



Breeding Influenza: The Political Virology of Offshore Farming

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Abstract: The geographic extent, xenospecificity, and clinical course of influenza A (H5N1), the bird flu strain, suggest the virus is an excellent candidate for a pandemic infection. Much attention has been paid to the virus's virology, pathogenesis and spread. In contrast, little effort has been aimed at identifying influenza's social origins. In this article, I review H5N1's phylogeographic properties, including mechanisms for its evolving virulence. The novel contribution here is the attempt to integrate these with the political economies of agribusiness and global finance. Particular effort is made to explain why H5N1 emerged in southern China in 1997. It appears the region's reservoir of near-human-specific recombinants was subjected to a phase change in opportunity structure brought about by China's newly liberalized economy. Influenza, 200 nm long, seems able to integrate selection pressures imposed by human production across continental distances, an integration any analysis of the virus should assimilate in turn.

Keywords: influenza, phylogeography, virulence, agribusiness, poultry intensification, AgFDI

Introduction: Panic in the City

Hong Kong, March 1997. An outbreak of deadly bird flu sweeps through poultry on two farms. The outbreak fizzles out, but 2 months later a 3-year-old boy dies of the same strain, identified as a highly pathogenic version of influenza A (H5N1). Officials are shocked. This appears the first time such a strain has jumped the species barrier and killed a human. Shocking too, the outbreak proves persistent. In November a 6-year-old is infected, recovering. Two weeks later, a teenager and two adults are infected. Two of the three die. Fourteen additional infections rapidly follow.

The deaths spur panic in the city and, with the onset of the regular flu season, send many patients to the hospital worried their symptoms might be those of the new flu. By mid-December poultry begin to die in droves in the city's markets and it now seems most humans infected had handled birds. Hong Kong acts decisively on that information. Authorities order the destruction of all of Hong Kong's 1.5 million poultry and block new imports from Guangdong, the mainland province across the Shenzhen River from which some of the infected birds had been transported. Despite another human death in January, the outbreak is broken.

The poultry infected with this version of the virus suffer more than the gastrointestinal condition typical of avian influenza. The clinical

manifestations include swelling of the wattles and infraorbital sinuses, congestion and blood spots on the skin of the hocks and shanks, and a blue discoloration of the comb and legs (Yuen and Wong 2005). The latter is characteristic of the cyanosis and oxygen deprivation suffered by many human victims of the 1918 pandemic. Internally, infected poultry are marked by lesions and hemorrhaging in the intestinal tract and the trachea, with blood discharge from the beak and cloaca. Many birds also suffer infection in other organs, including the liver, spleen, kidney, and the brain, the last infection leading to ataxia and convulsion.

Most worrisome for human health is this strain's capacity for broad xenospecific transmission. The Hong Kong outbreak, first alerting the world to H5N1, infected humans with an influenza much more pathogenic than the relatively mild infections of other avian outbreaks that have intermittently crossed over into human populations. These patients presented with high fever, later developing some combination of acute pneumonia, influenza-like illness, upper respiratory infections, conjunctivitis, pharyngitis, and a gastrointestinal syndrome that included diarrhea, vomiting, vomiting blood, and intestinal pain (Buxton Bridges et al 2000; de Jong et al 2006). Patients also suffered multiple-organ dysfunction, including that of the liver, kidney, and bone marrow. The respiratory attacks involved extensive infiltration of both lungs, diffuse consolidation of multiple infected loci, and lung collapse.

If much of H5N1's morbidity is distressing, its associated mortality is alarming. Once infected, the lungs' vasculature becomes porous and fibrinogen—a protein involved in blood clotting—leaks into the lungs (de Jong et al 2006). The resulting fibroblast exudates clog the lungs' alveolar sacs, where gas exchange takes place, and an acute respiratory disease syndrome results. In a desperate effort to save its charge, the immune system recruits such a storm of cytokines that the lungs suffer oedema. In effect, patients drown in their own fluid only days after infection.

After its first strike in Hong Kong, H5N1 slipped underground with outbreaks largely limited to birds in southern China. The virus underwent the first of a series of reassortment events, in which several genomic segments were replaced with those from other serotypes, before reemerging as a human infection in Hong Kong in 2002 (Li et al 2004; Webster et al 2006). The following year H5N1 again reemerged, this time with a vengeance. The Z genotype that surfaced as the dominant recombinant spread across China, into Vietnam, Thailand, Indonesia, Cambodia, Laos, Korea, Japan, and Malaysia. Two additional strains would subsequently materialize. Since 2005 the Qinghai-like strain (H5N1 hemagglutinin clade 2.2) has spread across Eurasia, as far west as England, and into Africa (Salzberg et al 2007). The Fujian-like strain (clade 2.3), emerging from its eponymous southern China province, has

spread regionally across Southeast Asia and, more recently, into Korea and Japan (Smith et al 2006).

Since 2003 H5N1 has infected 440 people, killing 262 (WHO, August 2009). Most of these infections have been poultry related; often the children of small farmers playing with a favorite bird. But documented cases of human-to-human transmission have accumulated—in Hong Kong, Thailand, Vietnam, Indonesia, Egypt, China, Turkey, Iraq, India, and Pakistan (Kandun et al 2006; Yang et al 2007). The short chains of transmission have largely consisted of relatives living with or tending a patient. The worry, well publicized, is that H5N1 will improve upon these first infections, evolving a human-to-human phenotype that ignites a worldwide pandemic along the lines of this year's swine flu but deadlier in its manifestation.

The geographic diffusion of the virus is intimately related to the emergence of such a phenotype. As are other pathogens, H5N1 is finding the regions of the world where animal health surveillance remains underdeveloped or degraded by structural adjustment programs associated with international loans or neoliberal trade agreements (Rweyemamu et al 2000). There is now, in addition, greater integration of stockbreeding, aquaculture and horticulture, a burgeoning live-bird market system, and widespread proximity to poultry (Cristalli and Capua 2007; Gilbert et al in press). Rural landscapes of many of the poorest countries are now characterized by unregulated agribusiness pressed against periurban slums (Fasina et al 2007; Guldin 1993). Unchecked transmission in vulnerable areas increases the genetic variation with which H5N1 can evolve human-specific characteristics. In spreading over three continents fast-evolving H5N1 also contacts an increasing variety of socioecological environments, including locale-specific combinations of prevalent host types, modes of poultry farming, and animal health measures.

In this way, by a type of escalating demic selection, H5N1 can better explore its evolutionary options (Wallace and Wallace 2003). A series of fit variants, each more transmissible than the next, can evolve in response to local conditions and subsequently spread. The Z reassortant, the Qinghai-like strain, and the Fujian-like strain all outcompeted other local H5N1 strains to emerge to regional and, for the Qinghai-like strain, continental dominance. The more genetic and phenotypic variation produced across geographic space, the more compressed the time until a human infection evolves.

How did we get into this fix? Why this deadly disease now? In a torrent rivaling the research conducted on the central mystery of Stanislaw Lem's *Solaris*, thousands of reports have been published on the virus's molecular structure, genetics, virology, pathogenesis, host biology, clinical course, treatment, modes of transmission, phylogenetics, and geographic spread. That body of work, much of it riveting, appears

predicated on a molecular narrative that portrays disease largely in terms of a conflict between virion and immunity, between viral evolution and humanity's capacity to produce adequate vaccines and antivirals, between nature red in glycoprotein and nurture white in lab coat (Braun 2007). Paradigms compete and in investing in one narrative—perhaps because of its political, commercial or institutional benefits—other explanations suffer. Some of the most basic questions about bird flu's nature appear lost in the blizzard of micrographs, sequence alignments, tertiary solution structures, SIR models, antigenic cartograms, and phylogenetic dendrograms. What of the virus's greater context?

Castree (2008a, 2008b) recently reviewed a new literature aimed at addressing just such context. The literature, at this point largely a loose affiliation of case studies, tracks the ways the present class of models of globalized finance and production, which structure so much of humanity's daily life, are embodied in the control and exploitation of nonhuman systems. The work traces the means by which nature is "neoliberalized". To Castree's examples—water management, fisheries, logging, mining, plant and animal genomics, and greenhouse gas emissions—we can add agriculture, breeding programs, and pharmaceutical excavation. This article represents another example, although it travels along something of an orthogonal direction. I review influenza as a case study of the inadvertent biotic fallout of efforts aimed at steering animal ontogeny and ecology to multinational profitability (Benton 1989).

Here I will explore the social origins of highly pathogenic influenza A (H5N1) and, as best as one can given the present literature, connect these with the evolution and spread of the virus. I will first review key concepts in pathogen virulence and diversification. I will hypothesize the means by which influenza's present virulence and diversity arose out of the Livestock Revolution. In the context of a now-globalized poultry, I will next explore a fundamental question so far ill-addressed, surprisingly so given the amount of work dedicated to characterizing the virus: why did pathogenic H5N1 evolve in southern China? Moreover, why did it do so in 1997? Locating bird flu virulence in China's poultry intensification efforts is one matter. Outbreak persistence there and elsewhere, however, is another: I will also review complications in influenza epidemiology apparent beyond the factory gate. Finally, I will propose a broad albeit preliminary program of intervention that extends beyond the provisional fiddling typically operationalized during each outbreak season. Along the way I will pursue epistemological aspirations. In bridging disciplines, I aim for an evolutionary virology that integrates humanity's impact on pathogen evolution from the very start of any investigation.

To begin, I explore bird flu's deadliness beyond listing molecular mechanisms by which the virus transforms cells into progeny, as important as these are.

Growing Deadly Influenza

Despite its impacts epidemiological and psychological, Hong Kong's H5N1 represented no first outbreak of bird flu. In fact, within the United States alone, where highly pathogenic H5N1 has not yet spread, a series of outbreaks have accrued over the past decade (Davis 2005). These outbreaks were typically low pathogenic, causing lesser damage to poultry. There was, however, an outbreak of highly pathogenic H5N2 in Texas in 2002. A low pathogenic H6N2 outbreak in California, beginning in large farms outside San Diego, evolved greater virulence as it spread through California's Central Valley. Another outbreak worthy of note is that of a low pathogenic strain of H5N1 in Michigan in 2002. H5N1, then, has already invaded the United States in a less deadly form, and with different internal genes, telling us that the molecular identity of a strain is insufficient for defining the danger of any single outbreak. Low and high pathogenic strains must be distinguished otherwise. Some mechanism must transform low pathogenic strains into more virulent ones (and, we should hope, back again).

The damage caused by pathogenic influenza may be in part due to an antigenic shift to which susceptible populations presently have no immunity. Humans, for instance, have this past century been infected almost exclusively by H1, H2, and H3 strains to which we eventually developed antibody memory. When many of us are confronted by a seasonal variant of these same types we can slow down the infection. We have partial immunity at the individual level and herd immunity at the population level. But as we have never been exposed to H5 infections *en masse* we have nothing to slow down infection within each person and nothing to keep it dampened down across the population. What cannot be slowed down arrives earlier. It is likely then that, as was the case for the 1957 and 1968 pandemics, the main waves of the next human influenzas will sweep the planet earlier than the typical flu season, with swine flu (2009) as early as August this year or, if another strain, some terrible year in the near future (Cliff, Haggett and Ord 1986).

But how are we to account for an increase in virulence *within* a particular flu subtype? Recall the low pathogenic strain of H5N1 in Michigan. And in something of the other direction, there is the macabre sight of H5N1-devastated waterfowl, which typically act as the natural (and unharmed) reservoir for multiple H5 strains. Another explanation leans on a large modeling literature (reviewed by Dieckmann et al 2002; Ebert and Bull 2008) that hypothesizes a relationship between the rate of transmission and the evolution of virulence, the amount of damage

a strain causes its host. Simply put, to start, there is a cap on pathogen virulence. Pathogens must avoid evolving the capacity to incur such damage to their hosts that they are unable to transmit themselves. If a pathogen kills its host before it infects the next host it destroys its own chain of transmission. But what happens when the pathogen “knows” that the next host is coming along much sooner? The pathogen can get away with being virulent because it can successfully infect the next susceptible in the chain before it kills its host. The faster the transmission rate, the lower the cost of virulence.

A key to the evolution of virulence is the supply of susceptibles (Lipsitch and Nowak 1995). As long as there are enough susceptibles to infect, a virulent phenotype can work as an evolutionary strategy. When the supply runs out it does not matter what virulence a pathogen has evolved. Time is no longer on the particular strain’s side. A failed supply of susceptibles, drained by high mortality or rebound immunity, forces all influenza epidemics to ultimately burn out at some point. That is, of course, cold comfort if millions of people are left dead in a pandemic’s wake.

Given the explanation, what circumstances changed the relationship between virus and host in such a way as to ramp up H5N1 to breathtaking virulence? Growing circumstantial evidence points to intensive poultry production or, in the more critical lexicon, factory farming (FAO 2004; Graham et al 2008; Greger 2006; Shortridge 2003a; US Council for Agriculture, Science and Technology 2005). Capua and Alexander (2004), reviewing recent influenza outbreaks worldwide, found no endemic highly pathogenic strains in wild bird populations, the ultimate source reservoir of nearly all influenza subtypes. Instead, multiple low pathogenic influenza subtypes in such populations developed greater virulence only once they entered populations of domestic birds. While domestic populations can be divided into backyard and industrial, the former have been raised in one form or another for centuries without the now unprecedented outburst of newly pathogenic influenzas.

The conditions for supporting such strains appear best represented in industrial poultry. Graham et al (2008) found significantly greater odds for H5N1 outbreaks in Thailand 2004 in large-scale commercial poultry operations than in backyard flocks. The pattern is repeated across influenza serotypes. In British Columbia in 2004, 5% of the province’s large farms hosted highly pathogenic H7N3 infections, while 2% of its small farms hosted outbreaks (Otte et al 2007). In the Netherlands in 2003, 17% of industrial farms hosted H7N7 outbreaks, while 0.1% of backyard farms hosted clusters.

Even if these and other such strains first developed on smallholdings, a possibility to which we will return, industrial livestock appear ideal populations for supporting virulent pathogens. Growing genetic monocultures of domestic animals removes whatever immune firebreaks

may be available to slow down transmission (Garrett and Cox 2008). Larger population sizes and densities facilitate greater rates of transmission. Such crowded conditions depress immune response. High throughput, a part of any industrial production, provides a continually renewed supply of susceptibles, the fuel for the evolution of virulence.

There are additional pressures on influenza virulence on such farms. As soon as industrial animals reach the right bulk they are killed. Resident influenza infections must reach their transmission threshold quickly in any given animal, before the chicken or duck or pig is sacrificed. The quicker viruses are produced, the greater the damage to the animal. Increasing age-specific mortality in industrial livestock should select for greater virulence. With innovations in production, the age at which chickens are processed has been reduced from 60 to 40 days (Striffler 2005), increasing pressure on viruses to reach their transmission threshold—and virulence load—that much faster.

A similar trajectory for the evolution of virulence has been superbly described for efforts at mitigating H5N1 outbreaks by mass culling (Shim and Galvani 2009): the greater the culling, the more pressure on the virus to evolve virulence. The model, however, misses where the virulence of a virus that requires culling arises in the first place. Industrial livestock production comprises little more than continuous culling. The resulting influenzas, expected to transmit out of younger and younger animals, are not only more virulent but able to grow in the face of a host population's more robust immune systems. In short, with a simple host switch, we find here a recipe for the emergence of a deadly pandemic targeting 15–45 year olds.

Although no smoking guns at present match the emergence of specific strains of deadly H5N1 to specific livestock farms, a growing phylogenetic literature fails to refute the working hypothesis. Duan et al (2007) identified low pathogenic relatives of highly pathogenic H5N1 in migratory birds, lineages dating as far back as the 1970s. None of the recently emergent low pathogenic H5 relatives became established in aquatic or terrestrial poultry. In contrast, the origins of recent H5 virulence appear characteristic of domestic poultry alone. Vijaykrishna et al (2008) meanwhile showed the source 1996 Guangdong strain entered regional poultry with all eight genomic segments intact. The subsequent diversification into multiple genotypes, including the deadly Z genotype that has dominated outbreaks since 2003, occurred in domestic ducks in China mid 1999 to 2000.

Much work, however, remains to be done. The phylogeny work continues apace, focusing on finer geographic scales across better inventoried landscapes. Research now under development tracks the evolution of H5N1 across Eurasia and Africa in niche envelopes defined by combinations of a number of agro-ecological variables, including

levels of poultry intensification (eg Cecchi et al 2008). A recent international conference of H5N1 scientists convened in Bangkok laid out a research program aimed at better integrating phylogeographic studies of the virus with geocoded value chain analyses of agricultural production.

In parallel, a growing number of studies are zeroing in on the seroepidemiology of poultry-dense regions of southern China, H5N1's putative epicenter, including within specific production plants. Lu et al (2009) showed Guangdong hosted a variety of influenzas. Seasonal influenzas H1N1 and H3N2 comprised most of the 1214 human cases discovered. But antibodies for H5N1 (2.5%) and H9N2 (4.9%) were also found among all tested, with a significantly greater prevalence of H9N2 antibodies (9.5%) in those occupationally exposed to birds. Wang, Fu and Zheng (2009) meanwhile detected very few cases of H5 among 2191 Guangzhou workers occupationally exposed to birds. H9, on the other hand, appeared widespread across the poultry commodity chain, especially among poultry market retailers (15.5%) and wholesalers (6.6%) and workers in large-scale poultry-breeding enterprises (5.6%). By way of explanation, H5 are targeted by vaccination campaigns, while H9 are generally not. Retailers handle different poultry species from multiple wholesalers, while wholesalers handle their own lots alone. Finally, Zhang et al (2008) followed H9N2 outbreaks over 5 years in a single broiler chicken operation in Shanghai. Virus across all outbreaks in the plant appeared related to that of the first outbreak despite vaccination efforts. The H9N2 isolates, with internal loci that arose via local reassortment with H5N1, showed evolution by antigenic drift across the study period. In short, meaningful advances are being made connecting the particulars of human production and influenza's spread and evolution.

Industrial production has already been implicated in increasing the diversity of human-friendly influenza. Over the past 15 years an unprecedented variety of influenzas capable of infecting humans have emerged across the global archipelago of industrial farms. Along with H5N1 there are now swine flu H1N1, H7N1, H7N3, H7N7, H9N2, in all likelihood H5N2, and perhaps even some of the H6 serotypes (Meyers et al 2007; Ogata et al 2008; Puzelli et al 2005; WHO 2005). A feedback appears to have emerged in kind: the very efforts pursued to control pathogenic bird flu may in passing increase viral diversification and persistence. In late 2006, virologist Guan Yi and his colleagues at the University of Hong Kong identified the previously uncharacterized Fujian-like H5N1 lineage (Smith et al 2006). The team ascribed the emergence of the strain as a viral evolutionary reaction to the Chinese government's campaign to vaccinate poultry. As in the case of other influenza serotypes (Escorcía et al 2008; Suarez, Lee and Swayne 2006),

the virus appeared to evolve out from underneath the pressure of vaccine coverage.

Factory practices provide what seems to be an amenable environment for the evolution of a variety of virulent influenzas, including pandemic strains. Swine flu H1N1, the most recent example arising early 2009 and on which we will touch only in passing, appears by definition industrial in origin. The closest ancestor for each of this H1N1's eight genomic segments is of swine origin. The segments have been identified as originating from different parts of the world: neuraminidase and the matrix protein from strains circulating in Eurasia, the other six from North America. No small farmer has the industrial capacity necessary to export livestock of any consequence across such long distances, nor the market entree livestock influenzas need to spread through international commodity chains.

If swine H1N1 or any subsequent human-specific influenza proves deadly, the epidemiological pollution, embodying O'Connor's (1998) "second contradiction", threatens the very existence of the livestock industry. But it seems to be a risk agribusiness is willing to weather for the immediately cheap manufacture of its products.

Exporting the Tyson Model

In Israel researchers recently selected for a lineage of featherless chickens (Yaron, Hadad and Cahaner 2004). At first glance one suffers a Latourian shock at how much the naked birds look like living groceries. Able to survive solely in warm climes, the chickens were developed in the interests of the producer, not the consumer. Consumers have long avoided plucking feathers, a step typically conducted at the factory. A featherless poultry will allow producers, on the other hand, to scratch off plucking from production. The bald bird offers the anatomical equivalent of the factory epidemiology agribusiness is imposing on poultry—generating artificial ecologies that could never persist in nature because of the disease costs they incur, but that allow more poultry to be processed faster. The resulting costs are shifted to the birds, of course, but also consumers, farm workers, taxpayers, local governments, and nearby wildlife.

The lengths to which agribusiness has changed livestock production are remarkable, including, more recently, in the present bird flu zone. Southern China serves as a regional incubator for new methods in poultry breeding (Luo, Ou and Zhou 2003). Sun et al (2007), for instance, describe a Guangdong program in which geese were exposed to a counter-seasonal lighting schedule that induced out-of-season egg laying. The innovation helped double profits for local goose production and expanded the market, and Chinese appetite, for goose meat. The resulting market advantages forced smaller farms out of business and

led to a consolidation of the province's agribusiness. The structural shift marks a perverse turn back toward the farm collectivization the Chinese government abandoned in 1980, this time, though, under the control of far fewer hands. As the result of such innovations, to which we will return, millions more birds have been put into production there.

Karl Marx (1990 [1867]) traced many of the fundamentals of such efforts at commodification. In the first chapter of the first volume of *Capital* Marx wrote that human-made objects have multiple characteristics. They have use value—a hammer can be used to beat down nails. In all human economies objects also sport an exchange value—how many other objects (say, screwdrivers) for which a hammer can be exchanged. A capitalist economy adds a third characteristic, turning objects into commodities. Surplus value is that part of the object's worth that accrues to capitalists as profit. Marx's contribution was showing that capitalists expropriate the surplus value by taking it out of the value that workers added to the commodity when they made it, usually by paying workers lower wages or increasing worker productivity, paying them the same or less for more work.

This is, of course, a rudimentary presentation of Marx's theory, and others have better elaborated on its applications to organisms and their ecologies (eg Castree 2008a; Foster 2000; Heynan et al 2007; Kovel 2002). But in these first efforts to better relate influenza's evolution to its social context we will address only the most general of Marx's points, namely that capitalists produce commodities not because commodities are useful—have use value—but because they accrue surplus value, to capitalists the most important characteristic of the object. Changing the color or style of a hammer to attract more consumers may seem negligible in effect, but for other objects changes in use value can have far-reaching, even dangerous, consequences. In this case, agribusiness has changed its commodity—living, breathing organisms—to maximize productivity. But what does it mean to change the use value of the creatures we eat? What happens when changing use value turns our poultry into plague carriers? Does out-of-season goose production, for instance, allow influenza strains to avoid seasonal extirpation, typically a natural interruption in the evolution of virulence? Are the resulting profits defensible at such a rapidly accruing cost?

Mass commodification of poultry emerged in what is now called the "Livestock Revolution". Before the shift, poultry was largely a backyard operation. In Boyd and Watts's (1997) map of poultry across the United States in 1929, each dot represents 50,000 chickens. We see wide dispersion across the country—300 million poultry total at an average flock size of only 70 chickens. The production filiere of that era shows local hatcheries sold eggs to backyard poultry producers and independent farmers, who in turn contracted independent truckers to bring live poultry to city markets.

That changed after World War II. Tyson, Holly Farms, Perdue, and other companies vertically integrated the broiler filiere, buying up other local producers and putting all nodes of production under each company's roof (Manning and Baines 2004; Striffler 2005). Boyd and Watts show by 1992 US poultry production is largely concentrated in the South and parts of a few other states. Each of their map's dots now represents 1 million broilers, 6 billion in total, with an average flock size of 30,000 birds. A 2002 map reproduced by Graham et al (2008) shows a similar geographic distribution but 10 years later hosting 3 billion more broilers. US hog and pig populations have similarly exploded in size, particularly over the past 15 years, and are now largely concentrated in North Carolina, Iowa, Minnesota, and parts of other Midwest states.

By the 1970s the new production model was so successful it was producing more poultry than people typically ate. How many roasted chickens were families prepared to eat a week? With the assistance of food science and marketing the poultry industry repackaged chicken in a mind-boggling array of new products, including chicken nuggets, strips of chicken for salads, and cat food. Multiple market shares were developed large enough to absorb the value-added production both domestically and abroad. The USA was for many years the world's leading poultry exporter.

Industrial poultry has since spread geographically. With production widespread, annual world poultry meat increased from 13 million tons in the late 1960s to about 62 million by the late 1990s, with the greatest future growth projected in Asia (FAO 2003). In the 1970s, Asia-based companies such as Charoen Pokphand (CP) set up vertical filieres in Thailand and, soon after, elsewhere in the region. Indeed, CP was the very first foreign company allowed to set up production in Guangdong under Deng Xiaoping's economic reforms. China has since hosted a veritable explosion in annual chickens and ducks produced (Gilbert et al in press). Increases in poultry have also occurred throughout Southeast Asia, though not nearly at the magnitude of that of China.

According to political economist David Burch (2005), the shift in the geography of poultry production has some interesting consequences. Yes, agribusinesses are moving company operations to the global South to take advantage of cheap labor, cheap land, weak regulation, and domestic production hobbled in favor of heavily subsidized agro-exporting (Manning and Baines 2004; McMichael 2006). But companies are also engaging in sophisticated corporate strategy. Agribusinesses are spreading their production line across much of the world. For example, the CP Group, now the world's fourth largest poultry producer, has poultry facilities in Turkey, China, Malaysia, Indonesia, and the USA. It has feed operations across India, China, Indonesia, and Vietnam. It owns a variety of fast food chain restaurants throughout Southeast Asia.

Such rearrangements falsify the widely promulgated assumption that the market can correct corporate inefficiencies. On the contrary, vertical multinationalism cushions companies from the consequences of their own mistakes. First, multinationals producing by way of economies of scale can price unprotected local companies out of business—the Wal-Mart effect. Consumers have nowhere else to go to punish subsequent corporate blunders. Second, by threatening to move operations abroad multinationals can control local labor markets; hobbling unions, blocking organization drives, and setting wages and working conditions. Unions are an important check on production practices that affect not only workers and consumers, but both directly and by proxy the animals involved in production. Third, vertical agribusiness acts as both poultry supplier and retailer. The CP Group, for instance, owns a number of fast food chains in a number of countries selling, what else, CP chicken. In short, fewer independent retailers exist to play suppliers off each other in a way that assures demands for better treatment of livestock are met.

In operating factories across multiple countries multinationals can hedge their bets in a variant of David Harvey's (2006 [1982]) spatial fix. The CP Group operates joint-venture poultry facilities across China, producing 600 million of China's 2.2 billion chickens annually sold (Burch 2005). When an outbreak of bird flu occurred in a farm operated by the CP Group in Heilongjiang Province, Japan banned poultry from China. CP factories in Thailand filled the market gap by increasing exports to Japan. A supply chain arrayed across multiple countries increases the risk of influenza spread even as it allows some companies the means by which to compensate for the resulting interruptions in business (Manning, Baines and Chadd 2007; Sanders 1999).

To protect the interests of agribusiness even as its operations struggle or fail, multinationals also fund politicians or field their own candidates. Telecommunications tycoon Thaksin Shinawatra, the Prime Minister of Thailand during the country's first bird flu outbreaks, came to power promising to run the country like a business, a promise on which he delivered (Phongpaichit and Baker 2004). Shinawatra's policies were at times hard to distinguish from the business plans of the Thai industries that supported him, including agribusiness. His administration played a prime role in blocking Thai efforts to control bird flu. As Mike Davis (2005) describes it, when outbreaks began in Thailand, corporate chicken-processing plants accelerated production. According to trade unionists, processing increased at one factory from 90,000 to 130,000 poultry daily, even as it was obvious many of the chickens were sick. Once the Thai press reported on the illness, Thailand's Deputy Minister of Agriculture made vague allusions to an "avian cholera" and Shinawatra and his ministers publicly ate chicken in a show of confidence.

It later emerged that the CP Group and other large producers were colluding with government officials to pay off contract farmers to keep quiet about their infected flocks. In turn, livestock officials secretly provided corporate farmers vaccines. Independent farmers, on the other hand, were kept in the dark about the epidemic, and they and their flocks suffered for it (Delforge 2007:endnote 25). Once the cover-up was exposed, the Thai government called for a complete modernization of the industry, including requiring all open-air flocks exposed to migratory birds be culled in favor of production in new biosecure buildings only wealthier farmers could afford.

Attempts to proactively change livestock production in the interests of stopping pandemic influenza can be met with severe resistance by governments beholden to corporate sponsors. In effect, influenzas such as H5N1, by virtue of their association with agribusiness, have some of the most powerful representatives available defending their interests in the halls of government. In covering up outbreaks to protect quarterly profits, these institutions contribute to the viruses' evolutionary fortunes. The very biology of influenza is enmeshed with the political economy of the business of food.

If multinational agribusinesses can parlay the geography of production into huge profits, regardless of the outbreaks that may accrue, who pays the costs? The costs of factory farms are routinely externalized. As Peter Singer (2005) explains, the state has long been forced to pick up the tab for the problems these farms cause; among them, health problems for its workers, pollution released into the surrounding land, food poisoning, and damage to transportation and health infrastructure. A breach in a poultry lagoon, releasing a pool of feces into a Cape Fear tributary that causes a massive fish kill, is left to local governments to clean up.

With the specter of influenza the state is again prepared to pick up the bill so that factory farms can continue to operate without interruption, this time in the face of a worldwide pandemic agribusiness helped cause in the first place. The economics are startling. The world's governments are prepared to subsidize agribusiness billions upon billions for damage control in the form of animal and human vaccines, Tamiflu, and clean-up operations. Along with the lives of millions of people, the establishment appears willing to gamble much of the world's economic productivity, which stands to suffer catastrophically if a deadly pandemic were to erupt, for instance, in southern China.

Why Guangdong? Why 1997?

In reorganizing its stockbreeding industries under the American model of vertically integrated farming, Chinese farming helped accelerate a phase change in influenza ecology, selecting for strains of greater

virulence, wider host range, and greater diversity. For decades a variety of influenza subtypes have been discovered emanating from southern China, Guangdong included (Chang 1969; Cheung et al 2007; Shortridge and Stuart-Harris 1982; Xu et al 2007). In the early 1980s, with livestock intensification under way, University of Hong Kong microbiologist Kennedy Shortridge (1982) identified 46 of the 108 different possible combinations of hemagglutinin and neuraminidase subtypes circulating worldwide at that time in a single Hong Kong poultry factory.

Shortridge detailed the likely reasons southern China has served, and will serve, as ground zero for influenza pandemics:

- Southern China hosts mass production of ducks on innumerable ponds, facilitating fecal–oral transmission of multiple influenza subtypes. Domestic ducks were first moved from rivers to cultivated rice fields at the start of the Qing Dynasty in the middle of the seventeenth century (Shortridge 2003b).
- The greater mix of influenza serotypes in southern China increases the possibility the correct combination of gene segments would arise by genetic reassortment, selecting for a newly emergent human strain.
- Influenza circulates year-round there, surviving the interepidemic period by transmitting by the fecal–oral mode of infection.
- The proximity of human habitation and a proliferation of live bird markets provide an ideal interface across which a human-specific strain may emerge.

The conditions Shortridge outlined 25 years ago have since only intensified with China's liberalizing economy. Millions of people have moved into Guangdong over the past decade, a part of one of the greatest migration events in human history, from rural China into cities of the coastal provinces (Fan 2005). Shenzhen, one of Guangdong's Special Economic Zones for open trade, grew from a small city of 337,000 in 1979 to a metropolis of 8.5 million by 2006. As discussed earlier, concomitant changes in agricultural technology and ownership structure have put hundreds of millions more animals into production (Burch 2005; Luo, Ou and Zhou 2003; Sun et al 2007). Poultry output increased in China from 1.6 million tons in 1985 to nearly 13 million tons by 2000.

As Mike Davis (2005:63) summarizes it, by the onset of pathogenic H5N1, only the latest pathogen to emerge under such socioecological conditions,

[S]everal subtypes of influenza were traveling on the path toward pandemic potential. The industrialization of south China, perhaps,

had altered crucial parameters in the already very complex ecological system, exponentially expanding the surface area of contact between avian and nonavian influenzas. As the rate of interspecies transmission of influenza accelerated, so too did the evolution of protopandemic strains.

The hemagglutinin protein of pathogenic H5N1 was first identified by Chinese scientists from a 1996 outbreak on a goose farm in Guangdong (Tang et al 1998). News reports during the initial H5N1 outbreak in Hong Kong detailed local health officials' decision to ban poultry imports from Guangdong from where several batches of infected chickens originated (Kang-Chung 1997). Phylogeographic analyses of the virus's genetic code have pointed to Guangdong's role in the emergence of the first and subsequent strains of pathogenic H5N1 (Wallace et al 2007). Scientists from Guangdong's own South China Agricultural University contributed to a 2005 report showing that a new H5N1 genotype arose in western Guangdong in 2003–2004 (Wan et al 2005).

Subsequent work has complicated the picture. With additional H5N1 samples from around southern China, Wang et al (2008) showed virus from the first outbreaks in Thailand, Vietnam and Malaysia appeared most related to isolates from Yunnan, another southern Chinese province. Indonesia's outbreaks were likely seeded by strains first isolated from Hunan Province. These are important results, showing the complexity of influenza's phylogeographic landscape. At the same time they need not absolve Guangdong. Even if some H5N1 strains emerged elsewhere in the region, Guangdong's socioeconomic centrality may have acted as an epidemiological attractant, drawing in novel poultry-trade-borne strains from around southern China before dispersing them again back out across China and beyond.

Mukhtar et al (2007) meanwhile traced the origins of the genomic segments from the original 1996 outbreak in Guangdong. The internal proteins (encoding for proteins other than surface proteins hemagglutinin and neuraminidase) appeared phylogenetically closest to those of H3N8 and H7N1 isolates sampled from Nanchang in nearby Jiangxi Province. The 1996 hemagglutinin and neuraminidase appeared closest to those of H5N3 and H1N1 isolates from Japan. In the months before the outbreak in Hong Kong several of the proteins were again replaced by way of reassortment, this time via strains of H9N2 and H6N1 (Guan et al 1999; Hoffmann et al 2000). H5N1 strains in the years that followed Hong Kong emerged by still more reassortment (Li et al 2004). The sociogeographic mechanisms by which the various segments first converged (and were repeatedly shuffled) in Guangdong remain to be better outlined. The results so far do indicate the spatial expanse over which reassortants originate may be greater than Kennedy Shortridge, or anyone else, previously imagined. But genomic origins

tell us little how this particular complement led to a virus that *locally evolved* such virulence other than showing the genetic variation upon which the virus drew.

A closer look at Guangdong's drastically shifting socioeconomic circumstances, then, appears necessary in better illuminating the local conditions that selected for such deadly pathogens so easily spread; not only H5N1, but a diverse viral portfolio, including influenza A (H9N2) (Liu et al 2003), H6N1 (Cheung et al 2007), and SARS (Poon et al 2004). What exactly are the "crucial parameters" for the area's disease ecosystem? What are the mechanisms by which changes in southern China's human–animal composite lead to regular viral pulses emanating out to the rest of China and the world? Why Guangdong? Why 1997 and thereafter?

700 Million Chickens

We begin with the death of Mao and the rehabilitation of Deng Xiaoping. In the late 1970s, China began to move away from a Cultural Revolution policy of self-sufficiency, in which each province was expected to produce most foods and goods for its own use. In its place the central government began an experiment centered about a reengagement with international trade in Special Economic Zones set up in parts of Guangdong (near Hong Kong), Fujian (across from Taiwan), and later the whole of Hainan Province. In 1984, 14 coastal cities—including Guangzhou and Zhanjiang in Guangdong—were opened up as well although not to the extent of the economic zones (Tseng and Zebregs 2003).

By macroeconomic indicators favored by establishment economists, the policy was a success. Between 1978 and 1993 China's trade-to-GNP ratio grew from 9.7% to 38.2% (Perkins 1997). Most of this growth stemmed from manufactured goods produced by foreign-funded joint ventures and township and village enterprises (TVE) allowed greater autonomy from central control. Starting in 1979, foreign direct investment (FDI) increased from zero to US\$45 billion by the late 1990s, with China the second greatest recipient after the USA. Sixty percent of the FDI was directed to cheap-labor manufacturing. Given the extent of China's smallholder farming, little FDI was initially directed to agriculture (Rozelle, Pray and Huang 1999).

That soon changed. Through the 1990s poultry production grew at a remarkable 7% per year (Hertel et al 1999). Processed poultry exports grew from US\$6 million in 1992 to US\$774 million by 1996 (Carter and Li 1999). China's Interim Provisions on Guiding Foreign Investment Direction, revised in 1997, aim to encourage FDI across a greater expanse of the country and in specific industries, agriculture included (Tseng and Zebregs 2003). The government's latest 5-year

plan sets sights on modernizing agriculture nationwide (Tan and Khor 2006). Since China joined the World Trade Organization in 2002, with greater obligations to liberalize trade and investment, agricultural FDI has doubled (Whalley and Xin 2006). But much opportunity for AgFDI remains available to a wider array of sources of investment. By the late 1990s Hong Kong and Taiwan's contribution to China's FDI had declined to 50% of the total, marking an influx of new European, Japanese, and American investment.

In something of a bellwether, in August 2008, days before the Beijing Olympics, US private equity investment firm Goldman Sachs bought 10 poultry farms in Hunan and Fujian for US\$300 million (Yeung 2008). The outright ownership appears a step beyond the joint ventures in which the firm had until then participated. Goldman Sachs already holds a minority stake in Hong Kong-listed China Yurun Food Group, a mainland meat products manufacturer, and 60% of Shanghai-listed Shuanghui Investment and Development, another meat packer. Goldman Sachs's new purchase, further up the filiere, signals a shift in the global fiscal environment. The firm adeptly moved out of high-risk US mortgages and, during a global food crisis, into the brave new world of offshore farming in China.

In October 2008 China's leadership finalized plans to formalize such privatization (Wong 2008). Under the rubric of land reform and doubling rural income, peasants will be allowed to engage in unrestricted trade as well as—this is key—buy and sell land-use contracts. These contracts are, in addition, to be extended from a ceiling of 30 years to 70 years. Contracting permits the government to retain land sovereignty as a political emblem. But as companies domestic and foreign are largely the only entities with the reserves on hand to enter such contracts, now extended to near perpetuity and for the incorporated dirt cheap, China's small farms will soon be open to a great land rush. We have, then, an "accumulation by dispossession" managed by a communist party (Harvey 2006).

Guangdong, as throughout, remains at the cutting edge of these economic shifts. It hosted the central government's first efforts at internationalizing the rural economy (Johnson 1992; Xueqiang et al 1995; Zweig 1991). Starting in 1978, Guangdong agricultural production was redirected from domestic grain to Hong Kong's market. Hong Kong businesses invested in equipment in return for new output in vegetables, fruit, fish, flowers, poultry and pig. In something of a reprisal of its historical role, Hong Kong ("the front of the store") also offered Guangdong ("the back of the store") marketing services and access to the international market (Heartfield 2005; Sit 2004). In a few short years Guangdong's economy again became entwined with and dependent upon Hong Kong's economic fortunes. And vice versa. As of the Hong Kong outbreak, investment in China comprised four-fifths

of Hong Kong's FDI outflow (Heartfield 2005). Much of Hong Kong-funded production is now conducted in Guangdong, with Hong Kong's industrial base increasingly hollowed out as a result.

Eighty-five percent of the agricultural FDI brought into China during the 1990s was funneled into Guangdong and several other coastal provinces (Rozelle, Pray and Huang 1999). Guangdong was allowed to invest more in its transportation infrastructure, in part as an invitation for further investment. Many of the province's companies were allowed to claim 100% duty drawbacks. Guangdong also developed trading arrangements with many of the 51 million Chinese overseas (Gu et al 2001; Heartfield 2005). As a class, the expatriates, nearly 200 years abroad, control large percentages of regional market capital, including in Indonesia, Thailand, Vietnam, the Philippines, Malaysia, and Singapore. At the time of the first H5N1 outbreaks, overseas Chinese collectively comprised the group with the greatest investment in mainland China (Haley et al 1998).

As a result of the area-specific liberalization, Guangdong accounted for 42% of China's total 1997 exports and generated China's largest provincial GDP (Gu et al 2001; Lin 2000). Of the coastal provinces, Guangdong hosted the greatest concentration of joint-venture export-oriented firms, with the lowest domestic costs for each net dollar of export income (Perkins 1997). Guangdong's three free economic zones (Shenzhen, Shantou and Zhuhai) boasted an export-to-GDP ratio of 67%, compared with a national average of 17%.

By 1997, and the first H5N1 outbreak in Hong Kong, Guangdong, home to 700 million chickens, was one of China's top three provinces in poultry production (Organisation for Economic Co-operation and Development 1998). Fourteen percent of China's farms with 10,000 or more broilers were located in Guangdong (Simpson et al 1999). Guangdong's poultry operations were by this point technically modernized for breeding, raising, slaughtering, and processing birds, and vertically integrated with feed mills and processing plants. AgFDI helped import grandparent genetic stock, support domestic breeding, and update nutrition feed milling/mixing (Rozelle, Pray and Huang 1999). Production has been somewhat constrained by access to interprovincial grain and the domestic market's preference for native poultry breeds less efficient at converting feed. Of obvious relevance, production also suffered from inadequate animal health practices.

The rate and magnitude of poultry intensification appears to have combined with the pressures placed on Guangdong wetlands by industry and a burgeoning human population to squeeze a diversifying array of influenza serotypes circulating year-round through something of a virulence ratchet. The resulting viral crop—for 1997, H5N1 by molecular happenstance—is exported out by easy access to international trade facilitated partly by expatriate capital.

Expanding Pathogens' Scope

Guangdong's ascension was not without its detractors, a dynamic with epidemiological consequences. Domestic producers in Hong Kong competed with Hong Kong–Guangdong joint ventures for export licenses (Zweig 1991). Landlocked provinces meanwhile chafed at the liberalization the central government proffered coastal provinces alone. With so much domestic currency on hand, the coastal provinces could outcompete inland provinces for livestock and grain produced by the inland's own TVEs. The coastal provinces were able to cycle their competitive advantage by turning cheap grain into more profitable poultry or flat-out re-exporting the inland goods, accumulating still greater financial reserves. At one point rivalries became so intense that Hunan and Guangxi imposed trade barriers upon interprovincial trade. The central government's efforts to negotiate interprovincial rivalries included spreading liberalization inland (Tan and Khor 2006). Provinces other than Guangdong and Fujian began to become entrained into market agriculture, albeit at a magnitude still outpaced by their coastal counterparts. Industrial poultry's expanding extent—by re-exporting and inland development—increases the geographic scope for H5N1's emergence and may explain the roles Yunnan and Hunan appear to have played in serving up H5N1 abroad.

An additional source of conflict, often forgotten in the cacophony of macroeconomic indicators, requires comment—the Chinese people themselves. China's state capitalism has induced such a polarization of wealth that, along with threatening its own economic growth, it impoverishes hundreds of millions of Chinese. In engaging in internally imposed structural adjustment China has largely turned away from its real and ideological investment in the health and wellbeing of its population (Hart-Landsberg and Burkett 2005a). Tens of millions of state industrial workers have been laid off. Labor income as a share of Chinese GDP fell from about 50% in the 1980s to under 40% by 2000 (Li 2008). FDI and private companies—under no obligation to offer housing, healthcare, or retirement benefits—are used to discipline Chinese workers who were long used to a living wage, basic benefits, and job protection (Hart-Landsberg and Burkett 2005b). Discipline, however, does not always take. Protests running now into the tens of thousands, some turning into riots requiring army deployment, have battered provincial governments accused of corruption, land confiscation, expropriating state assets, wage theft, and pollution. In something of an ironic twist, in defending foreign capital against its own people, China's communist leadership has taken on the role of the comprador class it first defeated in 1949 (Heartfield 2005).

Farmers have been particularly hard hit by the government's capitalist turn. While decollectivization of agricultural land to household control propped up by governmental price supports led to a doubling in rural

incomes by 1984, rural infrastructure and attendant social support have since deteriorated (Hart-Landsberg and Burkett 2005a). In the late 1980s, agricultural incomes stagnated, eaten away by inflation and a decline in price supports. Families began to abandon farming for informal industrial work in the cities. There, many rural migrants are treated as a reviled caste, discrimination codified by levels of officially designated migrant status and with attendant reductions in income (Fan 2001). China's macroeconomic growth has been unable to absorb many of the 100 million migrants.

Urbanization meanwhile has diffused out to the rural regions, eating up peasant land. One million Chinese hectares have been converted from agriculture to urban use (Davis 2006). Remote sensing shows from 1990 to 1996 13% of agricultural land in a 10-county region in Guangdong's Pearl River delta was converted into non-agricultural use, in all likelihood China's most rapid conversion (Seto, Kaurmann and Woodcock 2000). Rural towns have been transformed into growing industrial cities, some supporting populations tipping a million people (Lin 1997).

The termination of the commune system has left hundreds of millions of peasants without access to medical care and health insurance (Shi 1993). Universal health coverage has degraded to 21% of the rural population insured (French 2006). The number of affordable doctors has precipitously declined. Infant mortality has risen across many provinces. Rural public health has largely collapsed. Hepatitis and TB are now widespread. HIV incidence has increased in several southeastern provinces, Guangdong included (Tucker et al 2005). STI incidence by province is correlated with immigration associated with surplus men from rural regions separated from their families. Multitudes of malnourished and immunologically stressed peasant-factory workers cycle-migrating back and forth from what may be the geographic origins of an influenza pandemic would appear to compromise World Health Organization plans for intervening at any new infection's source.

Asia's Financial Flu

It is hard to discuss 1997 without mentioning two events of geopolitical significance. On 1 July that year, Hong Kong, long a British colony, was officially transferred to China as a Special Administrative Region, the first in a series of steps to full integration to be undertaken up through 2047. The next day the Bank of Thailand floated the baht off the US dollar. The baht had been hammered by currency speculation and a crippling foreign debt. International finance fled from the baht and soon, with the economic strength of Thailand's neighbors also under suspicion, from other regional currencies. The FDI-dependent economies of the Philippines, Malaysia, Indonesia, Taiwan, and South

Korea suffered in the ensuing wave of devaluation. The rest of the world too felt the effects of the infectious "Asian flu", as the crisis came to be called, with stock markets worldwide free-falling in response. Although Hong Kong's transfer to China and the Asian financial crisis followed the first outbreaks of bird flu in March, the events marked long-brewing shifts in regional political economy with apparent impact on viral evolution and spread.

Hong Kong's role in China's internally imposed structural adjustment, as we explored above, is amply documented. The intensification of Guangdong poultry went hand in hand with the ongoing transformation of the province's border with Hong Kong (Breitung 2002). The resulting poultry traffic, however, is in no way unidirectional. Hong Kong exports to mainland China large amounts of poultry, fruits, vegetables, nuts, oilseeds, and cotton (Carter and Li 1999). There is too a large illegal trade. At the time of the outbreak, Hong Kong chicken parts smuggled into China alone may have amounted to over US\$300 million per year (Carter and Li 1999; US Trade Representative 1998). Hong Kong is clearly less a victim of Guangdong's bird flu ecology, as often portrayed, than a willing participant.

The financial crisis meanwhile slowed China's economy. China, however, avoided the worst of the financial flu (Lin 2000). By staking billions in public works and loans, the central government kept the country's economic engine primed in the face of slowing exports. Prophetically, 4 years previous, the government introduced fiscal austerity measures to cool off inflation and the possibility of an overheated economy. An associated regulation package was initiated to control the kind of short-term speculation that would soon strain China's regional neighbors. The central state maintains tight control over the macroeconomy, capital flows, and corporate structure even as it cedes much of the day-to-day operations to provincial authorities. Concomitantly, China's economy is more than export driven. Even as austerity leaves millions of Chinese destitute in its wake (Hart-Landsberg and Burkett 2005a), up until the past year's global contraction the domestic economy continued to grow, albeit increasingly dependent on luxury goods and real estate speculation. Finally, exports out of China were until the 1997 crisis largely destined for East and Southeast Asia. During the crisis's aftermath China redirected more of its trade to Europe, North America, Africa, Latin America, and Oceania. China, then, was able to maintain a trade surplus, retain foreign investment, and prop the yuan against the fiscal buffet from abroad.

Be that as it may, China was something more than a bystander to the crisis. Its economy's growing size and hemispheric reach may have exposed its neighbors to the worst excesses of the neoliberal model (Hart-Landsberg and Burkett 2005a; Tan and Khor 2006). In attracting FDI at rates above and beyond those of its neighbors, China

has become the prime exporter in the region: textiles, apparel, household goods, televisions, desktop computers, an increasing array of high-end electronics, etc. The smaller economies were forced to restructure production in such a way as to complement China's increasingly diverse commodity output, in a type of regional division-of-labor. China's transnational impact on supply lines forces each of the other countries to depend on producing a smaller array of parts to be put together in China for final export.

The resulting economies are more dependent on what few foreign multinationals they are able to attract. The company town becomes the company country. Such economies are more "brittle"—less robust in reacting to and reorienting around downturns in any single industry, a particularly pernicious problem as the US begins to falter in its role of importer of last resort. Capital flight exposes countries to the temptations of currency speculation. To attract additional investment, establishment economists declare these countries, once burned by such speculation, must now remove remaining barriers to the movement of money, goods, and capital, leaving domestic production unprotected, the very conditions that brought about the 1997 crisis in the first place.

It would appear bird flu and the financial flu are intimately connected, their relationship extending beyond serendipitous analogy. Although agriculture has been until recently less export dependent than manufacturing, in part from its perishability and now endangered trade protections (Hertel et al 2000), there are already a number of epidemiological ramifications. These include a geographically expanding and intensifying poultry production, greater exposure to transnational poultry, wider illegal poultry trade, and a truncation in animal health infrastructure by austerity measures domestically imposed in return for international loans or by ideological imperatives (Rweyemamu et al 2000). More acutely, the aftermath of the financial flu may have also provided China a window for expanding regional poultry exports. A hypothesis worth testing is that some of these shipments seeded bird flu outbreaks abroad.

How do we operationalize this model? How do we determine whether transnational companies breed and spread influenza? Identifying poultry crates carrying H5N1 locality-to-locality remains a difficult but important task (Kilpatrick et al 2006). Tracing pathogens through commodity chains is increasingly viewed as a critical topic of study and mode of intervention (Duffy, Lyncha and Cagneya 2008). One difficulty centers about the willingness of government regulators to inspect livestock plants, including conditions under which pathogen virulence may evolve. At the same time, there is a danger such efforts, once successful, may detract from the larger political ecology that shapes influenza evolution. With billions annually at stake, a few unlucky contract farmers or truck drivers may be sacrificed to protect a system

stretching across the globe's interlocking markets. We have explored here the possibility a deadly bird flu is an unintended but not unexpected accessory to multinational efforts to export a growing portfolio of Chinese agricultural commodities. The problem of influenza is more than a police matter. It is systemic, buried deep in political tissue. The virus, moreover, is complicated by a causality that extends out beyond the factory gate.

Layers of Complication

Ending large livestock operations as we know them could make a great difference in Guangdong as elsewhere. Such politically protected operations appear to promote both pathogen virulence and transmission. Graham et al (2008) review a number of proximate environmental pathways by which pathogens can spread across and out of large confined animal feedlot operations, including via animal waste handling and use in aquaculture, workers' occupational exposure, open transport of animals between farms and processing plants, contamination of shipping containers, non-livestock animals such as rats and flies, and tunnel ventilation systems that blow animal materials out into the environment. It would appear "biosecure" operations are not so biosecure.

But there are additional layers of complication. There is no easy one-to-one relationship between poultry density and H5N1 outbreak at a variety of spatial scales. Across Asia, some areas where outbreaks have occurred support comparatively few poultry, while other areas with millions of chickens so far have been left untouched. There is a stochastic component to disease spread. Epidemics start somewhere, in this case in southern China, and take time to wend their way elsewhere, starting with regions nearby and, in part by due cause and in part by chance, farther abroad. There are, however, demonstrable causes other than those inside the poultry industry.

Thailand offers one such example. As mapped by ecologist Marius Gilbert and colleagues (Gilbert et al 2006, 2008), the distributions of both Thai broilers and backyard poultry appear little associated with H5N1 outbreaks. Local outbreaks appear better fitted to the densities of ducks that are allowed to graze freely outside. After harvests these ducks are brought in to feed on the rice that is left over on the ground. Satellite pictures show rice harvests matching duck densities. The more annual rice crops the more ducks (and the greater the association with H5N1 outbreaks). It seems these ducks, free to graze outdoors, exposed to migratory birds, and tolerant of a wider range of influenzas, serve as epidemiological conduits for infecting nearby poultry. While a rather ingenious agricultural practice, raising a cohort of ducks on fallen waste rice may carry serious epidemiological overhead.

Double and even triple cropping are practiced in other bird flu zones, including southeastern China, the final stretches of the Xun Xi River, the Ganges floodplain, and on the island of Java (Leff, Ramankutty and Foley 2004).

We have, then, an integrated viral ecology with highly complex dependencies. The variety of farming practices, for one, splits a twain a number of facile dichotomies. There is a panoply of farm types, beyond the rough polarities of “small” and “large”. In Thailand alone there are closed-off farms, open structures with netting to block passerine birds, the aforementioned free-grazing ducks, and backyard poultry (Songserm et al 2006). Even then, such a taxonomy implies a compartmentalization often absent in the field. On a recent trip to Lake Poyang in Jiangxi Province, China, an international team of experts discovered an astonishing farming ecology in which domesticated free-range ducks fed in fields, bathed in local estuaries, swam in the lake, and intermingled and presumably interbred with wild waterfowl. Some flocks daily commuted across dykes from their sheds to the open water and back again. The epidemiological implications are obvious. Indeed, the facility by which pathogens spread and evolve in the area is of an order that, according to local farmers, chickens cannot be raised around the lake. For some poultry species the region is epidemiologically radioactive.

Absent too from the taxonomy are profound structural changes imposed by economic pressures upon world farming (Weis 2007). For the past three decades, the International Monetary Fund and the World Bank have made loans to poorer countries conditioned on removing supports for domestic food markets. Small farmers cannot compete with cheaper corporate imports subsidized by the global North. Many farmers either give up for a life on periurban margins or are forced to contract out their services—their land, their labor—to livestock multinationals now free to move in (Lewontin 2007; Manning and Baines 2004). The World Trade Organization’s Trade-Related Investment Measures permit foreign companies, aiming to reduce production costs, to purchase and consolidate small producers in poorer countries (McMichael 2006). Under informal contract, small farmers must purchase transnational-approved supplies and are not always guaranteed their birds will be bought back by their transnational partner at fair market price or bought back at all (Delforge 2007). The new arrangements belie the superficial distinction that has been made between industrial farms exercising “biosecurity” on the one hand and small farmers whose flocks are exposed to the epidemiological elements. Factory farms ship day-old chicks to be raised piecemeal by contracted farmers. Once grown (and exposed to migratory birds), the birds are shipped back to the factory for processing. The violation of biosecurity appears built directly into the industrial model.

A third complication is the historical shift in the relationship between nature and farming. Maps in Phongpaichit and Baker (1995) show since 1840 Thailand has been transformed from primary wilderness into an agricultural state, a veritable bread basket. Agriculture's new girth comes at the expense of wetlands worldwide, either out-and-out destroyed, polluted, or irrigated dry. The latter abuse serves as another basis for conflicts between agribusiness and small farmers. Socially stratified power struggles over the Chao Phraya basin have wracked Thailand for hundreds of years (Molle 2007).

Wetlands have traditionally served as *Anatidae* migration pit stops (Lemly, Kingsford and Thompson 2000). A growing literature shows many migratory birds are no sitting ducks and have responded to the destruction of their natural habitat. Geese, for example, display an alarming behavioral plasticity, adopting entirely new migratory patterns and nesting in new types of wintering grounds, moving from deteriorating wetlands to food-filled farms. The shift has for some waterfowl populations substantially *increased* their numbers (Jeffries, Rockwell and Abraham 2004; Van Eerden et al 2005). The population explosions have initiated a destructive feedback in which the swarms of farm-fed migratory birds overgraze their Arctic breeding grounds to the point the tundra is transformed into a landscape of mud. In the course of colonizing our planet's natural habitats—some 40% of the world's usable land now supports agriculture—we may have unintentionally expanded the interface between migratory birds and domestic poultry.

Clearly agribusiness, structural adjustment, global finance, environmental destruction, climate change, and the emergence of pathogenic influenzas are more tightly integrated than previously thought. The nest of dependencies requires fuller investigation. But, given the stakes, the connections we have been able to make deserve immediate action.

The Political Will for an Epidemiological Way

Guangdong may only represent the front of a socioecological transformation spreading across much of southern China and now much of the world. The origins of highly pathogenic H5N1 *are* multifactorial, with many countries and industries and environmental sources at fault. Can we then place blame on the country, say, Indonesia or Vietnam or Nigeria, from which a human-to-human H5N1 infection might first emerge? Should we hold China accountable for repeatedly seeding outbreaks regionally and internationally? Should we broach Hong Kong's offshore farming? Or should we castigate the United States, where the industrial model of vertically integrated poultry first originated, with thousands of livestock packed in as so much food for flu? The answers are affirmative across the board. Responsibility, much

as the problem itself, must be distributed about its multiple levels of social and ecological organization.

To beat back industrial influenza, or at the very least promote some sort of sustainable epidemiological mitigation, a number of radically invasive changes are required, changes that challenge core premises of present political economy, neoliberal and state capitalist alike. Whether there exists the political will to change is an open question. Denial, jockeying and obfuscation are presently rampant. Chinese officials have expended much effort in denying responsibility for bird flu (Wallace 2007) or, in the epidemiological equivalent of the American practice of paying off the families of collateral damage without admitting guilt, offered small sums to affected countries. In 2007, China donated US\$500,000 to Nigeria's effort to fight bird flu. Never mind that Nigeria would never have needed the aid if China had not infected it—albeit indirectly—with bird flu in the first place. The Qinghai-like strain Nigeria now hosts first originated in southern China. Meanwhile, the US and EU, criticizing a stubborn Indonesia unwilling to share H5N1 samples, have blocked Indonesian efforts to reform a system of worldwide vaccine production that rewards pharmaceutical companies and the richest populations at the expense of the poorest (Hammond 2007, 2008).

What must be done to stop panzootic influenza, if the political will is found by, or forced upon, governments worldwide? In the short term, small farmers must be fairly compensated for animals culled in an effort to control outbreaks. Livestock trade must be better regulated at international borders (Capua and Alexander 2006; Kilpatrick et al 2006; Wallace and Fitch 2008). Livestock disease surveillance, largely voluntary at this point, must be made mandatory and conducted by well-funded governmental agencies. Frontline farm workers and the world's poor more generally must be provided epidemiological assistance, including vaccine and antiviral at no cost (Cristalli and Capua 2007; Ferguson 2007; Graham et al 2008). Structural adjustment programs degrading animal health infrastructure in the poorest countries must be terminated.

For the long term, we must end the livestock industry as we know it. Influenzas now emerge by way of a globalized network of corporate feedlot production and trade, wherever specific strains first evolve. With flocks and herds whisked from region to region—transforming spatial distance into just-in-time expediency (Harvey 2006 [1982])—multiple strains of influenza are continually introduced into localities filled with populations of susceptible animals. Such domino exposure may serve as the fuel for the evolution of viral virulence. In overlapping each other along the links of agribusiness's transnational supply chains, strains of influenza also increase the likelihood they can exchange genomic segments to produce a recombinant of pandemic potential. In addition

to the petroleum wasted and the loss of local food sovereignty, there are epidemiological costs to the geometric increase in food miles.

We might instead consider devolving much of the production to regulated networks of locally owned farms. While the argument has been made that corporate food supplies the cheap protein many of the poorest need, the millions of small farmers who fed themselves (and many millions more) would never have needed such a supply if they had not been pushed off their lands in the first place. A reversal need not involve ending global trade or an anachronistic turn to the small family farm, but might include domestically protected farming at multiple scales (Brown and Getz 2008; Levins 1993, 2007). Farm ownership, infrastructure, working conditions, and animal health are inextricably linked. Once workers have a stake in both input and output—the latter by outright ownership, profit sharing, or the food itself—production can be structured in such a way that respects human welfare, and, as a consequence, animal health. With locale-specific farming, genetic monocultures of domesticated animals which promote the evolution of virulence can be diversified back into heirloom varieties that can serve as immunological firebreaks. The economic losses influenza imposes upon global livestock can be tempered: fewer interruptions, eradication campaigns, price jolts, emergency vaccinations, and wholesale repopulations (Van Asseldonk et al 2005). Rather than jury-rigged with each outbreak, the capacity for restricting livestock movement is built naturally into the regional farm model.

The devil of such a domain shift is in its details. Richard Levins (2007:359), with decades of experience, collaborating with local researchers and practitioners on ecological approaches to Cuban agriculture and public health, summarizes some of the many adjustments a new agriculture anywhere may require:

Instead of having to decide between large-scale industrial type production and a “small is beautiful” approach *a priori*, we saw the scale of agriculture as dependent on natural and social conditions, with the units of planning embracing many units of production. Different scales of farming would be adjusted to the watershed, climatic zones and topography, population density, distribution of available resources, and the mobility of pests and their enemies.

The random patchwork of peasant agriculture, constrained by land tenure, and the harsh destructive landscapes of industrial farming would both be replaced by a planned mosaic of land uses in which each patch contributes its own products but also assists the production of other patches: forests give lumber, fuel, fruit, nuts, and honey but also regulate the flow of water, modulate the climate to a distance about 10 times the height of the trees, create a special microclimate downwind from the edge, offer shade for livestock and the workers, and provide

a home to the natural enemies of pests and the pollinators of crops. There would no longer be specialized farms producing only one thing. Mixed enterprises would allow for recycling, a more diverse diet for the farmers, and a hedge against climatic surprises. It would have a more uniform demand for labor throughout the year.

Rather than to the expectations of an abstract neoclassical model of production, the scale and practice of agriculture can be flexibly tailored to each region's physical, social and epidemiological landscapes on the ground. At the same time, it needs to be acknowledged that under such an arrangement not all parcels will be routinely profitable. As Levins points out, whatever reductions in income farms accrue in protecting the rest of the region must be offset by regular redistributive mechanisms.

Transforming the business of farming so broadly, as outlined here or otherwise, is likely only one of many large steps necessary to stop influenza and other pathogens. For one, migratory birds, which serve as a fount of influenza strains, must concomitantly be weaned off agricultural land where they cross-infect poultry. To do so, wetlands worldwide, waterfowl's natural habitat, must be restored. Global public health capacity must also be rebuilt (Garrett 2001). That capacity is only the most immediate bandage for the poverty, malnutrition, and other manifestations of structural violence that promote the emergence and mortality of infectious diseases, including influenza (Farmer 2004; Kim et al 2000). Pandemic and inter-pandemic flu have the greatest impact on the poorest (Davis 2005). As for many pathogens, particularly for such a contagious virus, a threat to one is a threat to all.

In implementing interventions for an industrial pollutant that evolves, we will also be forced to re-imagine a virology that extends out from underneath the microscope. Disease interventions, at both the individual and population levels, are, with a few bright exceptions, faltering across multiple pathogens. Vaccines, pharmaceuticals, and low-tech solutions, such as bed nets and water filters, while successful in addressing many reductionist diseases, cannot contain pathogens that use interactions at one level of biocultural organization to evolve out from underneath interventions directed at them at another. Such holistic diseases, operating across fluctuating swaths of space and time, infect and kill millions annually. HIV, tuberculosis, malaria, along with influenza, confound even the most concerted efforts.

New ways of thinking about basic biology, evolution, and scientific practice are in order. In a world in which viruses and bacteria evolve in response to humanity's multifaceted infrastructure—agricultural, transportation, pharmaceutical, public health, scientific, political—our epistemological and epidemiological intractabilities may be in fundamental ways one and the same. Some pathogens evolve into population states in which we cannot or, worse, refuse to think (Wallace

and Wallace 2004). None of the broader factors shaping influenza evolution and drug response can be found underneath the microscope, no matter how many more automated microplates can now be loaded or how much industrial computing power becomes available. A geography connecting relationships among living organisms and human production across scale and domain may help us make the mental transitions necessary to excavate those population states in which influenza is able to shield itself. It may be only then that we can better control a pathogen seemingly capable—by distributed epiphenomena—of a chilling premeditation.

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