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Dead Cities and Other Tales

The Monster at Our Door

The Global Threat of Avian Flu

Mike Davis

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their public-health systems, Toronto and Hong Kong are likewise affluent cities with superb laboratory medicine.

SARS in Bangladesh, Afghanistan, or Zaire would have been a different pandemic. This is exactly the "What if?" that haunted the Royal Society's postmortem on the SARS pandemic: "[S]uppose the virus had flown from Hong Kong to Durban instead of Toronto. It is a city of similar size but without a similar health infrastructure, and with a significant proportion of its inhabitants immune-compromised owing to HIV-1 infection. Then Africa could have become endemic for SARS by now."[131] An influenza pandemic, to be sure, would not neglect the poor countries of the world.

7

The Triangle of Doom

We need to look in our own backyard for where the next pandemic may appear.[132]

Christopher Olsen

The SARS pandemic ratified Guangdong's exceptional importance as a disease epicenter. But does Guangdong have a unique franchise? Some influenza experts believe that all pandemics originate in the mixed swine-and-poultry agriculture of south China, a near-dogma that makes them resist compelling evidence that the 1918 reassortant first emerged in Kansas.[133] Other researchers, however, argue that the environmental preconditions for the rapid interspecies evolution of influenza are now found elsewhere, and they point specifically to the ecological impacts of the export-led industrialization of poultry and pork production since the 1980s.

This so-called Livestock Revolution has been primarily driven by Third World urbanization and the rising demand in developing countries—above all, China—for poultry, pork, and dairy products. Although Third World urban dwellers are obviously poorer than their OECD counterparts, a much larger percentage of income growth is expended on animal protein, and this is the demand engine that currently drives huge

increases in chicken and swine populations. According to Australian researchers, "The [global] share of meat and milk consumed in developing countries rose from 37 to 53 percent and from 34 to 44 percent, respectively, from 1983 to 1997. . . . By contrast, both per capita and aggregate milk and meat consumption stagnated in the developed world, where saturation levels of consumption have been reached and population growth is small." From the standpoint of influenza ecology, moreover, it is striking that pork and poultry constitute 76 percent of the developing world's increased meat consumption, and poultry has accounted for almost all of the small net

Table 7.1.
The Livestock Revolution[134]

(million metric tons)

	1983	*1997*	*2020*
DEVELOPED WORLD			
Total meat	88	99	117
Pork	34	36	39
Poultry	19	28	39
THIRD WORLD			
Total meat	50	112	217
Pork	20	46	81
Poultry	10	29	70
CHINA			
Total meat	16	53	107

increase in rich countries' food consumption.[135] The viral "food supply"—poultry, swine, and humans—has been dramatically enlarged.

Like the Green Revolution before it, the Livestock Revolution has favored corporate producers rather than peasants and family farmers. As a recent UN report emphasizes, "large-scale, industrial production accounts already for roughly 80 percent of the total production increase in livestock products in Asia since 1990. In the future, most production, especially of pigs and poultry, is expected not to come from traditional production systems that have characterized the region for centuries, but from industrial, large-scale production."[136]

The world icon of industrialized poultry and livestock production is giant Tyson Foods, which, like Wal-Mart, grew up in hardscrabble Arkansas. Tyson, which kills 2.2 billion chickens annually, has become globally synonymous with scaled-up, vertically coordinated production; exploitation of contract growers; visceral antiunionism; rampant industrial injury; downstream environmental dumping; and political corruption. The global dominance of behemoths like Tyson has forced local farmers to either integrate with large-scale chicken- and pork-processing firms or perish. "These firms," write Donald Stull and Michael Broadway, "owned not only the broilers they supplied to contract growers, but the eggs that hatched the birds, the feed that went into them, and the plants that processed and then sold them to grocery stores."[137] Whether in the Ozarks, Holland, or Thailand, entire farming districts have been converted to the warehousing of poultry, with farmers serving as little more than chicken custodians. At the same time, livestock has been disintegrated from agriculture; thus creating a new geography

where grain and feed production is spatially separate from the raising of chickens and pigs.[138]

The result has been extraordinary population concentrations of poultry. A crucial requirement of the modern chicken industry, for example, is "production density," the compact location of broiler farms around a large processing plant.[139] As a result, there are now regions in North America, Brazil, western Europe, and South Asia with chicken populations in the hundreds of millions—in western Arkansas and northern Georgia, for example, more than 1 billion chickens are slaughtered annually. Similarly, the raising of swine is increasingly centralized in huge operations, often adjacent to poultry farms and migratory bird habitats. The superurbanization of the human population, in other words, has been paralleled by an equally dense urbanization of its meat supply. (One swine megafarm in Milford Valley, Utah, reputedly produces more sewage than the city of Los Angeles.) Might not one of these artificial Guangdongs be a pandemic crucible as well? Could production density become a synonym for viral density?

The answer to these questions was revealed in March of 2003. While scientists were desperately trying to figure out the identity of an atypical pneumonia in China, chickens were dying on a farm in the Gelder Valley (Gelderland) of Holland. The Netherlands is the world's leading exporter of eggs and live chickens, as well as a major producer of turkeys and geese; the hundreds of chicken farms in the Gelderland are at the center of the highly rationalized, $2 billion-per-year Dutch poultry industry. Many of the farms also keep pet flocks of ducks and swans.[140] With its intimate juxtaposition of wetlands, wild birds, poultry, and high urban density, as well as its hub-like role in the

European Union's global commerce, the Netherlands recapitulates many of the distinctive features of the Pearl River Delta; the March epidemic, in fact, was later traced back to a farm whose free-range chickens were in contact with wild waterfowl in an adjacent canal.

Although vigilant Dutch agricultural authorities quickly quarantined the movement of chickens and temporarily halted poultry exports, the Highly Pathogenic Avian Influenza (HPAI) swept like wildfire through the Gelderland. The virus was identified as an H7N7 strain more or less identical to a strain isolated in mallards several years earlier.[141] By April, turkeys were dying in North Brabant, and the first HPAI cases were reported in Meeuwen-Gruitrode in neighboring Belgium. Even more disturbingly, evidence of the infection was discovered in pigs on several farms in the Gelderland, increasing the dangerous likelihood of H7N7's reassortment with swine and human influenzas. (The pigs were promptly slaughtered.) As European Union agricultural experts fretted over the potential for a pan-European epidemic, the Dutch government came under immense domestic and foreign pressure to act more aggressively. The Hague decided to exterminate all the poultry in the Gelderland and other infected areas and to dispose of thousands of tons of virus-laden chicken manure. As thousands of unhappy farmers clamored in protest, crews of poultry workers, aided by the Dutch army, began the epic slaughter of more than 30 million chickens, almost one-third of Holland's entire poultry population.[142]

Although HPAI was an enormous threat to the poultry industry, there was little apprehension of any public-health danger. A few years earlier, there had been a serious H7N7 outbreak

among chickens in Italy, but serological analysis found no evidence of any transmission to humans. Moreover, all the personnel involved in the Dutch cull wore protective clothing, including goggles and mouth-and-nose masks. Even when a veterinarian who been involved in the early identification of the outbreak developed acute conjunctivitis, experts expressed surprise but not alarm: in 1996, an English duck owner had developed mild conjunctivitis after contact with a sick bird and there was an extraordinary case where an avian H7 had been transmitted to a human from a sick seal, but did not cause serious illness; H7N7 was also known to be endemic in horses. The virus's modest talent for crossing species barriers had never been accompanied by corresponding virulence—on rare occasions the virus apparently could inflame cells around the eye but it had shown no ability to replicate in the human respiratory tract or other tissues.[143]

This benign view of H7N7, however, was quickly challenged by a chorus of complaints from poultry workers with conjunctivitis, and in a few cases, reports of classical flu symptoms. Because some immigrant workers, now unemployed after the cull, had already returned to their native countries, there was concern that they might seed new outbreaks. The prestigious Dutch National Institute of Public Health and the Environment quickly dispatched an expert investigation team, under the leadership of Dr. Marion Koopmans, to the Gelderland. A medical command center was established, and from 8 March nurses visited every household that might have had contact with infected birds. Since the ordinary flu season was in progress, vaccinations were made obligatory for poultry workers and their families, although this policy was implemented too late to prevent several

worrisome cases of co-infection by H7N7 and normal H3N1. Meanwhile, the outbreak team was stunned by the scale of infection they discovered: 553 people out of an exposed population of approximately 4,500 reported conjunctivitis or other symptoms; subsequent serological studies demonstrated that, in fact, as many as 2,000 of the exposed group had been infected but not always sickened. Surgical masks and goggles, for whatever reason, had afforded the poultry cullers little or no protection against the virus.[144]

Moreover, relatives and housemates of poultry workers, who had no direct contact with infected birds, also developed conjunctivitis. Public-health officials were convinced that the virus had acquired a limited but real ability to spread via person-to-person contact, although the exact mode of transmission was unclear. The outbreak team also found evidence that H7N7 was accumulating dangerous mutations as it passed through the human population. The event's most frightening moment was the death of a fifty-seven-year-old veterinarian on 19 April; soon after exposure to sick chickens, he had developed viral pneumonia (and later ARDS) instead of relatively benign conjunctivitis. Previously in good health, he was not immune-compromised, nor did he have any underlying disease. Alarmingly, his catastrophic decline matched the gruesome clinical descriptions of the 1997 deaths in Hong Kong, or for that matter, the acute cases in 1918.[145]

An urgent analysis of viral samples removed from the vet's lungs revealed that the strain that killed him was not an avian–human reassortant, as some had feared, but a variant of the original H7N7 virus which had undergone twelve amino acid substitutions; some mutations affected its hemagglutinin, while others modified the PB2 protein, part of the polymerase

complex that helped replicate the virus. While HA has always been influenza's celebrity protein because of its crucial role in determining host range, and possibly, virulence, the Dutch researchers, like colleagues elsewhere, were coming around to the idea that mutations in internal proteins—such as PB2 or the nonstructural protein NS2—might be important co-factors in the severity of infection. They knew that previous research had shown that a mutation in PB2 had increased the virulence of H5N1 in mice—perhaps H7N7 reacted the same way. In any event, the Dutch outbreak, with its deadly index case, now had the WHO's attention, even if the world press was diverted by the ongoing battle against SARS.[146]

After H7N7's brief forays into Belgium and Germany, the outbreak was officially contained in August. Dutch experts regarded it as another harrowingly close call with a potentially deadly pandemic:

> Although we launched a large and costly outbreak investigation (using a combination of pandemic and bioterrorism preparedness protocols), and despite decisions being made very quickly, a sobering conclusion is that by the time full prophylactic measures were reinforced . . . more than 1000 people from all over the Netherlands and from abroad had been exposed. Therefore if a variant with more effective spreading capabilities had arisen, containment would have been very difficult.[147]

Like the earlier H9 outbreak, the Gelderland epidemic demonstrated that multiple subtypes (including H9, H7, and possibly

H4 and H6, as well as reborn H2) were racing H5 to the pandemic finish line. The rapidity and scale of the Dutch outbreak also proved that south China no longer had a monopoly on deadly influenza: there were now multiple epicenters.

The H7N7 crisis also provided an additional reason for public-health officials and human influenza researchers to talk to their expert animal-virus counterparts. In the past, human and veterinary medicines had been parallel sciences that only occasionally intersected during rare interspecies disease events, but now the two viral universes, animal and human, seem to be locked together in a frenetic evolutionary embrace that makes the old dualism seem obsolete. Let me suggest an analogy: during the Second World War, the Allies and Nazis fought a secret high-stakes war over remote weather stations in Greenland, because knowledge of weather-front conditions in East Greenland anticipated Western Europe's weather by several days; such intelligence was of incalculable value in planning strategic surprises such as D-Day or the Battle of the Bulge. Likewise, the March 2003 Dutch epidemic proved how crucial veterinary surveillance has become for anticipating human outbreaks. To avoid a catastrophic pandemic surprise, it is urgent to know what is happening on farms months, even years, ahead of any human transmission.

Several specific developments in the wake of the global Livestock Revolution have especially put scientists' nerves on edge. One is the sudden viral chaos on pig farms since 1997. For the previous sixty or seventy years, swine influenza—a lineage derived from the H1N1 of 1918—exhibited extraordinary genetic stability. Although individual pigs occasionally became mixing vessels for avian strains (as many believed happened in 1957 and again in 1968), the H1N1 dynasty was otherwise as

unremitting as the Habsburgs. Then in 1997, the hogs on one of North Carolina's megafarms caught H3N2, a human flu; this subtype soon reassorted with avian and classic swine viruses, and "by late 1999, the novel viruses could be found wherever there were pigs in North America and so were presumably spread by cross-country transport." The emergent menagerie includes an H1N2 that is the offspring of human and swine subtypes, as well as an H1N1 that preserves the classical outer proteins but whose internal proteins are human and avian. All novel subtypes are dangerous, but an H4N6 virus, a wholly avian strain that passed to Canadian hogs from ducks, is perhaps the most sinister, because it has "already acquired genetic mutations that give it the potential to bind to human cell receptors." "Such an event," warns one research team, "could be catastrophic, as humans have no immunity to H4 viruses."[148]

The new swine flu pandemic threat apparently has arisen directly from the increasing scale of hog production; researchers told *Science* that swine influenza's sudden burst of mutational energy has probably been stimulated by parallel changes in herd size, interstate transport of hogs, and vaccination practice. Since 1993, U.S. pork production has been restructured around the Tyson, or "poultry model," of very large, industrialized units. In a single decade, from 1993 to 2003, the percentage of hogs raised on factory farms with more than 5,000 animals increased from 18 percent to 53 percent. Such large herds maximize the opportunities for new viruses to replicate and develop epidemic momentum. "With a group of 5000 animals," an agricultural statistician explained to *Science*, "if a novel virus shows up, it will have more opportunity to replicate and potentially spread than in a group of 100 pigs on a small farm."[149]

Increased shipping of hogs over distance simultaneously expands the radius of potential infection. Meanwhile, "in less than a decade, vaccination has become the norm for breeding sows, which in turn pass their maternal antibodies on to their progeny . . . but the vaccine is not protecting against all new strains." What seems to be happening, instead, is that influenza vaccinations—like the notorious antibiotics given to steers—are probably selecting for resistant new viral types. In the absence of any official surveillance system for swine flu, a dangerous reassortant could emerge with little warning.[150]

Another "in our own backyard" trend that raises anxiety is the prevalence of so-called Low Pathogenic Avian Influenza (LPAI); LPAI infections, according to the *Terrestrial Animal Health Code* published by the Office International des Epizooties (OIE), are endemic in wild birds, causing mild symptoms and low mortality in poultry. In the United States, the Department of Agriculture responds to all HPAI outbreaks, but control of LPAI is left to individual states whose agricultural agencies are often captives of local agribusiness. In an era of crumbling species barriers and increasing pandemic risk, such special-interest federalism poses unacceptable public-health risks: consider the secret LPAI epidemic in California in 2000–4.

In 2000 an H6N2 influenza began circulating in Southern California poultry. The virus intrigued the scientists who sequenced its genome, because its proteins appeared to derive from both North American and Eurasian lineages of waterfowl: this was considered to be a warning that previously separate genetic hemispheres had now been bridged and that East Asia viruses have arrived in the United States.[151] In its early stages the new virus caused very few clinical symptoms, but it quickly

evolved more lethal genotypes. By January 2002 a particularly virulent strain appeared on a San Diego farm and spread to other local poultry ranches; infected hens from Southern California were then shipped to Turlock in the Central Valley. A major poultry processing center, Turlock became the hub of an explosive epidemic. As a study published by the Institute of Medicine explains: "Millions of birds shedding viruses traveling in trucks easily spread the infection to farms along the route. That is when the Turlock region, which is bound by three major roads, became known as the Triangle of Doom: a bird couldn't enter the region without becoming infected with H6N2. Tens of millions of birds in California became infected with this H6N2 virus during a four-month period beginning in March 2002."[152]

This massive epidemic—in contrast to the HPAI outbreak in Holland—was largely invisible. From the very beginning, growers used only their own veterinarians and did not release the diagnoses, "not to the state or to other potentially affected states, not to the OIE, not even to neighboring farms, who might have better protected their flocks from infection had they known about it." The emergence of this so-called "Triangle of Doom" was also kept quiet "by corporate decision-makers who feared that consumer demand would plummet if the public knew they were buying infected meat and eggs."[153] As with the SARS outbreak in China the following year, economic interests trumped any concern for public health.

But what, exactly, is the human risk from H6N2? Carol Cardona, a University of California veterinary scientist, emphasizes that LPAI viruses all have the "potential to donate genetic material to potential pandemic strains. The interaction of animal

agriculture and the public is complex and dynamic and we do not fully understand the risks associated with various types of contacts between humans and birds."[154] Indeed, many researchers feel that the official distinction between LPAI and HPAI outbreaks is scientifically unsustainable and should not be allowed to dictate different levels of surveillance and response.[155] It is also imperative that agribusiness's bottom line not be allowed to supersede the global priorities of pandemic surveillance and human biosecurity. Amongst the influenzas increasingly seen in the North American poultry industry are H5 and H7 subtypes that display a disturbing tendency to rapidly evolve from LPAIs to HPAIs (Table 7.2). The full danger of not taking LPAIs seriously

Table 7.2.
H5 and H7 (LPAI) Outbreaks in the USA Since 1997[156]

1997	H7N3	Utah
1997–98	H7N2	Pennsylvania
2000	H7N2	Florida
2001	H7N2	Pennsylvania, Maryland, Connecticut
2002	H7N2	Shenandoah Valley, New York, New Jersey
2002	H5N3	Texas
2002	H5N2	New York, Maine, California
2002	H5N8	New York
2002	H5N1	Michigan
2003	H7N2	Connecticut, Rhode Island
2003	H7N2	Human infection in New York
2004	*H5N2*	*Texas (HPAI)*
2004	H7N2	Maryland, Delaware, New Jersey

as human health threats was demonstrated in British Columbia's Fraser Valley in February to May 2004.

In early February of 2004 chickens starting dying on a farm in Abbotsford, east of Vancouver. Authorities classified it as an H7N3 LPAI outbreak and denied rumors that several workers had developed flu symptoms. Canadian Food Inspection Agency officials also withheld information about possible human infection from the provincial Centre for Disease Control, an omission (according to the latter agency) that "could have had severe consequences."[157] The agricultural agents attempted to contain the outbreak within a five-kilometer hot zone, but the virus rapidly mutated to a highly deadly HPAI form, killing whole flocks. (Sequencing later confirmed that a mutation in the hemagglutinin that made it more promiscuously cleavable by host proteases was probably responsible for H7N3's enhanced ability to replicate systemically.)[158] As the epidemic approached the outskirts of Vancouver, the Canadian Food Inspection Agency ordered British Columbia to slaughter the Fraser Valley's entire domestic bird population.

Several dozen workers involved in the gassing and incineration of the 19 million chickens subsequently developed conjunctivitis and/or flu-like symptoms; two definite H7N3 cases were confirmed but the victims were infected by different strains, evidence that the virus was evolving at very high speed.[159] There was also considerable controversy about the disposal of infected chicken excrement after expert testimony that the virus might survive for as long as three months in manure. Although government spokespeople reassured the public that H7 viruses were "quite mild" and not remotely in the same league as the Asian H5N1, Canadian microbiologists warned that H7's "lower

virulence should not be inferred to indicate lower pandemic potential since subclinical or mild infections may have greater opportunity through surreptitious spread to reassort, and through mutation, to become more virulent."[160]

The provincial government's management of the outbreak was a fiasco, as even British Columbia's Minister of Agriculture, Food, and Fisheries John van Dongen conceded.[161] Simultaneous epidemics in February 2004 of highly pathogenic H5N2 in Gonzales County, Texas, and LPAI H7N2 in farms in Pennsylvania and in live bird markets in New Jersey only increased the scientific pressure on U.S. and Canadian agricultural authorities to reclassify *all* H5 and H7 outbreaks as HPAI and to expand their respective federal commitments to bird flu surveillance and intervention. The bottom line: world public health cannot afford any holes or blind spots in the pandemic early warning system. As Robert Webster has long advocated, the human-animal interface needs comprehensive monitoring, with local public-health officials around the world supplied with a suitable kit of reagents to allow them to swiftly identify any influenza subtype.[162] The chief lesson taught by the successive poultry epidemics in the Netherlands, California, and British Columbia is that, with avian influenza, the local is always global.