epidemics, nor without placing these interacting epidemics within the context of deleterious social conditions characteristic of the place at that time (Herring and Young 2005).

York Factory is located on a flat, marshy peninsula on the western shore of Hudson Bay near the mouth of the Hayes River. Established in 1714 as a fur trade post, it was the main port of entry for European trade goods to western Canada and quickly became the Hudson's Bay Company's (HBC's) single most important trading post on the bay (Beardy and Coutts 1996). Cree and Assiniboine middlemen were the lynchpins of the business, acquiring furs from a far-flung network of groups in the interior plains and woodlands (Ray 1974:72). Some settled semi-permanently in the immediate vicinity of the post at York Factory. The Home Guard Cree, as they came to be called, trapped, hunted and fished for the company and were the backbone of its success.

Over the centuries of its operation, York Factory boomed to prosperity along with the fur trade and then declined during the course of the nineteenth century. Game was depleted in the Northwest, and declining fur harvests prompted the Hudson's Bay Company to close many of its trading posts. Places like York Factory were basically "trapped out" by the mid nineteenth century and their economies were failing.

The disease ecology changed over the course of its history, in concert with the westward expansion of the American frontier, growth of urban disease pools, and improved transportation efficiency, which allowed diseases with short periods of infectivity to spread more easily into the Canadian north (Hackett 1991, 2002). Tuberculosis had become a major health problem, both as a specific cause of death and as an underlying condition that reduced resistance to other infectious diseases (Stone 1925: 79). The soaring tuberculosis problem resulted in a tuberculosis death rate among Indians in the western provinces of Canada that was ten to twenty times higher than that for non-Aboriginal people (Stewart 1936: 675).

It is against this backdrop of declining economic and health conditions that a virulent epidemic of whooping cough struck York Factory in the autumn of 1927. Whooping cough is the common name for *pertussis*, which means "violent cough." Its name is derived from the diagnostic "whoop" cough: a high-pitched intake of air followed by rapid, consecutive coughs. This almost unmistakable symptom allows pertussis to be diagnosed with relative accuracy in nonmedical settings. It is a strictly human infection, primarily affecting children under the age of 6, and most often caused by the bacterium *Bordetella pertussis* (Cherry 1999). About 50 percent of cases occur in children under the age of 2, and most deaths occur among infants

(under the age of 1). A highly contagious infection easily spread within households, whooping cough is acquired by droplet infection through close contact with sufferers, often adults.

The 1927 whooping cough epidemic at York Factory was exceptionally severe. Approximately 40 percent of all deaths recorded in the parish registers that year were attributed to it (ACCA 1864–1929). About 35 five percent of the whooping cough deaths occurred among infants—where most deaths are expected to occur— but it is unusual to see older children succumbing to the disease in such large numbers. The mortality rate for children under 6 years of age was an astonishing 237 per 1,000. When the deaths of two teenage girls are taken into account, whooping cough mortality under the age of 15 reached 157 per 1,000.

Why was the epidemic so lethal? A syndemic perspective requires close consideration of other epidemics afflicting the community, whether they could have been acting synergistically with whooping cough, and whether co-occurring epidemics can be tied to deleterious social conditions that allowed each to flourish and capacitate the other.

A closer look at 1927 shows that it was a terrible year at York Factory. In addition to the autumn whooping cough epidemic, the community had been devastated by an influenza epidemic the previous February. In fact, about 32 percent of the recorded deaths in 1927 were attributed to this deadly outbreak. Entries in the *York Factory Post Journal* indicate how overwhelming the epidemic was: “We are having difficulty to get men and dogs. Most of the men are sick with Flu” (HCBA 1794–1939, Feb. 21, 1927). “The flu epidemic ... [is] very vicious ... everyone laid up” (HBCA 1794–1939, Feb. 26, 1927).

Influenza took its greatest toll among adults; 87 percent of the deaths occurred in people between the ages of 21 and 65, the age group most productive in fur trade activities. With the whole community laid up during a crucial time in the annual fur harvest, the outbreak not only debilitated the people but undermined the local economy that year “Disgusted with [the fur] trade. This has been the poorest spring trade for many years” (HBCA 1794–1939, June 13, 1927).

To recapitulate, in 1927 a severe influenza epidemic killed mostly adults and led to a poor fur harvest, followed by a fall whooping cough epidemic that killed mostly children. All of this occurred against a backdrop of endemic tuberculosis.

Were the epidemics intertwined? There is every possibility that they were. All three are respiratory diseases that affect the lungs. As an underlying condition, tuberculosis opens up already compromised immune systems and debilitated lungs to other respiratory infections (Noymer 2006), which would have made tubercular members of the community more vulnerable to influenza and whooping cough. Active tuberculosis, moreover, can exacerbate influenza infection, making it worse. Influenza, in turn, enhances bacterial lung disease, impairs normal recover mechanisms, and impairs the immune system (Couch 1981). The virus has a lethal synergy with pneumococcus bacteria when infection with influenza precedes pneumococcal infection (McCullers and Rehg 2002). Severe influenza pneumonia in humans, more-

over, is frequently caused by combined viral-bacterial infections (Scheiblaue et al. 1992). Influenza infection can also provoke latent tuberculosis and chronic nephritis to erupt into active cases (Couch 1981).

Consequently, when the whooping cough epidemic broke out in September, its effects would have been increased by preexisting tubercular disease and by the devastating influenza epidemic from which the community was still recovering. Whooping cough affects the respiratory tract, destroys cells in the respiratory lining, and makes it necessary to cough to remove mucous from the lungs. This may have provoked existing lung disease in the form of tuberculosis. Adults already suffering with tuberculosis, in turn, may have been more likely to experience whooping cough. At the very least, adults infected with *Bordatella pertussis* would have infected susceptible children with whom they were in contact. In other words, we are most likely seeing endemic and epidemic diseases interacting synergistically, thereby magnifying the effects of each and increasing the community's disease burden.

But synergies become syndemics when they are underlain by harmful social conditions and injurious social connections. What deleterious social conditions existed in 1927?

York Factory was a dying community. It had lost its strategic importance in the international trade network as the northern sea route from Europe declined and as trade with the US increased, prompting a shift in trade toward the new steamship and railway routes in the south. The surrounding region had never been rich in game and small mammals. After over two hundred years of harvesting, York Factory's fur-bearing mammal resource base was severely depleted. To make matters worse, competition from non-Aboriginal trappers and non-HBC outfits was on the rise. Fluctuating fur prices, periodic supply shortages, and over hunting produced environmental degradation. Medical parties in the 1930s and 1940s identified worrying levels of malnutrition in many parts of the Canadian north, including York Factory (Herring et al. 2003; Herring and Sattenspiel 2007). By the 1920s, the lack of a sustainable economy and the difficulties in living off the land accelerated out-migration to more prosperous places in the south. Erstwhile center of the North American fur trade, York Factory was being abandoned in the early twentieth century as residents migrated to more prosperous locations further south (Beardy and Coutts 1996).

A syndemic perspective on the 1927 whooping cough outbreak makes it possible to see how intertwined epidemics of respiratory disease—tuberculosis, influenza, and whooping cough—were the biological expression and emblem of declining health conditions and growing impoverishment at York Factory in the early twentieth century (Herring and Young 2005).

## References

- ACCA (Anglican Church of Canada Archives, General Synod Office, Toronto, Canada). 1864–1929. York Factory Burials. *Diocese of Keewatin Records*, Ms. 217, Reel #2.

- Anand, N.N. 2005–6. The economic impact of a flu pandemic. *Lab Business*, Winter 2005–2006: 18–21.
- Antonovics, J., M.E. Hood, and C.H. Baker. 2006. Was the 1918 flu avian in origin? *Nature* 440: E9.
- Appenzeller, T. 2005. Tracking the next killer flu. *National Geographic* October: 2–31.
- Armelagos, G. J. 1998. The viral superhighway. *The Sciences* 38: 24–30.
- Barker, T. 2005. The avian flu and the end of the world. *The Manitoban Online* 93 no. 10, October 26.
- Barrett, R., C.W. Kuzawa, T. McDade, and G.J. Armelagos. 1998. Emerging and re-emerging infectious diseases: the third epidemiologic transition. *Annual Reviews of Anthropology* 27: 247–71.
- Barry, J.M. 2004a. *The Great Influenza: the Epic Story of the Deadliest Plague in History*. New York: Viking.
- . 2004b. The site of origin of the 1918 influenza pandemic and its public health implications. *Journal of Translational Medicine* 2(1): 3.
- Basu, P. 2006. Panic over bird flu premature, experts say. *Nature Medicine* 12(3): 258.
- Beardy, E. and R. Coutts. 1996. *Voices from Hudson Bay: Cree Stories from York Factory*. Montreal: McGill-Queen's University Press.
- Burnet, F.M. and E. Clark. 1942. *Influenza*. London: Macmillan.
- Butler, D. 2006. Doubts hang over source of bird flu spread. *Nature* 439: 772.
- Butler, D. and J. Ruttiman. 2006. Avian flu and the New World. *Nature* 441(11): 137–39.
- Cartwright, F.F. 1983. Pandemics past and future. In *Disease in Ancient Man*, ed. Gerald D. Hart. Toronto: Clarke Irwin.
- Cherry, J.D. 1999. Pertussis in the preantibiotic and prevaccine era, with emphasis on adult pertussis. *Clinical Infectious Diseases* 28 Suppl 2: S107–11.
- Cohn, S.K., Jr. 2002. *The Black Death Transformed: Disease and Culture in Early Renaissance Europe*. London: Arnold.
- Couch, R.B. 1981. The effects of influenza on host defences. *Journal of Infectious Diseases* 144(3): 284–91.
- Crosby, A.W. 1989. *America's Forgotten Pandemic: The Influenza of 1918*. Cambridge: Cambridge University Press.
- Dull, H.B. and W.R. Dowdle. 1980. Influenza. In *Maxcy-Rosenau Preventive Medicine and Public Health*, ed. J. Last. New York: Appleton-Century-Crofts.
- Farmer, P. 1999. *Infections and Inequalities: The Modern Plagues*. Berkeley: University of California Press.
- Fox, Daniel M. and Elizabeth Fee. 1988. *AIDS: The Burdens of History*. Berkeley: University of California Press.
- Garrett, L. 2005. The next pandemic? *Foreign Affairs* 84(4): 3–22.
- Gibbs, M.J. and A.J. Gibbs. 2006. Was the 1918 pandemic caused by a bird flu? *Nature* 440: E8.
- GRAIN. 2006. *Fowl Play: The Poultry Industry's Central Role in the Bird Flu Crisis*. GRAIN Briefing, February 2006. [Online] Available: <http://www.grain.org/briefings> [15 March 2006].
- Grmek, M.D. 1990. *History of AIDS: Emergence and Origin of a Modern Pandemic*. Princeton: Princeton University Press.
- Grove, R.D. and A.M. Hetzel. 1968. *Vital Statistics Rates in the United States 1940–1960*. Washington, D.C.: U.S. Government Printing Office.



- Hackett, F.P. 1991. *The 1819–20 Measles Epidemic: Its Origin, Diffusion and Mortality Effects upon the Indians of the Petit Nord*. MA thesis, University of Manitoba, Winnipeg.
- . 2002. *A Very Remarkable Sickness: Epidemics in the Petit Nord, 1670 to 1846*. Winnipeg: University of Manitoba Press.
- HBCA (Hudson's Bay Company Archives, Winnipeg, Canada). 1794–1939. *York Factory Post Journal*, B.239/a/186–96.
- Herring, D.A. 1994. 'There were young people and old people and babies dying every week': the 1918–1919 influenza pandemic at Norway House. *Ethnohistory* 41: 73–105.
- Herring, D.A., S. Abonyi, and R.D. Hoppa. 2003. Malnutrition among northern peoples of Canada in the 1940s: an ecological and economic disaster. In *Human Biologists in the Archives: Demography, Health, Nutrition and Genetics in Historical Populations*, eds. D.A. Herring and A.C. Swedlund. Cambridge: Cambridge University Press.
- Herring, D.A. and R.D. Hoppa. 1999. Endemic tuberculosis among nineteenth century Cree in the central Canadian Subarctic. *Perspectives in Human Biology* 4(1): 189–99.
- Herring, D.A. and J. Padiak. 2008. The geographical epicentre of the 1918 influenza pandemic. In *Proceedings of the Eighth Annual Conference of the British Association for Biological Anthropology and Osteoarchaeology*, eds. M. Brickley and M. Smith. BAR International Series 1743. Oxford: Archaeopress.
- Herring, D.A. and L. Sattenspiel. 2003. Death in winter: Spanish flu in the Canadian subarctic. In *The Spanish Influenza Pandemic of 1918–19: New Perspectives*, eds. H. Phillips and D. Killingray. London and New York: Routledge.
- Herring, D.A. and L. Sattenspiel. 2007. Social contexts, syndemics, and infectious disease in northern Aboriginal populations. *American Journal of Human Biology* 19(2): 190–202.
- Herring, D.A. and A.C. Swedlund, eds. 2003. *Human Biologists in the Archives: Demography, Health, Nutrition and Genetics in Historical Populations*. Cambridge: Cambridge University Press.
- Herring, D.A. and C. Young. 2005. A syndemic perspective on respiratory disease epidemics in the central Canadian subarctic. *American Journal of Human Biology* 17(2): 243.
- Hollenbeck, J.E. 2005. An avian connection as a catalyst to the 1918–1919 influenza pandemic. *International Journal of Medical Science* 2(2): 87–90.
- Ito, T., J.N.S.S. Couceiro, S. Kelm, L. G. Baum, et al. 1998. Molecular basis for the generation in pigs of Influenza A viruses with pandemic potential. *Journal of Virology* (September): 7367–73.
- Joffe, H. 1999. *Risk and The Other*. Cambridge: Cambridge University Press.
- Jordan, E.O. 1927. *Epidemic Influenza: A Survey*. Chicago: American Medical Association.
- Johnson, N.P.A.S. and J. Mueller. 2002. Updating the accounts: global mortality of the 1918–1920 Spanish influenza pandemic. *Bulletin of the History of Medicine* 76: 105–15.
- Jones, E.W. 2005. Co-operation in all human endeavour: Quarantine and immigrant disease vectors in the 1918–1919 influenza pandemic in Winnipeg. *Canadian Bulletin of Medical History (CBMH/BCHM)* 22(1): 57–82.
- Kaplan, M.M. and R.G. Webster. 1977. The epidemiology of influenza. *Scientific American* 237(6): 88–106.
- Kilbourne, E.D. 2006. Influenza pandemics of the 20<sup>th</sup> century. *Emerging Infectious Diseases* 12 (1): 9–14.
- Klempner, M.S. and D.S. Shapiro. 2005. Crossing the species barrier—one small step to man, one giant leap to mankind. *New England Journal of Medicine* 350: 1171–72.

- Kobasa, D., S.M. Jones, K. Shinya, J.C. Kash, et al. 2007. Aberrant innate immune response in lethal infection of macaques with the 1918 influenza virus. *Nature* 445(18): 319–23.
- Last, J. 1999. Fouling and cleansing our nest: human-induced ecological determinants of infectious disease. *Perspectives in Human Biology* 4(1): 145–57.
- Lederberg, J. 1988. Pandemic as a natural evolutionary phenomenon. *Social Research* 55(3): 344–59.
- Lee, S., L.Y.Y. Chan, A.M.Y. Chau, K.P.S. Kwok, and A. Kleinman. 2005. The experience of SARS-related stigma at Amoy Gardens. *Social Science & Medicine* 61: 2038–46.
- Li, K.S., Y. Guan, J. Wang, et al. 2004. Genesis of a highly pathogenic and potentially pandemic H5N1 influenza virus in eastern Asia. *Nature* 430: 209–13.
- Linder, F.E. and R.D. Grove. 1947. *Vital Statistics Rates in the United States 1900–1940*. Washington, D.C.: U.S. Government Printing Office.
- Lockerbie, S. and D.A. Herring. In press. Global panic, local repercussions: economic and nutritional effects of bird flu in Vietnam. In *Anthropology and Public Health: Bridging Differences in Culture and Society*, 2<sup>nd</sup> ed., eds. R. Hahn and M. Inhorn. Oxford: Oxford University Press.
- Lux, M. 1997. The Bitter Flats: The 1918 influenza epidemic in Saskatchewan. *Saskatchewan History* 49(1): 3–13.
- MacDougall, H. 1985. The fatal flu. *Horizon Canada* 8(8): 2089–95.
- Mamelund, S-V. 2006. A socially neutral disease? Individual social class, household wealth and mortality from Spanish influenza in two socially contrasting parishes in Kristiania 1918–19. *Social Science & Medicine* 62: 923–40.
- McCullers, J.A. and J.E. Rehg. 2002. Lethal synergism between influenza virus and *Streptococcus pneumoniae*: characterization of a mouse model and the role of platelet-activating factor receptor. *Journal of Infectious Diseases* 186: 341–50.
- Monto, A.S. 2005. The threat of an avian influenza pandemic. *New England Journal of Medicine* 352(4): 323–25.
- Morse, S.S. 1993. Examining the origins of emerging viruses. In *Emerging Viruses*, ed. S.J. Morse. Oxford: Oxford University Press.
- Noymer, A. 2006. *Testing the influenza tuberculosis selective mortality hypothesis with Union Army data*. [Online] Available: <http://www.paa2006.princeton.edu/abstractViewer.aspx?submissionId=61257>. [20 May, 2006].
- Noymer, A. and M. Garenne. 2000. The 1918 influenza epidemic's effects on sex differentials in mortality in the United States. *Population and Development Review* 26(3): 565–81.
- . 2003. Long-term effects of the 1918 'Spanish' influenza epidemic on sex differentials of mortality in the USA. In *The Spanish Influenza Pandemic of 1918–19: New Perspectives*, eds. H. Phillips and D. Killingray. London and New York: Routledge.
- Omran, A.R. 1971. The epidemiologic transition: a theory of the epidemiology of population change. *Millbank Memorial Fund Quarterly* 42(4): 509–37.
- Osterholm, M.T. 2005. Preparing for the next pandemic. *Foreign Affairs* 84(4): 24–37.
- Oxford, J.S., A. Sefton, R. Jackson, W. Innes, et al. 2002. World War I may have allowed the emergence of Spanish influenza. *Lancet—Infectious Disease* 2(2): 111–14.
- Oxford, J.S., R. Lambkin, A. Sefton, R. Daniels, et al. 2005. A hypothesis: the conjunction of soldiers, gas, pigs, ducks, geese and horses in Northern France during the Great War provided the conditions for the emergence of the Spanish influenza pandemic of 1918–1919. *Vaccine* 23: 940–45.

- Palese, P. 1993. Evolution of influenza and RNA viruses. In *Emerging Viruses*, ed. S.J. Morse. Oxford: Oxford University Press.
- Phillips, H. 2004. The re-appearing shadow of 1918: trends in the historiography of the 1918–19 influenza pandemic. *Canadian Bulletin of Medical History (CBMH/BCMh)* 21(1): 121–34.
- Ray, A.J. 1974. *Indians in the Fur Trade: Their Role as Hunters, Trappers and Middlemen in the Lands Southwest of Hudson Bay, 1660–1870*. Toronto: University of Toronto Press.
- Reid, A.H., T.G. Fanning, J.V. Hultin, and J.K. Taubenberger. 1999. Origin and evolution of the 1918 Spanish influenza virus hemagglutinin gene. *Proceedings of the National Academy of Science* 96: 1651–56.
- Rosenberg, C. 1992. *Explaining Epidemics*. Cambridge: Cambridge University Press.
- Rosenthal, E. 2006. Human-to-human infection of bird flu virus is confirmed. *New York Times*, June 24: A8.
- Scheiblaue, H., M. Reinacher, M. Tashiro, and R. Rott. 1992. Interactions between bacteria and influenza A virus in the development of influenza pneumonia. *Journal of Infectious Diseases* 166: 783–91.
- Scholtissek, C. 1992. Cultivating a killer virus. *Natural History* 1: 3–6.
- . 1994. Source for influenza pandemics. *European Journal of Epidemiology* 4(10): 455–58.
- Shinya, K., M. Ebina, S. Yamada, M. Ono, et al. 2006. Avian flu: influenza virus receptors in the human airway. *Nature* 440: 435–36.
- Singer, M. and S. Clair. 2003. Syndemics and public health: reconceptualizing disease in bio-social context. *Medical Anthropology Quarterly* 17(4): 423–41.
- Snacken, R., A.P. Kendal, L.R. Haaheim, and J.M. Wood. 1999. The next influenza pandemic: lessons from Hong Kong 1997. *Emerging Infectious Diseases* 5(2) [Online]. Available: <http://www.cdc.gov/pub/EID/vol4no3/ascii/webs.txt> [7 March 1999].
- Stewart, D.A. 1936. The Red Man and the White Plague. *Canadian Medical Association Journal* 35: 674–76.
- Stöhr, K. 2005. Avian influenza and pandemics—research needs and opportunities. *New England Journal of Medicine* 352(4): 405–7.
- Stone, E.L. 1926. Health and disease at the Norway House Indian Agency. Reprinted in *Native Studies Review* 5(1), 1989: 237–56.
- Taubenberger, J.K. and D.M. Morens. 2006. 1918 influenza: the mother of all pandemics. *Emerging Infectious Diseases* 12(1): 15–22.
- Taubenberger, J.K., A.H. Reid, A.E. Krafft, K.E. Bijwaard, and T.G. Fanning. 1997. Initial genetic characterization of the 1918 Spanish influenza virus. *Science* 275(5307): 1793–96.
- Taubenberger, J.K., A.H. Reid, R.M. Lourens, R. Wang, et al. 2005. Characterization of the 1918 influenza virus polymerase genes. *Nature* 437(7060): 889–93.
- . 2006. Taubenberger et al. Reply. *Nature* 440: E9–10.
- Tomes, N. 2000. The making of a germ panic, then and now. *American Journal of Public Health* 90(2): 191–98.
- Ungar, S. 1998. Hot crises and media reassurance: a comparison of emerging diseases and Ebola Zaire. *The British Journal of Sociology* 49(1): 36–56.
- U.S. Department of Health and Human Services. Pandemics and pandemic threats since 1900. [Online]. Available: <http://www.PandemicFlu.gov> [23 May 2006].
- Wade, N. 2006. Studies suggest avian flu pandemic isn't imminent. *New York Times*: 23 March.

- Walters, J.H. 1978. Influenza 1918: the contemporary perspective. *New York Academy of Medicine* 54(9): 855–64.
- Waltner-Toews, D. 1995. Changing patterns of communicable disease—who's turning the kaleidoscope? *Perspectives in Biology & Medicine* 39: 43–55.
- Washer, P. 2004. Representations of SARS in the British newspapers. *Social Science & Medicine* 59: 2561–71.
- . 2006. Representations of mad cow disease. *Social Science & Medicine* 62: 456–66.
- Webster, R.G. 1993. Influenza. In *Emerging Viruses*, ed. S.J. Morse. Oxford: Oxford University Press.
- . 1998. Influenza: an emerging disease. *Emerging Infectious Diseases* 4(3): 436–41.
- World Health Organization (WHO). 2006a. Cumulative number of confirmed human cases of avian influenza A/(H5N1) reported to WHO. [Online]. Available: [http://www.who.int/csr/disease/avian\\_influenza/country/cases\\_table\\_2006\\_02\\_02/en/index.html](http://www.who.int/csr/disease/avian_influenza/country/cases_table_2006_02_02/en/index.html) [1 February 2006].
- . 2006b. Epidemiology of WHO-confirmed human cases of avian influenza A (H5N1) infection. *Weekly Epidemiological Record* 26, 81: 249–60.
- . 2008a. Areas reporting confirmed occurrence of H5N1 avian influenza in poultry and wild birds since 2003. [Online]. Available: [http://gamapserv.who.int/mapLibrary/Files/Maps/Global\\_SubNat\\_H5N1inAnimalConfirmedCUMULATIVE\\_20080303.png](http://gamapserv.who.int/mapLibrary/Files/Maps/Global_SubNat_H5N1inAnimalConfirmedCUMULATIVE_20080303.png) [7 March 2008].
- . 2008b. Cumulative number of confirmed human cases of avian influenza A/(H5N1) Reported to WHO. [Online]. Available: [http://www.who.int/csr/disease/avian\\_influenza/country/cases\\_table\\_2008\\_02\\_20/en/index.html](http://www.who.int/csr/disease/avian_influenza/country/cases_table_2008_02_20/en/index.html) [28 February 2008].

