

The Humane Society Institute for Science and Policy
Animal Studies Repository

2007

Their Bugs Are Worse than Their Bite: Emerging Infectious Disease and the Human-Animal Interface

Michael Greger

The Humane Society of the United States

Follow this and additional works at: http://animalstudiesrepository.org/sota_2007

 Part of the [Animal Studies Commons](#), [Other Animal Sciences Commons](#), and the [Veterinary Infectious Diseases Commons](#)

Recommended Citation

Greger, M. (2007). Their bugs are worse than their bite: Emerging infectious disease and the human-animal interface. In D.J. Salem & A.N. Rowan (Eds.), *The state of the animals 2007* (pp. 111-127). Washington, DC: Humane Society Press.

This Book Chapter is brought to you for free and open access by the Humane Society Institute for Science and Policy. It has been accepted for inclusion by an authorized administrator of the Animal Studies Repository. For more information, please contact eyahner@humanesociety.org.

Their Bugs Are Worse than Their Bite: Emerging Infectious Disease and the Human-Animal Interface

6 CHAPTER

Michael Greger, M.D.

1981: Ronald Reagan takes the oath of office as president of the United States, MTV starts broadcasting, Raiders of the Lost Ark hits movie theaters, and Pac-Mania is all the rage. The Centers for Disease Control (CDC) issues a bulletin of just nine brief paragraphs: five men in Los Angeles with a strange cluster of symptoms are dying.

In the twenty-five years since that announcement, what we now know as AIDS has killed 20 million people (National AIDS Trust 2005). Where did the AIDS virus—and other emerging diseases, such as severe acute respiratory syndrome (SARS), Ebola, mad cow—come from?

The First Age of Disease

The Smithsonian Institution has identified three periods of disease since the beginning of human evolution (Armelaḡos, Barnes, and Lin 1996), and humankind's relationship with animals has played a key role in each of these "epidemiological transitions."

The first period started ten thousand years ago with the domestica-

tion of animals. When human beings confined animals to a barnyard, we corralled their diseases with them. They were not just any diseases. Species that have a herd instinct are the easiest to domesticate. Unfortunately such animals also evolved epidemic diseases that can exploit their large, dense numbers. Archeological evidence suggests that humans, on the other hand, evolved in tight hunter/gatherer bands too small to support epidemics and, as such, hardly suffered from contagious disease at all (Torrey and Yolken 2005a). Then human beings became herders, triggering what the director of Harvard University's Center for Health and the Global Environment called the mass "spillover" of animal disease into human populations (Epstein, Chivian, and Frith 2003). The World Health Organization (WHO) defined the term "zoonoses" to describe this phenomenon (Mantovani 2001), from the Greek *zoion* for "animal" and *nosos* for "disease."

Humanity's biblical "dominion over the fish of the sea and over the birds of heaven; and every living thing that moved upon the earth" has unleashed a veritable Pandora's ark full of humankind's greatest killers. Human beings domesticated

goats, and they, in turn, may have given human beings tuberculosis (Espinosa de los Monteros et al. 1998). This "captain of all these men of death" (Dubos and Dubos 1952, 8) in the last century alone killed about one hundred million people (Torrey and Yolken 2005b) and is today killing more people than ever (Reichman and Hopkins 2001). A disease that may have started out in goats now infects one-third of humanity (WHO 2000).

Tuberculosis is jumping species to this day. In a 2000 study, doctors tested children with tuberculosis in San Diego and found that one-third of the tuberculosis cases weren't human tuberculosis. They were *bovine* tuberculosis, caught, the researchers suspect, from drinking inadequately pasteurized milk from an infected cow. The investigators conclude, "These data demonstrate the dramatic impact of this underappreciated cause of zoonotic TB on U.S. children...." (Dankner and Davis 2000, E79).

When human beings first domesticated cattle, we also domesticated their rinderpest virus, which is thought to have turned into human measles (Daszak and Cunningham 2002). Now regarded as a relatively benign disease, measles

has killed two hundred million people worldwide over the last 150 years (Torrey and Yolken 2005b). In a sense, all those deaths can ultimately be traced back a few hundred generations to the taming of the first cow or bull (Diamond 1992).

Smallpox may also have been caused by a mutant cattle virus (McMichael 2001). Human beings domesticated pigs, and the result was whooping cough; we domesticated chickens and got typhoid fever; and we domesticated ducks and got influenza (Torrey and Yolken 2005b). Before then, it is likely that no one ever got the flu. Leprosy likely came from water buffalo, and the cold virus from horses (McMichael 2001). How often did wild horses have the opportunity to sneeze into humanity's face until they were broken and bridled? Before then the common cold was presumably common only to them.

Diamond (1997) explains how barnyard diseases decimated 95 percent of Native Americans, who had never before been exposed to diseases like tuberculosis, measles, and smallpox. Before Europeans arrived, bringing their goats with them, tuberculosis didn't exist in the Americas. There were no domesticated buffalo, so there was no measles or smallpox. There were no pigs, so no pertussis; no chickens, so no Typhoid Marys. While people in Europe and Asia died by the millions of killer scourges, none was dying in the New World because there were no farm animals to domesticate (Diamond 1997).

Such events aren't confined to centuries past. New diseases from domesticated farm animals continue to be discovered. *H. pylori*, a bacteria living in the human stomach, causes stomach cancer and the vast majority of peptic ulcers worldwide (De Groote, Ducatelle, and Haesebrouck 2000). Roughly half of the world's population is

now infected with it (Suerbaum and Michetti 2002). This ulcer-causing bacterium is thought to have originated in sheep's milk, but is now spread person-to-person. What is now probably the most common chronic infection afflicting humanity (Centers for Disease Control and Prevention 2005) came about because we decided thousands of years ago to start drinking the milk of another species (Dore et al. 2001).

H. pylori is not an isolated find. *H. pullorum*, a cousin of *H. pylori*, is a bacterium found in chicken meat. Hepatitis E, a new hepatitis virus that can kill pregnant women, has been found to be rampant in North American pork operations (Yoo et al. 2001). Unlike a disease like trichinosis, which only affects those who actually consume undercooked pork, once hepatitis E crosses the species line, it can be spread person-to-person. One may not have eaten infected pork, but the person from whom one got a blood transfusion may have.

The Second Age of Disease

The second great era of human disease started with the Industrial Revolution of the eighteenth and nineteenth centuries, when an epidemic of the so-called diseases of civilization, such as cancer, heart disease, stroke, and diabetes, began. These chronic diseases, considered largely preventable through changes in diet and lifestyle, now account for seven of ten deaths in the United States and the majority of deaths worldwide (Centers for Disease Control and Prevention 2006a). Interestingly, our domestication of animals also plays a role.

In 2004 WHO published its long-awaited *Global Strategy on Diet, Physical Activity, and Health*, unanimously endorsed by the United Nations' 192 member countries.

WHO is considered one of the world's most reputable sources of nutrition information because it is seen as less beholden to the multitrillion-dollar food industry than government agencies can be. (For example, U.S. government recommendations, allegedly at the sugar industry's behest, have long allowed added refined sugar to make up an astounding 25 percent of our daily caloric intake [Doyle 2003]).

WHO blames the growing epidemic of global chronic disease in part on "greater saturated fat intake (mostly from animal sources), reduced intakes of complex carbohydrates and dietary fiber, and reduced fruit and vegetable intakes." As such, it is calling for limiting the consumption of saturated animal fat and "increasing the consumption of fruits, vegetables, legumes [beans, peas, and lentils], whole grains, and nuts" (World Health Organization 2003, n.p.).

Barnard, Nicholson, and Howard (1995) estimate that meat consumption may account for up to one-quarter of the cases of heart disease in the United States, one-third of the diabetes, maybe four out of ten common cancers, half of the obesity, two-thirds of the nation's high blood pressure, and as many as three-fourths of all gallbladder operations.

M. Nestle, one of world's most highly respected nutrition experts, former director of nutrition policy at the U.S. Department of Health and Human Services, and longtime chairwoman of the nutrition department at New York University, has said, "The evidence is so strong and overwhelming and produced over such a long period of time that it is no longer debatable.... There is no question that largely vegetarian diets are as healthy as you can get" (in Liebman 1996, n.p.). The fewer animals in the human diet and the more healthy plant foods—the WHO's "fruits,

vegetables, beans, whole grains, and nuts”—the lower the risk of developing many chronic diseases (WHO 2003, n.p.).

The Third Age of Disease

By the mid-twentieth century, humankind had developed penicillin, conquered polio, and eradicated smallpox. The age of infectious disease was thought to be over. Indeed, in 1948 the U.S. secretary of state pronounced that the conquest of all infectious diseases was imminent (Najera 1989). Twenty years later the U.S. surgeon general declared victory: “The war against diseases has been won” (Crawford 2000). Even Nobel laureates were seduced into the heady optimism. To write about infectious disease, one Nobel-winning virologist wrote in the 1962 text *Natural History of Infectious Disease*, “is almost to write of something that has passed into history.” “[T]he most likely forecast about the future of infectious disease,” he pronounced, “is that it will be very dull” (Burnet and White 1962).

Then something changed. After years of declining infectious disease mortality in the United States, the last three decades have seen a reversal in that trend (Gill, Rechtschaffen, and Rubenstein 2000): the number of Americans dying from infectious diseases has started going back up (Cohen and Larson 1996). Beginning in approximately 1975 (National Agricultural Research, Extension, Education, and Economics 2004), new diseases started to surface at a pace unheard of in the annals of medicine (Epstein, Chivian, and Frith 2003)—more than thirty new diseases in thirty years, most of them newly discovered viruses (Woolhouse 2002). The concept of “emerging infectious diseases” has now changed from a mere curiosity in the field of medicine to an entire

discipline that has moved to center stage (Brown 2000). We may soon be facing, according to the Institute of Medicine, a “catastrophic storm of microbial threats” (Weinhold 2004).

We are currently living in the third era of human disease, described by medical historians as the age of “the emerging plagues” (Glasser 2004). Never in medical history have so many new diseases appeared in so short a time—and almost all of them have entered the human population from animals. Animals were domesticated ten thousand years ago: what has changed in recent decades to bring this sobering reality upon us?

Human beings have been changing the way animals live. One example: during World War II, when leading cattle-producing nations were at war, Argentina took advantage of the situation by dramatically expanding its beef industry at the expense of its forests. There human beings discovered the deadly Junin virus (or, more accurately, it discovered human beings), which is now known as the cause of Argentine hemorrhagic fever. This “hamburg-erization” of the rainforests subsequently played a role in uncovering the Machupo virus in Bolivia, the Sabia virus in Brazil, and the Venezuelan hemorrhagic fever virus in Venezuela (Hoff and Smith 2000).

Deforestation also contributes to global warming. The millions of cattle and other farm animals, and the billions of tons of their manure, are primary global contributors of the greenhouse gas methane (Mossa, Jouanyb, and Newbold 2000), which also plays a significant role in climate change (Ramanujan 2005). The warming trend could dramatically expand the reach of insect-borne diseases like the West Nile virus. According to an international panel of experts, if the average world temperature were to increase by three degrees, the zone in which malaria is spread would expand from 45

percent of the world’s population to 60 percent (Nolen 2005), causing fifty to eighty million new cases of malaria (Stapp 2004).

Inroads into Africa’s rainforests have blazed trails on which other hemorrhagic fever viruses escaped—the Lassa virus, Rift Valley Fever, and Ebola. “These zoonotic viruses seem to adhere to the philosophy that says, ‘I won’t bother you if you don’t bother me,’” (Culliton 1990, 279). But as people began “pushing back forests, or engaging in agricultural practices that are ecologically congenial to viruses, the viruses could make their way into the human population and multiply and spread” (Culliton 1990, 279).

Radical alterations of forest ecosystems can be—indeed, are—hazardous, whether in the Amazon Basin or the woods of Connecticut. Lyme disease was first recognized in New England’s forests in 1975 and has since moved across all fifty states (Dryden’s Grant Information 2005), affecting an estimated hundred thousand Americans (National Institute of Allergy and Infectious Diseases 2000). Lyme disease is spread by bacteria-infested ticks who live on deer and mice, animals with whom people have always shared wooded areas. Suburban sprawl in recent decades has chopped America’s woods into subdivisions, scaring away the foxes and bobcats who had previously kept mouse populations in check.

Cookie-cutter subdivisions weren’t the reason Africa’s rainforests were cut down. Rather, transnational timber corporations, hacking logging roads deep into the remotest regions of the continent, paved the way for a mass human migration into the rainforests to set up concessions to support the commercial logging operations. One of the main sources of food for these migrant workers is bushmeat—wild animals killed for food (Walters 2003), including upwards of twenty-six different species of primates (Avasthi 2004). Thousands

of endangered great apes—gorillas and chimpanzees—are shot, butchered, smoked, and sold for human consumption (Rose 1996). To support the logging industry's infrastructure (Rose 1998), a veritable army of commercial bushmeat hunters is bringing the great apes to the brink of extinction (Walsh et al. 2003). "These logging companies have been promoting the bushmeat trade themselves," says Fox (2000, n.p.). "It is easier to hand out shotgun shells than to truck in beef" (Fox 2000).

By cannibalizing fellow primates, human beings are exposing themselves to pathogens particularly fine-tuned to human primate physiology. Recent human outbreaks of Ebola, for example, have been traced to exposure to the dead bodies of infected great apes hunted for food (Karesh et al. 2005). Ebola, one of humanity's deadliest infections, is not efficiently spread, though, compared to a virus like human immunodeficiency virus (HIV).

The leading theory about the emergence of HIV is "direct exposure to animal blood and secretions as a result of hunting, butchering, or other activities (such as consumption of uncooked contaminated meat)" (Hahn et al. 2000). Experts believe the most likely scenario is that HIV arose from humans sawing their way into the forests of west equatorial Africa on logging expeditions, butchering chimpanzees for their flesh along the way (Laurance 2004).

In some countries the prevalence of HIV now exceeds 25 percent of the adult population (Davis and Lederberg 2001), leaving millions of orphaned children in its wake (United Nations 2004). Five people die from AIDS every minute (Lamprey et al. 2002). The most current thinking leads one to believe that, because someone butchered a chimp a few decades ago, twenty million people are now dead (National AIDS Trust 2005).

Wild animals have been hunted for a hundred thousand years, but at nothing like the current rate. Growing human populations and increasing demand for wildlife meat exceed local populations of affected species (Karesh et al. 2005). This has resulted in an enormous (and largely illegal) transboundary trade of wildlife and the setting up of intensive captive production farms in which wild animals are raised, often subjected to poor sanitation, in unnatural stocking densities before being packed together into markets for sale. These factors favor the spread and emergence of mutant strains of pathogens capable of infecting hunters, farmers, and grocery shoppers (Gilbert, Wint, and Slingenbergh 2004). Live-animal markets have been described by the director of the Wildlife Conservation Society as veritable human and animal "disease factories" (Lawrie 2004). These viral swap meets are blamed for the transformation of a class of viruses previously known for causing the common cold into a killer named SARS (Lee and Krilov 2005).

The intensive commercial bushmeat trade started in the live-animal markets of Asia (Bell, Robertson, and Hunter 2004), particularly in Guangdong, the southern province surrounding Hong Kong from which the deadly avian influenza strain H5N1 arose (Chen et al. 2004). Literature from the Southern Song Dynasty (1127–1279) describes the residents of Guangdong eating "whatever food, be it birds, animals, worms, or snakes" (Jun 2004). Today, live-animal markets cater to the unique tastes of the people of Guangdong, where shoppers can savor "Dragon-Tiger-Phoenix Soup," a brew made of snake, cat, and chicken (Bray 2005) or delicacies like *san jiao*, or "three screams"—the wriggling baby rat is said to scream first when hefted with chopsticks, a second time when dipped

into vinegar, and a third time as she or he is bitten into (Lynch 2003).

In China animals are eaten for enjoyment, sustenance, and their purported medicinal qualities. There are reports of dogs being "savagely beaten before death to increase their aphrodisiac properties" (Lawrie 2004). Cats are killed and boiled down into "cat juice," used to treat arthritis. Many of the cats are captured ferals in ill health, so "consuming such diseased cats is a time bomb waiting to explode," claimed the chief veterinarian of the Australian RSPCA.

The cat-like masked palm civet has been a popular commodity in Chinese animal markets (Brummitt 2004). Civets are raised for their flesh, and the civet cat penis is soaked in rice wine for use as an aphrodisiac (Bell, Robertson, and Hunter 2004). These animals also produce the most expensive coffee in the world (Kasper n.d.). So-called fox-dung coffee is produced by feeding coffee beans to captive civets and then recovering the partially digested beans from the feces (Marshall 1999). A musk-like substance of buttery consistency secreted by the anal glands gives the coffee its characteristic flavor and smell (William 2003).

The masked palm civet has been blamed for the SARS epidemic (Lee and Krilov 2005). "A culinary choice in south China," one commentator summed up in *Lancet*, "led to a fatal infection in Hong Kong, and subsequently to 8,000 cases of severe acute respiratory syndrome (SARS), and nearly 1,000 deaths in thirty countries on six continents" (Mack 2005). Ironically, one reason civets are eaten is for protection from respiratory infections (Davis 2005c). As noted in *The China Daily*, "We kill them. We eat them. And, then, we blame them" (Ming 2004, n.p.).

Viruses can escape the rain forests in animals living or dead, as pets or as meat. The international trade in exotic pets is a multibillion-dollar

industry, and exotic pets can harbor exotic germs (Avasthi 2004). Wildlife trafficking—the illegal trade in wildlife and wildlife parts—is a soaring black market worth \$10 billion a year in the United States alone (U.S. Department of State 2005). The United States imports an unbelievable 350,000 different species of live animals. The deputy director of the U.S. Fish and Wildlife Service testified before a Senate committee in 2003 that the United States imports more than 200 million fish, 49 million amphibians, 2 million reptiles, 365,000 birds, and 38,000 mammals in a single year (Weinhold 2004).

Whether for exotic pets or exotic cuisine, imported animals transported together under cramped conditions end up in holding areas in dealer warehouses, where they—and their viruses—can mingle further. The 2003 monkeypox outbreak across half a dozen states in the Midwest was traced to monkeypox-infected Gambian giant rats shipped to a Texas animal distributor, along with eight hundred other small mammals snared from the African rain forest. The rodents were housed with prairie dogs, who contracted the disease and made their way into pet stores and swap meets via an Illinois distributor. One week the virus is in a rodent in the dense jungles of Ghana, along the Gold Coast of West Africa—a few weeks later, that same virus finds itself in a three-year-old Wisconsin girl whose mother bought her a little prairie dog at a 4-H swap meet. “Basically you factored out an ocean and half a continent by moving these animals around and ultimately juxtaposing them in a warehouse or a garage somewhere,” said Wisconsin’s chief epidemiologist (Marchione 2003). As one expert quipped, “It was probably easier for a Gambian rat to get into the United States than [it was for] a Gambian” (Marchione 2003).

Bird smuggling may actually have been what brought the West Nile virus to the Western hemisphere

(Johnson 2003). West Nile hit New York in 1999 and has since spread across forty-eight states and Canada (Stapp 2004), with thousands of cases in 2005 and more than a hundred deaths (Centers for Disease Control and Prevention 2006b). Its continued expansion suggests that the virus has become permanently established in the United States, all, perhaps, because of a single, illegally imported pet bird (Ludwig et al. 2003).

This movement of disease agents can also threaten wildlife. The greatest animal plague ever recorded was the “Great Rinderpest Pandemic” at the end of the nineteenth century. The use of cattle by the Italian army to pull gun carriages into sub-Saharan Africa is thought to have triggered the outbreak of rinderpest, a measles-like disease of cloven-hoofed animals that wiped out not only up to 95 percent of cattle in some countries (Waltner-Toews 2002), but also up to 90 percent of other large ungulate species such as African buffalo and giraffe (Alfonso 1999). Societies based on the cattle economy were devastated. As one Masai man described, the corpses of cattle and people were “so many and so close together that the vultures had forgotten how to fly” (Plowright 1982). No longer can natural barriers like the Saharan desert protect populations against the spread of epidemic disease.

A contemporary example is an emerging fungal disease discovered in 1998 (Williams et al. 2002) that causes massive die-offs and even extinctions of amphibian wildlife across five continents (Williams et al. 2002). Ecologists now suspect the international restaurant trade in the North American bullfrog (for its fleshy legs) may have played a key role in global dissemination of this disease (Ginsburg 2004).

According to WHO’s coordinator for zoonoses control, “The chief risk factor for emerging zoonotic diseases is environmental degrada-

tion by humans.” This includes degradation wrought by global climate change, deforestation, and, as described by WHO, “industrialization and intensification of the animal production sector” (WHO and Office International des Epizooties 1999, n.p.).

In 2005 China, the world’s largest producer of pork (RaboBank International 2003), suffered an unprecedented outbreak in scope and lethality of *Streptococcus suis*, a newly emerging zoonotic pig pathogen (Gosline 2005). *Strep. suis* is a common cause of meningitis in intensively farmed pigs worldwide (Merck Veterinary Manual, n.p.) and presents most often as meningitis in people as well (Huang et al. 2005), particularly those who butcher infected pigs or handle infected pork products (Gosline 2005). Due to involvement of the auditory nerves connecting the inner ears to the brain, half of the disease’s human survivors are rendered deaf (Altman 2005).

WHO reported that it had never seen so virulent a strain (Nolan 2005) and blamed intensive confinement conditions as a predisposing factor in its sudden emergence, given the stress-induced suppression of the pigs’ immune systems (WHO 2005). The U.S. Department of Agriculture (USDA) explains that these bacteria can exist as a harmless component of a pig’s normal bacterial flora, but stress due to factors like crowding and poor ventilation can drop the animal’s defenses long enough for the bacteria to become invasive and cause disease (USDA 2005b). China’s assistant minister of commerce admitted that the disease was “found to have direct links with the foul environment for raising pigs” (China View 2005, n.p.). The disease can spread through respiratory droplets or directly via contact with contaminated blood on improperly sterilized castration scalpels, tooth-cutting pliers, or tail-docking knives (Du 2005).

China boasts an estimated fourteen thousand concentrated animal-feeding operations (CAFOs) (Nierenberg 2005), colloquially known as factory farms, which tend to have stocking densities conducive to the emergence and spread of disease (Arends et al. 1984).

The United States is the world's second-largest pork producer (FAO-STAT Database 2005), and *Strep. suis* infection is also an emerging pathogen in North America pig production, especially in intensive confinement settings (Du 2005). According to *The Journal of Swine Health and Production*, human cases of meningitis in North America are likely underdiagnosed and misidentified (Gottschalk 2004) due to the lack of adequate surveillance (Cole, Todd, and Wing 2000). WHO encourages careful pork preparation (WHO 2005), and North American agriculture officials urge *Strep. suis* disease awareness for people "who work in pig barns, processing plants, as well as in the home kitchen" (Du 2005, n.p.).

The first human case of *Strep. suis* was not in Asia or in the United States, but in Europe. The Dutch pig belt, extending into parts of neighboring Belgium and Germany, has the densest population of pigs in the world, more than twenty thousand per square mile. This region has been hit in recent years with major epidemics of hog cholera and foot and mouth disease, leading to the destruction of millions of animals. "With more and more pigs being raised intensively to satisfy Europe's lust for cheap pork, epidemics are inevitable," wrote MacKenzie (1998, n.p.). "And the hogs may not be the only ones to get sick."

This *Strep. suis* outbreak followed years after the emergence of the Nipah virus on an intensive industrial pig farm in Malaysia. Nipah turned out to be one of the deadliest of human pathogens, killing 40 percent of those infected, a toll that propelled it onto the U.S.

list of potential bioterrorism agents (Fritsch 2003). This virus is also noted for its "intriguing ability" to cause relapsing brain infections in some survivors (Wong et al. 2002) many months after initial exposure (Wong et al. 2001). Even more concerning, a 2004 resurgence of Nipah virus in Bangladesh showed a case fatality rate on a par with Ebola—75 percent—and showed evidence of human-to-human transmission (Harcourt et al. 2004). The Nipah virus, like all contagious respiratory diseases, is a density-dependent pathogen (U.S. Central Intelligence Agency 2006). "Without these large, intensively managed pig farms in Malaysia," the director of the Consortium for Conservation Medicine said, "it would have been extremely difficult for the virus to emerge" (Nierenberg 2005, 44).

Even industry groups like the American Association of Swine Veterinarians cite "[e]merging livestock production systems, particularly where they involve increased intensification" as a main reason why zoonotic diseases are of increasing concern. These intensive systems, in addition to their high population density, "may also generate pathogen build-ups or impair the capacity of animals to withstand infectious agents" (Meredith 2004, n.p.). Increasing consumer demand for animal products worldwide over the past few decades has led to a global explosion in massive animal agriculture operations that have come to play a key role in the third age of emerging human disease (McMichael 2004).

Whether it be from *E. coli* O157:H7 in hamburgers, antibiotic-resistant *Salmonella* in eggs, *Listeria* in hot dogs, "flesh-eating" bacteria in oysters, or *Campylobacter* in chickens and Thanksgiving turkeys, the CDC estimates that seventy-six million Americans come down with foodborne illness every year (Mead et al. 1999). In the twenty years between 1975 (around

the time when the dean of Yale's School of Medicine famously told students that there were "no new diseases to be discovered") and 1995, seventeen foodborne pathogens emerged, almost one each year (Liang 2002). According to the executive editor of *Meat Processing* magazine, "Nearly every food consumers buy in supermarkets and order in restaurants can be eaten with certainty for its safety—except for meat and poultry products" (Bjerklie 1999).

Animals were domesticated ten thousand years ago. With billions of feathered and curly-tailed test-tubes for viruses to incubate and mutate within, a WHO official described the last few decades as "the most ambitious short-term experiment in evolution in the history of the world" (Cookson 1993, n.p.).

Global public health experts have identified specific "dubious practices used in modern animal husbandry" beyond the inherent overstocking, stress, and unhygienic conditions that have directly or indirectly launched deadly new diseases (Phua and Lee 2005). One such "misguided" brave new farm practice is the continued feeding of livestock slaughterhouse waste, blood, and excrement to save on feed costs (Stapp 2004).

Feed expenditures remain the single largest industry expense (Lawrence and Otto 2006). The livestock industry has experimented with feeding newspaper, cardboard, cement dust, and sewer sludge to farm animals (Rampton and Stauber 1997). Satchell and Hedges (1997, n.p.) report: "Cattle feed now contains things like manure and dead cats." The Animal Industry Association (1989) defends these practices, arguing that the average U.S. farm animal "eats better than the average U.S. citizen." Forcing natural herbivores like cows, sheep, and other animals to be carnivores and even cannibals has turned out to have serious public health implications.

A leading theory on the origin of bovine spongiform encephalopathy (or “mad cow disease”) is that cows got it by eating diseased sheep (Kimberlin 1992). In modern corporate agribusiness, protein concentrates (or “meat and bone meal,” euphemistic descriptions of “trimmings that originate on the killing floor, inedible parts and organs, cleaned entrails, fetuses” [Ensminger 1990]) are fed to dairy cows to increase milk production (Flaherty 1993) as well as to most other livestock (The Economist 1990). Nearly ten million metric tons of slaughterhouse waste is fed to livestock every year (WHO and Office International des Epizooties 1999). Recycling the remains of infected cattle into cattle feed was probably what led to the British mad cow epidemic’s explosive spread (Collee 1993) to nearly two dozen countries around the world in the subsequent twenty years (USDA 2005a). Dairy producers can use corn or soybeans as a protein feed supplement, but slaughterhouse by-products can be cheaper (Albert 2000).

The meat industry has long known that cannibalistic feeding practices could have human health consequences, as *Salmonella* epidemics in poultry linked to the recycling of animal remains back into animal feed had been described well before the mad cow disease epidemic (Waltner-Toews 2002). Despite the known potential hazards to humans, the meat industry remains opposed to a total ban on feeding slaughterhouse waste, blood, and excrement to farm animals (Murphy 2003).

In 2004 the Worldwatch Institute (2004) published *Meat: Now, It’s Not Personal*, whose title alludes to intensive methods of production that have placed all human beings at risk, regardless of what they eat. In the age of antibiotic resistance, which has been fueled by the industrial feeding of antibiotics to farm animals to promote growth, a sim-

ple scrape can turn into a mortal wound, and a simple surgical procedure can be anything but simple. At least these “superbugs” are not effectively spread from person to person. Given the propensity of industrial animal agriculture to churn out novel lethal pathogens, what if they produced a virus capable of a global pandemic?

Last Great Plague

The dozens of emerging zoonotic disease threats that have characterized this third era of human disease must be put into context. *Strep. suis* infected scores of human beings and killed dozens. Nipah infected hundreds and killed scores. SARS infected thousands and killed hundreds. AIDS has infected millions. Only one virus we know of can infect billions—influenza.

Influenza, the “last great plague of man” (Kaplan and Webster 1977), is the only known pathogen capable of truly global catastrophe (Silverstein 1981). Unlike other devastating infections like malaria, which is confined equatorially, or HIV, which is only fluid-borne, influenza is considered by the CDC’s K. Fukuda to be the only pathogen carrying the potential to “infect a huge percentage of the world’s population inside the space of a year” (in Davies 1999, n.p.).

Because of its extreme mutation rate, influenza is a perpetually emerging disease. A. Fauci, NIH’s pandemic planning czar, calls it “the mother of all emerging infections” (Davis 2005b, n.p.). In its 4,500 years of infecting humans since the first domestication of wild birds, influenza has always been one of the most contagious pathogens (Taylor 2005). Only since 1997 has it also emerged as one of the deadliest.

H5N1, the new killer strain of avian influenza spreading out of Asia, had only killed about a hundred people by mid-2006 (WHO 2006). In a world in which millions

die of diseases like malaria, tuberculosis, and AIDS, why is there so much concern about bird flu? The answer is, because the flu has killed before. An influenza pandemic in 1918 became the deadliest plague in human history, killing up to a hundred million people around the world (Johnson and Mueller 2002). The 1918 flu virus was likely a bird flu virus (Belshe 2005); that virus made more than a quarter of all Americans ill and killed more people in twenty-five weeks than AIDS has killed in twenty-five years (Barry 2004). In 1918 the case mortality rate was less than 5 percent (Frist 2005). H5N1 has so far officially killed *half* of its human victims (WHO 2006).

H5N1 took its first human life in Hong Kong in 1997 (Davies 1999) and has since rampaged west to Russia, the Middle East, Africa, and Europe (Lancet Infectious Diseases 2006). It remains almost exclusively a disease of birds, but as the virus has spread, it has continued to mutate. It has become more lethal and more environmentally stable and has begun taking more species under its wing (Stöhr 2005). Influenza viruses don’t typically kill mammals like rodents, but experiments have shown that the latest H5N1 mutants can kill 100 percent of infected mice, practically dissolving their lungs (Garrett 2005). “This is the most pathogenic virus that we know of,” declared one lead investigator. “One infectious particle—one single infectious virion—kills mice. Amazing virus” (Drexler 2002, 180).

The virus also started killing cats, both pets (WHO 2004) and tigers and leopards in zoos (Keawcharoen et al. 2004). Before H5N1 no influenza virus was known even to make felines sick (Kuiken et al. 2004). According to WHO (2004, n.p.), “The reported infection of domestic cats with H5N1 is an unusual event in what is an historically unprecedented situation.”

Currently in humans H5N1 is good at killing, but not at spread-

ing. Three essential conditions are necessary to produce a pandemic.

- A new virus must arise from an animal reservoir, such that humans have no natural immunity to it.
- The virus must evolve to be capable of killing human beings efficiently. (H5N1 has met these first two conditions.)
- The virus must succeed in jumping efficiently from one human to the next. (For H5N1 it's one small step to man, but one giant leap to mankind!)

If the bird flu virus triggers a human pandemic, it will not be peasant farmers in Vietnam dying after handling dead birds or raw poultry—it may be New Yorkers, Parisians, Londoners, and people in every city, township, and village in the world dying after shaking someone's hand, touching a door-knob, or simply inhaling in the wrong place at the wrong time.

Mathematical models suggest that it might be possible to snuff out an emerging flu pandemic at the source if caught early enough (Ferguson et al. 2005; Longini et al. 2005), but practical considerations may render this an impossibility (Center for Infectious Disease and Research Policy 2005). Even if we were able to stamp it out, as long as the same underlying conditions remain, the virus would presumably soon pop back up again as it has in the past (Heiberg 2005).

The current dialogue surrounding avian influenza speaks of a potential H5N1 pandemic as if it were a natural phenomenon—like hurricanes, earthquakes, or even a “viral asteroid on a collision course with humanity” (Davis 2005a, n.p.)—which human beings could not hope to control. The reality, however, is that the next pandemic may be more of an *unnatural* disaster of our own design.

Bird flu in chickens has gone from an exceedingly rare disease to one that crops up every year. The number of serious outbreaks in the

first few years of the twenty-first century has already exceeded the total number of outbreaks recorded for the entire twentieth century. As a leading flu scientist told *Science*, “We've gone from a few snowflakes to an avalanche” (Enserink 2005, 341).

The increase in chicken outbreaks has gone hand-in-hand with more transmission to humans. A decade ago, human infection with bird flu was essentially unheard of. Since H5N1 emerged in 1997, chicken viruses H9N2 infected children in China in 1999 and 2003, H7N2 infected residents of New York and Virginia in 2002 and 2003, H7N7 infected people in the Netherlands in 2003, and H7N3 infected poultry workers in Canada in 2004 (Enserink 2005) and a British farmer in 2006. The bird flu virus in the Netherlands outbreak infected more than a thousand people (Enserink 2005). To slow down or stop this sudden, rapid, recent emergence of highly pathogenic flu viruses, humane beings must understand what has triggered this “avalanche” in the first place.

Free-ranging flocks and wild birds have been blamed for the recent emergence of H5N1, but people have kept chickens in their backyards for thousands of years, and birds have been migrating for millions. What has changed in recent years that led us to this current crisis? At a November 2005 Council on Foreign Relations Conference on the Global Threat of Pandemic Influenza, the senior correspondent of the PBS television program *The NewsHour with Jim Lehrer*, R. Suarez, asked such a question of the “godfather of flu research” (Council on Foreign Relations 2005), R. Webster.

SUAREZ: Was there something qualitatively different about this last decade that made it possible for this disease to do something that it either hasn't done before...a change in conditions that suddenly lit

a match to the tinder?

WEBSTER: [F]arming practices have changed. Previously, we had backyard poultry...Now we put millions of chickens into a chicken factory next door to a pig factory, and this virus has the opportunity to get into one of these chicken factories and make billions and billions of these mutations continuously. And so what we've changed is the way we raise animals and our interaction with those animals. And so the virus is changing in those animals and now finding its way back out of those animals into the wild birds. That's what's changed. (Council on Foreign Relations 2005, n.p.)

The big change in the ecology of avian influenza has been the industrialization of the global poultry sector. Over the last few decades, meat and egg consumption has exploded in the developing world (Kazmin 2004), leading to industrial-scale commercial chicken farming, the perfect environment for the emergence and spread of new superstrains of influenza. When tens of thousands of animals are crammed into filthy, football-field-size sheds to stand beak-to-beak in their own manure, human beings are asking for trouble.

WHO in part blames the emergence of deadly Asian viruses—such as H5N1, SARS, and Nipah—on the “over-consumption of animal products” and intensive animal agriculture (Oshitani, n.d.). The World Organization for Animal Health blames in part the shorter production cycles and greater animal densities of modern poultry production, which result in “greater number of susceptible animals reared per given unit of time” (Capua and Marangon 2003, n.p.).

The Food and Agriculture Organization of the United Nations (FAO) notes that

[T]here seems to be an acceleration of the human influenza

problems over the last few decades, involving an increasing number of species, and this is expected to largely relate to intensification of the poultry (and possibly pig) production. (Gilbert, Wint, and Slingenbergh 2004, n.p.)

The FAO elaborates in an internal document:

[C]hicken-to-chicken spread, particularly where assisted by intensive husbandry conditions, promotes the virus to shift (adaptation) to more severe type (highly pathogenic type) of infection.... Intensive production conditions favor rapid spread of infection within units and “hotting-up” of virus from low pathogenicity to a highly pathogenic types. (FAO 2004, n.p.)

The United Nations specifically calls on governments to fight what it calls factory farming:

Governments, local authorities, and international agencies need to take a greatly increased role in combating the role of factory farming [which combined with live bird markets] provide[s] ideal conditions for the virus to spread and mutate into a more dangerous form. (United Nations 2005, n.p.)

All bird flu viruses seem to start out harmless to both birds and people. In its natural state, the influenza virus has existed for millions of years as an innocuous, intestinal, waterborne infection of aquatic birds such as ducks (Webster et al. 1992). How does a duck’s intestinal bug end up in a human cough?

In the viruses’ natural aquatic bird reservoir, the duck doesn’t get sick, because the virus doesn’t need to make the duck sick to spread. In fact, it’s in the virus’s best interest for the bird *not* to get sick so as to spread farther. After all, dead ducks don’t fly. The virus silently multiplies in the duck’s intestinal lining

to be excreted into the pond water and then swallowed by another duck who alights for a drink; the cycle continues as it has for millions of years, and no one gets hurt.

If, for example, an infected duck is dragged to a live poultry market, though, and crammed into a cage stacked high enough to splatter virus-laden droppings over many different species of land-based birds, the virus then has a problem. No longer can the virus rely on the ease of pond water spread: it must mutate or die (Shortridge 1992). Thankfully for the virus, mutating is what influenza viruses do best (Suarez 2000). In aquatic birds the virus is perfectly adapted in total evolutionary stasis (Webster 1998), but, when thrown into a new environment—land-based birds like chickens—it quickly starts mutating to adapt to the new host (Suarez et al. 1998). In the open air, it must resist dehydration (Dronamraju 2004), for example, and may spread to other organs to find a new way to travel. Sometimes it finds the lungs.

The more virulent the virus becomes, the quicker it may be able to overwhelm the immune system of its new victims (Van Blerkom 2003), but it must take care not to become too deadly. In an outdoor setting, if the virus kills the host too quickly, the animal may be dead before it has a chance to infect another. So there’s a limit to how virulent these viruses can get (Dimmock, Easton, and Leppard 2001)—or at least there was until now.

Enter intensive poultry production.

When the next beak is inches away, there may be fewer limits to how nasty the virus can get. Evolutionary biologists believe that this is the key to the emergence of so-called predator-like (McGirk, Adiga, and Glacier 2005) viruses like H5N1—disease transmission from immobilized hosts (Ewald 1994). When you have a situation where the healthy animals can’t escape the diseased, then there

may be no stopping rapidly mutating viruses from becoming truly ferocious (Rennie 2005).

This may have been what occurred in the crowded trenches, troop transports, and army camps of World War I leading up to the 1918 pandemic. Boxcar capacity was labeled “eight horses or forty men” (Byerly 2005, 94). Millions of people were forced into close quarters where there was no escaping a sick comrade. This may have been where the flu virus of 1918 gained its virulence (Byerly 2005).

From the virus’s point of view, these same trench warfare conditions exist today in every industrial chicken shed. Birds are intensively confined, crowded, and stressed, not just by the millions but by the *billions*. Mabbett (2005, 34) offers a concise explanation of the role of large-scale poultry production:

The AI virus lives harmlessly in the ducks popular in Asia to control insect pests and snails in rice paddies. If this duck ‘flu passes to chickens kept nearby, it can mutate into a deadly and highly contagious strain that speeds rapidly with accompanying high mortality. The larger the flocks and the more intensive the production level, the more scope there is for the disease to spread for genetic changes to the virus.

The industry admits to

[T]he growing realization that viruses previously innocuous to natural host species have in all probability become more virulent by passage through large commercial populations. (Shane 2005, 22)

Unfortunately for us, through some quirk of evolution, the respiratory tract of a chicken seems to bear a striking resemblance (on a virus receptor level) to our own respiratory tract (Gambaryan, Webster, and Matrosovich 2002). So as the virus gets better at infecting and killing chickens, it may be getting better at infecting and killing us.

Virologist E. Brown is a specialist in the evolution of influenza viruses: “You have to say that high intensity chicken rearing is a perfect environment for generating virulent avian flu virus” (in Bueckert 2004, 6). To lower the risk of generating increasingly dangerous bird flu viruses, the global poultry industry must reverse course away from greater intensification.

Might not human beings want birds confined indoors away from waterfowl, though? Does it matter from a public health standpoint if the environment inside poultry sheds can transform harmless viruses into deadly viruses if the harmless virus can’t get inside in the first place? Unfortunately, studies have uncovered widespread disregard for this so-called biosecurity (Schmit 2005)—even in the United States, where the industry claims to have the best biosecurity in the world (Canning 2005, n.p.).

According to Vaillancourt (2002, 12): “High biosecurity and proper monitoring are still wishful thinking in many areas of intensive poultry production.” A 2002 bird flu outbreak in Virginia led to the deaths of millions of birds and found its way inside two hundred farms (Senne, Holt, and Akey 2003), highlighting just how wishful is the thinking that industrial poultry populations are biosecure. Based on the rapid spread of bird flu in the United States in 2002, leading USDA poultry researchers concluded the obvious: “[B]iosecurity on many farms is inadequate” (Suarez, Spackman, and Senne 2003, 896).

University of Maryland researchers surveyed commercial chicken facilities throughout the Delmarva Peninsula, perhaps the densest concentration of chickens in the world, and concluded that U.S. flocks “are constantly at risk of infection triggered by poor biosecurity practices” (Tablante et al. 2002, 896).

The intensive global poultry industry is not only playing with

fire with no way to put it out, but it is also fanning the flames, and firewalls to contain the virus do not exist. “Unfortunately,” leading USDA poultry virologist D. Senne told an international gathering of bird flu scientists, “that level of biosecurity does not exist in U.S. poultry production and I doubt that it exists in other parts of the world” (in Stegeman 2003, n.p.). S.M. Shane (2003, 22) notes a “decline in the standards of biosecurity in an attempt to reduce costs in competitive markets.” The decline is a contributing factor, Shane concludes, in the frequency and severity of disease outbreaks.

Biosecurity measures as currently practiced are better than nothing but may not be something on which to stake millions of human lives for the sake of cheaper chicken. A pandemic of H5N1, or a comparable future bird flu virus, has the capacity to spark the greatest medical catastrophe of all time. It may be wiser to move away from intensive poultry production altogether or, at the very least, stop encouraging its movement into the developing world.

Avian health expert K. Rudd, drawing on thirty-seven years’ experience within the industry, warns:

Now is the time to decide. We can go on with business as usual, hoping for the best as we charge headlong toward lower costs. Or we can begin making the prudent moves needed to restore a balance between economics and long-range avian health. We can pay now or we can pay later. But it should be known and it must be said, one way or another we will pay. (Rudd 1995, 20)

As the United Nations has urged, combating factory farming may prevent the emergence of future viruses, but there seems little hope of eradicating H5N1. M. Osterholm, the director of the U.S. Center for Infectious Disease Research and Policy and an associate director

within the U.S. Department of Homeland Security, has tried to describe what an H5N1 pandemic could look like. He suggests policy makers consider the devastation of the 2004 tsunami in South Asia: “Duplicate it in every major urban centre and rural community around the planet simultaneously, add in the paralyzing fear and panic of contagion, and we begin to get some sense of the potential of pandemic influenza” (in Kennedy 2005, A1).

“An influenza pandemic of even moderate impact,” Osterholm writes,

[W]ill result in the biggest single human disaster ever—far greater than AIDS, 9/11, all wars in the twentieth century and the recent tsunami combined. It has the potential to redirect world history as the Black Death redirected European history in the fourteenth century. (In Kennedy 2005, A1)

One hopes the direction world history will take is away from raising birds by the billions under intensive confinement to potentially lower the risk of our ever being in this same precarious situation in the future.

Will We Survive?

Former U.S. Senate Majority Leader B. Frist described the recent slew of emerging diseases in almost biblical terms: “All of these [new diseases] were advance patrols of a great army that is preparing way out of sight” (in Dennehy 2005, n.p.). J. Lederberg, who won the Nobel Prize in medicine for his discoveries in bacterial evolution, has said,

Some people think I am being hysterical [referring to pandemic influenza], but there are catastrophes ahead. We live in evolutionary competition with microbes—bacteria and viruses. There is no guarantee that we will be the survivors. (In Culli-

ton 1990, 279)

In host-parasite evolutionary dynamics, the so-called Red Queen hypothesis attempts to describe the unremitting struggle between immune systems and the pathogens against which they fight, each constantly evolving to try to outsmart the other (Lythgoe and Read 1998). Its name is taken from L. Carroll's *Through the Looking Glass*, in which the Red Queen instructs Alice, "Now, here, you see, it takes all the running you can do to keep in the same place" (Carroll 1872, n.p.). Because the pathogens keep evolving, human immune systems have to keep adapting as well just to keep up. According to the theory, animals who "stop running" go extinct.

So far our immune systems have largely retained the upper hand, but the fear is that, given the current rate of disease emergence, the human race is losing the race (Culliton 1990). Mitchison (1993, 136) writes:

Has the immune system, then, reached its apogee after the few hundred million years it had taken to develop? Can it respond in time to the new evolutionary challenges? These perfectly proper questions lack sure answers because we are in an utterly unprecedented situation [given the number of newly emerging infections].

According to Torrey and Yolken (2005a), "Considering that bacteria, viruses, and protozoa had a more than two-billion-year head start in this war, a victory by recently arrived *Homo sapiens* would be remarkable."

J. Lederberg ardently believes that emerging viruses may imperil human society itself (in Drexler 2002). D. Morens says:

When you look at the relationship between bugs and humans, the more important thing to look at is the bug. When an enterovirus like polio goes through the human gas-

trointestinal tract in three days, its genome mutates about two percent. That level of mutation—two percent of the genome—has taken the human species eight million years to accomplish. So who's going to adapt to whom? (In Drexler 2002, 8)

Pitted against that kind of competition, Lederberg concludes that the human evolutionary capacity to keep up "may be dismissed as almost totally inconsequential" (Drexler 2002, 180). To help prevent the evolution of viruses as threatening as H5N1, the least we can do is take away a few billion feathered test-tubes in which viruses can experiment, a few billion fewer spins at pandemic roulette.

The human species has existed in something like our present form for approximately 200,000 years. "Such a long run should itself give us confidence that our species will continue to survive, at least insofar as the microbial world is concerned. Yet such optimism," wrote A. Mitchison (1993, n.p.), the Ehrlich prize-winning former chairman of zoology at the University College of London, "might easily transmute into a tune whistled whilst passing a graveyard."

According to a WHO spokesperson:

The bottom line is that humans have to think about how they treat their animals, how they farm them, and how they market them—basically, the whole relationship between the animal kingdom and the human kingdom is coming under stress. (Torrey and Yolken 2005a)

Along with human culpability, though, comes hope. If changes in human behavior can cause new plagues, changes in human behavior may prevent them in the future.

Literature Cited

- Albert, D. 2000. EU meat meal industry wants handout to survive ban. *Reuters World Report*. December 5.
- Alfonso, T. 1999. International economic considerations concerning agricultural diseases and human health costs of zoonotic diseases. *Annals of the New York Academy of Sciences* 894: 80–82.
- Altman, L.K. 2005. Pig disease in China worries UN. *New York Times*, August 5. iht.com/bin/print_ipub.php?file=/articles/2005/08/05/news/pig.php.
- Animal Industry Foundation. 1989. *Animal agriculture: Myths and facts*. Arlington, Va.: Animal Industry Foundation.
- Arends, J.P., N. Hartwig, M. Rudolph, and H.C. Zanen. 1984. Carrier rate of *Streptococcus suis* capsular type 2 in palatine tonsils of slaughtered pigs. *Journal of Clinical Microbiology* 20(5): 945–947.
- Armelaços, G.J., K.C. Barnes, and J. Lin. 1996. Disease in human evolution: The re-emergence of infectious disease in the third epidemiological transition. *National Museum of Natural History Bulletin for Teachers* 18(3).
- Avasthi, A. 2004. Bush-meat trade breeds new HIV. *New Scientist*. www.newscientist.com/article.ns?id=dn6239.
- Barnard, N.D., A. Nicholson, and J.L. Howard. 1995. The medical costs attributable to meat consumption. *Preventive Medicine* 24: 646–655.
- Barry, J.M. 2004. Viruses of mass destruction. *Fortune*, November 1.
- Bell, D., S. Robertson, and P.R. Hunter. 2004. Animal origins of SARS coronavirus: Possible links with the international trade in small carnivores. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences* 359(1447): 1107–1114.

- Belshe, R.B. 2005. The origins of pandemic influenza—Lessons from the 1918 virus. *New England Journal of Medicine* 353 (21): 2209–2211.
- Bjerklie, S. 1999. Starting over. *Meat Processing* (90).
- Bray M. 2005. Unhealthy mix of animals, humans. CNN.com International. cnm.com/2005/WORLD/asiacpf/05/03/eyeonchina.virus/.
- Brown, C. 2000. Emerging infectious diseases of animals: An overview. In *Emerging diseases of animals*, ed. C. Brown and C. Bolin, 1–12. Washington, D.C.: ASM Press.
- Brummitt, C. 2004. Indonesians enjoy civet-dropping coffee. *USA Today*. www.usatoday.com/news/offbeat/2004-01-20-civet-coffee_x.htm.
- Bueckert, D. 2004. Avian flu outbreak raises concerns about factory farms. *Daily Herald-Tribune* (Grande Prairie, Alberta), April 8: 6. cp.org/english/online/full/agriculture/040407/a040730A.html.
- Burnet, M., and D.O. White. 1962. *Natural history of infectious disease*, 4th ed. Cambridge: Cambridge University Press.
- Byerly, C.R. 2005. *Fever of war: The influenza epidemic in the U.S. army during World War I*. New York: New York University Press.
- Canning, K. 2005. A matter of pride. Refrigerated and frozen foods. www.refrigeratedfrozenfood.com/content.php?s=RF/2005/12&p=8.
- Capua, I., and S. Marangon. 2003. The use of vaccination as an option for the control of avian influenza. World Organization for Animal Health 71st General Session in Paris, France, May 18–23. www.oie.int/eng/AVIAN_INFLUENZA/A_71_percent20SG_12_CS3E.pdf.
- Carroll, L. 1872. *Through the looking glass and what Alice found there*. London: Macmillan.
- Centers for Disease Control and Prevention National Center for Infectious Diseases/Division of Bacterial and Mycotic Diseases. 2005. Helicobacter pylori infections (H. pylori). October 12. www.cdc.gov/ncidod/dbmd/diseaseinfo/hpylori_t.htm.
- Centers for Disease Control and Prevention National Center for Chronic Disease Prevention and Health Promotion. 2006a. Chronic disease prevention. June 29. www.cdc.gov/nccdphp/.
- . 2006b. West Nile virus: Statistics, surveillance, and control. February 14. www.cdc.gov/ncidod/dzbid/westnile/surv&controlCaseCount06_detailed.htm.
- Center for Infectious Disease and Research Policy. 2005. Roche to give flu drug to WHO to fight pandemic. August 24. cidrap.umn.edu/cidrap/content/influenza/panflu/news/aug2405who.html.
- Chen H., G. Deng, Z. Li, G. Tian, Y. Li, P. Jiao, L. Zhang, Z. Liu, R.G. Webster, and K. Yu. 2004. The evolution of H5N1 influenza viruses in ducks in southern China. *Proceedings of the National Academy of Sciences of the United States of America* 101(28): 10452–10457.
- China View. 2005. China drafts, revises laws to safeguard animal welfare. November 4. news.xinhuanet.com/english/2005-11/04/content_3729580.htm.
- Cohen, F.L., and E. Larson. 1996. Emerging infectious diseases: Nursing responses. *Nursing Outlook* 44: 164–168.
- Cole D., L. Todd, and S. Wing. 2000. Concentrated swine feeding operations and public health: A review of occupational and community health effects. *Environmental Health Perspectives* 108: 685–699.
- Collee, G. 1993. BSE stocktaking 1993. *Lancet* 342(8874): 790–3. www.cyber-dyne.com/~tom/essay_collee.html.
- Cookson, C. 1993. Bugs that come to plague us: The renewed war against disease. *Financial Times* (London), August 21.
- Council on Foreign Relations. 2005. Session 1: Avian flu—Where do we stand? Conference on the Global Threat of Pandemic Influenza, November 16. cfr.org/publication/9230/council_on_foreign_relations_conference_on_the_global_threat_of_pandemic_influenza_session_1.html.
- Crawford, D. 2000. *The invisible enemy: A natural history of viruses*. New York: Oxford University Press.
- Culliton, B.J. 1990. Emerging viruses, emerging threat. *Science* 247: 279–280.
- Dankner, W.M., and C.E. Davis. 2000. Mycobacterium bovis as a significant cause of tuberculosis in children residing along the United States-Mexico border in the Baja California region. *Pediatrics* 105: E79–83.
- Daszak, P., and A.A. Cunningham. 2002. Emerging infectious diseases: A key role for conservation medicine. In *Conservation medicine: Ecological health in practice*, ed. A.A. Aguirre, R.S. Ostfeld, G.M. Tabor, C. House, and M.C. Pearl, 40–61. New York: Oxford University Press.
- Davies, P. 1999. The plague in waiting. *Guardian*, August 7. guardian.co.uk/birdflu/story/0,,1131473,00.html.
- Davis, M. 2005a. Avian flu: A state of unreadiness. *The Nation*, July 18–25: 27–30.
- . 2005b. Has time run out? Commentary: On the monster at our door—The coming flu pandemic. *Mother Jones*. motherjones.com/commentary/columns/2005/08/has_time_run_out.html.
- . 2005c. *The monster at our door: The global threat of avian flu*. New York: The New Press.
- Davis, R.J., and J. Lederberg, eds. 2001. *Emerging infectious diseases from the global to the local perspective: A summary of a workshop of the forum of emerging infections*. Washington, D.C.: National Academies Press.

- darwin.nap.edu/books/0309071844/html/.
- De Groot, D., R. Ducatelle, and F. Haesebrouck. 2000. Helicobacters of possible zoonotic origin: A review. *Acta Gastro-Enterologica Belgica* 63: 380–387.
- Dennehy, K. 2005. Frist warns of pandemic future. *Cape Cod Times*, August 4.
- Diamond, J. 1992. The arrow of disease. *Discover* 13(10): 64–73.
- . 1997. *Guns, germs and steel: The fates of human societies*. New York: Norton and Company.
- Dimmock, N.J., A. Easton, and K. Leppard. 2001. *Introduction to modern virology*. Boston: Blackwell Publishing.
- Dore, M.P., A.R. Sepulveda, H. El-Zimaity, Y. Yamaoka, M.S. Osato, K. Mototsugu, A.M. Nieddu, G. Realdi, and D.Y. Graham. 2001. Isolation of *Helicobacter pylori* from sheep: Implications for transmission to humans. *American Journal of Gastroenterology* 96(5): 1396–1401.
- Doyle, W. 2003. Sugar intake: You asked a dietician. BBC News, April 28. news.bbc.co.uk/1/hi/talking_point/2981599.stm.
- Drexler, M. 2002. *Secret agents: The menace of emerging infections*. Washington, D.C.: Joseph Henry Press.
- Dronamraju, K., ed. 2004. *Infectious disease and host-pathogen evolution*. Cambridge: Cambridge University Press.
- Dryden's Grant Information. 2005. Washington and Jefferson College. Tick-borne disease research. www.washjeff.edu/tickresearch/.
- Du, W. 2005. *Streptococcus suis*, (*S. suis*) pork production, and safety. Ontario Ministry of Agriculture, Food and Rural Affairs. www.omafra.gov.on.ca/english/livestock/swine/news/novdec05a5.htm.
- Dubos, R., and J. Dubos. 1952. *The white plague: Tuberculosis, man, and society*. Boston: Little, Brown.
- Economist, The. 1990. Mad, bad and dangerous to eat? *The Economist*, February: 89–90.
- Enserink, M. 2005. Veterinary scientists shore up defenses against bird flu. *Science* 308(5720): 341.
- Ensminger, M.E. 1990. *Feeds and nutrition*. Clovis, Calif.: Ensminger Publishing Co.
- Epstein, P.R., E. Chivian, and K. Frith. 2003. Emerging diseases threaten conservation. *Environmental Health Perspectives* 111(10): A506–507.
- Espinosa de los Monteros, L.E., J.C. Galán, M. Gutierrez, S. Samper, J.F. Garcia Marin, C. Martin, L. Dominguez, L. de Rafael, F. Baquero, E. Gomez-Mampaso, and B. Blazquez J. 1998. Allele-specific PCR method based on pncA and oxyR sequences for distinguishing *Mycobacterium bovis* from *Mycobacterium tuberculosis*: Intraspecific *M. bovis* pncA sequence polymorphism. *Journal of Clinical Microbiology* 36: 239–242.
- Ewald, P. 1994. *Evolution of infectious disease*. Oxford: Oxford University Press.
- FAOSTAT Database. 2005. Agricultural data. faostat.fao.org/faostat/collections?version=ext&hasbulk=0&subset=agriculture.
- Ferguson, N.M., D.A. Cummings, S. Cauchemez, C. Fraser, S. Riley, A. Meeyai, S. Lamsirithaworn, and D.S. Burke. 2005. Strategies for containing an emerging influenza pandemic in Southeast Asia. *Nature* 437(7056): 209–214.
- Flaherty, M. 1993. Mad cow disease dispute: U.W. conference poses frightening questions. *Wisconsin State Journal*, September 26: 1C.
- Food and Agriculture Organization of the United Nations (FAO). 2004. Questions and answers on avian influenza: Briefing paper prepared by AI Task Force, International FAO document, January 30. animal-health-online.de/drms/faoinfluenza.pdf.
- Fox, M. 2000. The killer out of Africa. *Hobart Mercury* (Australia), February 9.
- Frist, B. 2005. Manhattan project for the 21st century. Harvard Medical School Health Care Policy Seidman lecture, Cambridge, Mass., June 1. frist.senate.gov/_files/060105manhattan.pdf.
- Fritsch, P. 2003. Containing the outbreak: Scientists search for human hand behind outbreak of jungle virus. *Wall Street Journal*, June 19.
- Gambaryan, A., R. Webster, and M. Matrosovich. 2002. Differences between influenza virus receptors on target cells of duck and chicken. *Archives of Virology* 147: 1197–1208.
- Garrett, L. 2005. The next pandemic? Probable cause. *Foreign Affairs* 84(4). www.foreignaffairs.org/20050701faessay84401/laurie-Garrett/the-next-pandemic.html.
- Gilbert, M., W. Wint, and J. Slingenbergh. 2004. The ecology of highly pathogenic avian influenza in East and South-east Asia: Outbreaks distribution, risk factors, and policy implications. Consultancy report for the Animal Health Service of the Animal Production and Health Division of the Food and Agriculture Organization of the United Nations, Rome, Italy.
- Gill, J.M., J.A. Rechtschaffen, and L.R. Rubenstein. 2000. Expect the unexpected: The West Nile virus wake up call. Report of the Minority Staff, Senate Governmental Affairs Committee, July 24. www.senate.gov/~gov_affairs/wnfinafinalreport.pdf.
- Ginsburg, J. 2004. Dinner, pets, and plagues by the bucketful. *The Scientist* 18(7): 28.
- Glasser, R.J. 2004. We are not immune: Influenza, SARS, and the collapse of public

- health. *Harper's Magazine*, July. www.harpers.org/WeAreNotImmune.html.
- Gosline, A. 2005. Mysterious disease outbreak in China baffles WHO. *Newscientist.com*. July. www.newscientist.com/article.ns?id=dn7740.
- Gottschalk, M. 2004. Porcine *Streptococcus suis* strains as potential sources of infections in humans: An underdiagnosed problem in North America? *Journal of Swine Health and Production* 12(4): 197–199.
- Hahn, B.H., G.M. Shaw, K.M. De Cock, and P.M. Sharp. 2000. AIDS as a zoonosis: Scientific and public health implications. *Science* 287: 607–614.
- Harcourt, B.H., L. Lowe, A. Tamin, X. Liu, B. Bankamp, N. Bowden, P.E. Rollin, J.A. Comer, T.G. Ksiazek, M.J. Hossain, E.S. Gurley, R.F. Breiman, W.J. Bellini, and P.A. Rota. 2004. Genetic characterization of Nipah virus, Bangladesh, 2004. Centers for Disease Control and Prevention, *Emerging Infectious Diseases* 11(10). www.cdc.gov/ncidod/EID/vol11no10/05-0513.htm. www.cdc.gov/ncidod/EID/vol11no10/05-0513.htm.
- Heiberg, M. 2005. Two studies model containment strategies for pandemic flu. CIDRAP News, August 3. www.cidrap.umn.edu/cidrap/content/influenza/panflu/news/aug0305panflu.html.
- Hoff, B., and C. Smith, III. 2000. *Mapping epidemics: A historical atlas of disease*. New York: Grolier Publishing.
- Huang, Y.T., L.J. Teng, S.W. Ho, and P.R. Hsueh. 2005. *Streptococcus suis* infection. *Journal of Microbiology, Immunology, and Infection* 38: 306–313. jmii.org/content/abstracts/v38n5p306.php.
- Johnson, N.P.A.S., and J. Mueller. 2002. Updating the accounts: Global mortality of the 1918–1920 “Spanish” influenza pandemic. *Bulletin of the History of Medicine* 76: 105–115.
- Johnson, R.T. 2003. Emerging viral infections of the nervous system. *Journal of NeuroVirology* 9: 140–147.
- Jun, Y. 2004. Are wild animals safe? *China Daily*, November 12. www.china.org.cn/english/environment/111979.htm.
- Kaplan, M.M., and R.G. Webster. 1977. The epidemiology of influenza. *Scientific American* 237: 88–106.
- Karesh, W.B., R.A. Cook, E.L. Bennett, and J. Newcomb. 2005. Wildlife trade and global disease. *Emerging Infectious Diseases* 11(7). www.cdc.gov/ncidod/EID/vol11no07/05-0194.htm.
- Kasper, L.R. n.d. A recipe for shower flower cake. Scripps Howard News Service. www.diynetwork.com/diy/lc_beverages/article/0,2041,DIY_13997_2278719,00.html.
- Kazmin, A. 2004. Greater livestock density blamed for disease rise. *Financial Times*, January 28.
- Keawcharoen, J., K. Oraveerakul, T. Kuiken, R.A. Fouchier, A. Amonsin, S. Payungporn, S. Noppornpanth, S. Wattanodorn, A. Theambooniers, R. Tantilertcharoen, R. Pattanarangsarn, N. Arya, P. Ratanakorn, D.M. Osterhaus, and Y. Poovorawan. 2004. Avian influenza H5N1 in tigers and leopards. *Emerging Infectious Diseases* 10: 2189–2191.
- Kennedy, M. 2005. Bird flu could kill millions: Global pandemic warning from WHO. “We’re not crying wolf. There is a wolf. We just don’t know when it’s coming.” *Gazette* (Montreal), March 9: A1.
- Kimberlin, R.H. 1992. Human spongiform encephalopathies and BSE. *Medical Laboratory Sciences* 49: 216–217.
- Kuiken, T., G. Rimmelzwaan, D. van Riel, G. van Amerongen, M. Baars, R. Fouchier, and A. Osterhaus. 2004. Avian H5N1 influenza in cats. *Science* 306: 241.
- Lampthey, P., M. Wigley, D. Carr, and Y. Collymore. 2002. Facing the HIV/AIDS Pandemic. *Population Bulletin* 57(3): 1. Washington, D.C.: Population Reference Bureau.
- Lancet Infectious Diseases. 2006. Avian influenza goes global, but don’t blame the birds. 6:185. *list.web.net/archives/shudgewatch-l/2006-April/001692.html.*
- Laurance J. 2004. New diseases pose threat to world health. *Independent*, January 14. www.findarticles.com/p/articles/mi_qn4158/is_20040114/ain9687521.
- Lawrence, J., and D. Otto. 2006. Economic importance of Montana’s cattle industry. Cattleman’s Beef Board and National Cattleman’s Beef Association. www.beef.org/NEWSECONOMICIMPORTANCEOFMONTANASCATTLEINDUSTRY2711.aspx.
- Lawrie, M. 2004. Animal welfare gains from avian influenza? *Australian Veterinary Journal* 82: 135.
- Lee, P.J., and L.R. Krilov. 2005. When animal viruses attack: SARS and avian influenza. *Pediatric Annals* 34(1): 43–52.
- Liang, A.P. 2002. Current state of foodborne illness. Conference for Food Safety Education. Orlando, Fla., September 27. fsis.usda.gov/Orlando2002/presentations/aliang/aliang.pdf.
- Liebman, B. 1996. Plants for supper? *Nutrition Action Health Letter*, October. cspinet.org/nah/10veggie.html.
- Longini, I.M., Jr, A. Nizam, S. Xu, K. Ungchusak, W. Hanshaoworakul, D.A. Cummings, and M.E. Halloran. 2005. Containing pandemic influenza at the source. *Science* 309(5737): 1083–1087.
- Ludwig, B., F.B. Kraus, R. Allwinn, H.W. Doerr, and W. Preiser. 2003. Viral zoonoses—A threat under control? *Intervirology* 46: 71–78.
- Lynch, D.J. 2003. Wild animal markets in China may be breeding SARS. *USA Today*, October 28.
- Lythgoe, K.A., and A.F. Read. 1998. Catching the Red Queen? The advice of the rose. *Trends Ecology and Evolution* 13: 473–474.

- Mabbett, T. 2005. People, poultry, and avian influenza. *Poultry International* (44)9: 34–39.
- Mack, T.M. 2005. The ghost of pandemics past. *Lancet* 365(9468): 1370–1372.
- MacKenzie, D. 1998. This little piggy fell ill. *New Scientist*, September 12: 1818.
- Mantovani, A. 2001. Notes on the development of the concept of zoonoses. WHO Mediterranean Zoonoses Control Centre Information Circular 51. www.mzcp-zoonoses.gr/pdf/en/circ_51.pdf.
- Marchione, M. 2003. Globetrotting boosts exotic diseases. *Milwaukee Journal Sentinel*, June 15: 1. www.findarticles.com/p/articles/mi_qn4196/is_20030615/ai_n10879847.
- Marshall, S. 1999. Coffee that satisfies a discerning civet cat is excellent indeed. *Wall Street Journal*, March 17.
- McGirk, T., A. Adiga, and S. Glacier. 2005. Will the next pandemic rival 1918? *Times Asia*, July 4. time.com/time/asia/magazine/printout/0,13675,501050711-1079528,00.html.
- McMichael, A.J. 2004. Environmental and social influences on emerging infectious diseases: Past, present, and future. *Philosophical Transactions of the Royal Society of London* 359: 1049–1058.
- McMichael, T. 2001. *Human frontiers, environments, and disease*. Cambridge: Cambridge University Press.
- Mead, P.S., L. Slutsker, V. Dietz, L.F. McCaig, J.S. Bresee, C. Shapiro, P.M. Griffin, and R.V. Tauxe. 1999. Food-related illness and death in the United States. *Emerging Infectious Diseases* 5(5). www.cdc.gov/ncidod/eid/vol5no5/mead.htm.
- Merck Veterinary Manual. n.d. *Streptococcus suis* infection. [merckvetmanual.com/mvm/index.jsp?cfile=htm/bc/54302.htm&word=Strep percent2csuis](http://merckvetmanual.com/mvm/index.jsp?cfile=htm/bc/54302.htm&word=Strep%20suis).
- Meredith, M. 2004. Zoonotic disease risks—2004 update. *American Association of Swine Veterinarians*. October 1. www.aasv.org/news/story.php?id=1221.
- Ming, Z. 2004. Humans should shoulder blame for SARS. *China Daily*, October 14. english.people.com.cn/200410/12/eng20041012_159879.html.
- Mitchison, A. 1993. Will we survive? As host and pathogen evolve together, will the immune system retain the upper hand? *Scientific American*, September, 136–144.
- Mossa, A.R., P. Jouanyb, and J. Newbold. 2000. Methane production by ruminants: Its contribution to global warming. *Annales De Zootechnie* 49: 231–253.
- Murphy, D. 2003. FDA changes in feed restriction won't reduce BSE risk, industry groups say. *Meatingplace.com*, January 15.
- Najera, J.A. 1989. Malaria and the work of the WHO. *Bulletin of the World Health Organization* 67: 229–243.
- National Agricultural Research, Extension, Education, and Economics. 2004. Protecting our food system from current and emerging animal and plant diseases and pathogens: Implications for research, education, extension, and economics. NAREEE Advisory Board Meeting and Focus Session, Washington Court Hotel, Washington, D.C., October 27–29.
- National AIDS Trust. 2005. Global statistics. Fact sheet. www.worldaidsday.org/files/stats_global_2005.doc.
- National Institute of Allergy and Infectious Diseases. 2000. Lyme disease vaccine: Preventing an emerging disease, January 13. niaid.nih.gov/publications/discovery/lyme.htm.
- Nierenberg, D. 2005. Happier meals: Rethinking the global meat industry. *Worldwatch* paper 171, September. www.world-watch.org/pubs/paper/171/.
- Nolan, T. 2005. 40 people die from pig-borne bacteria. AM radio transcript. www.abc.net.au/cm/content/2005/s1441324.htm.
- Nolen, R.S. 2005. Tug-of-war: Steps must be taken to turn tide of public-microbial war. *Journal of the American Veterinary Medical Association News*, February 15. www.avma.org/onlnews/javma/feb05/050215c.asp.
- Oshitani, H. n.d. Communicable diseases in the Western Pacific region. Inaugural ceremony of the Scientific Advisory Structure of the Centre for Health Protection, Department of Health, Hong Kong. www.info.gov.hk/gia/general/200406/23/ppt2.pdf.
- Phua, K., and L.K. Lee. 2005. Meeting the challenges of epidemic infectious disease outbreaks: An agenda for research. *Journal of Public Health Policy* 26: 122–32.
- Plowright, W. 1982. The effects of rinderpest and rinderpest control on wildlife in Africa. *Symposia of the Zoological Society of London* 50: 1–28.
- RaboBank International 2003. China's meat industry overview. Food and Agribusiness Research. May. www.rabobank.com/Images/rabobank_publication_china_meat_2003_tcm25-139.pdf.
- Ramanujan, K. 2005. Methane's impacts on climate change may be twice previous estimates. PhysOrg.com.physorg.com/news5258.html.
- Rampton, S., and J. Stauber. 1997. *Mad Cow USA*. Monroe, Me.: Common Courage Press.
- Reichman, L., with J. Hopkins. 2001. *Timebomb: The global epidemic of multi-drug resistant tuberculosis*. New York: McGraw Hill.
- Rennie, J. 2005. Bird reaper, Part III: Paul Ewald replies. *SciAm Observations*, November 2. blog.sciam.com/index.php?title=bird_reaper_pt_iii_paul_ewald_replies&more=1&c=1&tb=1&pb=1.

- Rose, A.L. 1996. The African great ape bushmeat crisis. *Pan Africa News* 3(2): 1–6.
- . 1998. Growing commerce in bushmeat destroys great apes and threatens humanity. *African Primates* 3: 6–10.
- Rudd, K. 1995. Poultry reality check needed. *Poultry Digest*, December: 12–20.
- Satchell, M., and S.J. Hedges. 1997. The next bad beef scandal? Cattle feed now contains things like manure and dead cats. *U.S. News and World Report*, September 1.
- Schmit, J. 2005. Poultry farm tactics may thwart bird flu. *USA Today*, November 14. usatoday.com/news/nation/2005-11-13-farmers-birdflu_x.htm?esp=N009.
- Senne, D.A., T.J. Holt, and B.L. Akey. 2003. An overview of the 2002 outbreak of low-pathogenic H7N2 avian influenza in Virginia, West Virginia, and North Carolina. In *Proceedings of the Frontis workshop on Avian influenza: Prevention and control*, ed. R.S. Schrijver and G. Koch, 41–47. Wageningen, The Netherlands.
- Shane, S.M. 2003. Disease continues to impact the world's poultry industries. *World Poultry* 19(7): 22–27.
- . 2005. Global disease update: AI overshadowing erosive diseases. *World Poultry* 21(7): 22–23.
- Shortridge, K.F. 1992. Pandemic influenza: A zoonosis? *Seminars in Respiratory Infections* 7: 11–25.
- Silverstein, A.M. 1981. *Pure politics and impure science: The swine flu affair*. Baltimore: Johns Hopkins University Press.
- Stapp, K. 2004. Scientists warn of fast-spreading global viruses. IPS-Inter Press Service, February 23.
- Stegeman, A. 2003. Workshop 1: Introduction and spread of avian influenza. In *Proceedings of the Frontis workshop on avian influenza: Prevention and control*, ed. R.S. Schrijver and G. Koch. library.wur.nl/frontis/avian_influenza/workshop1.pdf.
- Stöhr, K. 2005. Avian influenza and pandemics: Research needs and opportunities. *New England Journal of Medicine* 352(4): 405–407.
- Suarez, D.L. 2000. Evolution of avian influenza viruses. *Veterinary Microbiology* 74: 15–27.
- Suarez, D.L., E. Spackman, and D.A. Senne. 2003. Update on molecular epidemiology of H1, H5, and H7 influenza virus infections in poultry in North America. *Avian Diseases* 47: 888–897.
- Suarez, D.L., M.L. Perdue, N. Cox, T. Rowe, C. Bender, J. Huang, and D.E. Swayne. 1998. Comparisons of highly virulent H5N1 influenza A viruses isolated from humans and chickens from Hong Kong. *Journal of Virology* 72: 6678–6688.
- Suerbaum, S., and P. Michetti. 2002. Helicobacter pylori infection. *New England Journal of Medicine* 347: 1175–1186.
- Tablante, N.L., M. San Myint, Y.J. Johnson, K. Rhodes, M. Colby, and G. Hohenhaus. 2002. A survey of biosecurity practices as risk factors affecting broiler performance on the Delmarva Peninsula. *Avian Diseases* 46: 730–734.
- Taylor, M. 2005. Is there a plague on the way? *Farm Journal*, March 10. www.agweb.com/get_article.asp?pageid=116037.
- Torrey, E.F., and R.H. Yolken. 2005a. *Beasts of the earth: Animals, humans, and disease*. New Brunswick, N.J.: Rutgers University Press.
- . 2005b. Their bugs are worse than their bite. *Washington Post*, April 3: B01.
- United Nations. 2004. AIDS orphans in sub-Saharan Africa: A looming threat to future generations. www.un.org/events/tenstories/story.asp?storyID=400.
- . 2005. UN task forces battle misconceptions of avian flu, mount Indonesian campaign. UN News Centre, October 24. un.org/apps/news/story.asp?NewsID=16342&Cr=bird&Cr1=flu.
- U.S. Central Intelligence Agency. 2006. Malaysia. CIA world fact book. March 29. cia.gov/cia/publications/factbook/geos/my.html.
- U.S. Department of Agriculture/Animal and Plant Health Inspection Service. 2005a. List of USDA-recognized animal health status of countries/areas regarding specific livestock or poultry diseases, April, 12. oars.aphis.usda.gov/NCIE/country.html.
- U.S. Department of Agriculture, Veterinary Services, Center for Emerging Issues. 2005b. Streptococcus suis outbreak, swine and human, China: Emerging disease notice. www.aphis.usda.gov/vs/ceah/cei/taf/emergingdisease_notice_files/strep_suis_china.htm.
- U.S. Department of State. 2005. United States announces global coalition against animal trafficking. Office of the spokesman. September 23. www.state.gov/r/pa/prs/ps/2005/53926.htm.
- Vaillancourt, J.P. 2002. Biosecurity now. *Poultry International* 411: 12–18.
- Van Blerkom, L.M. 2003. Role of viruses in human evolution. *Yearbook of Physical Anthropology* 46: 14–46. tinyurl.com/ksh92.
- Walsh, P.D., K.A. Abernethy, M. Bermejo, R. Beyers, P. De Wachter, M.E. Akou, B. Huijbregts, D.I. Mambounga, A.K. Toham, A.M. Kilbourn, S.A. Lahm, S. Latour, F. Maisels, C. Mbina, Y. Mihindou, S.N. Obiang, E.N. Effa, M.P. Starkey, P. Telfer, M. Thibault, C.E. Tutin, L.J. White, and D.S. Wilkie. 2003. Catastrophic ape decline in western equatorial Africa. *Nature* 422: 611–614.
- Walters, M.J. 2003. *Six modern plagues and how we are causing them*. Washington, D.C.: Island Press.
- Waltner-Toews, D. Veterinary public health. 2002. In *Encyclopedia of public health*, ed. L. Breslow, n.p.

- New York: Macmillan Reference. idmed.slu.se/VPH/VPH-Waltner-Toews.pdf.
- Webster, R.G. 1998. Influenza: An emerging microbial pathogen. In *Emerging infections*, ed. R.M. Krause, J.I. Gallin, and A. Fauci, 275–300. San Diego, Calif.: Academic Press.
- Webster, R.G., W.J. Bean, O.T. Gorman, T.M. Chambers, and Y. Kawaoka. 1992. Evolution and ecology of influenza A viruses. *Microbiological Reviews* 56(1): 152–179.
- Weinhold B. 2004. Infectious disease: The human costs of our environmental errors. *Environmental Health Perspectives* 112(1): A32–39.
- William, J. 2003. The story of civet. *The Pharmaceutical Journal* 271(7280): 859–861.
- Williams, E.S., T. Yuill, M. Artois, J. Fischer, and S.A. Haigh. 2002. Emerging infectious diseases in wildlife. *Revue Scientifique et Technique Office International des Epizooties* 21: 139–157.
- Wong, K.T., W.J. Shieh, S.R. Zaki, and C.T. Tan. 2002. Nipah virus infection, an emerging paramyxoviral zoonosis. *Springer Seminars in Immunopathology* 24: 215–228.
- Wong, S.C., M.H. Ooi, M.N.L. Wong, P.H. Tio, T. Solomon, and M.J. Cardoso. 2001. Late presentation of Nipah virus encephalitis and kinetics of the humoral immune response. *Journal of Neurology, Neurosurgery, and Psychiatry* 71: 552–554.
- Woolhouse, M.E.J. 2002. Population biology of emerging and re-emerging pathogens. *Trends in Microbiology* 10: S3–7.
- World Health Organization. n.d. Frequently asked questions about the WHO global strategy on diet, physical activity, and health. <http://www.who.int/diet-physicalactivity/faq/en/>.
- . 2003. Diet, nutrition, and the prevention of chronic diseases. Geneva: World Health Organization. www.who.int/hpr/NPH/docs/who_fao_expert_report.pdf.
- . 2004. Avian influenza A(H5N1)—Update 28: Reports of infection in domestic cats (Thailand), situation (human) in Thailand, situation (poultry) in Japan and China. February 20. www.who.int/csr/don/2004_02_20/en/.
- . 2005. Streptococcus suis fact sheet. www.wpro.who.int/media_centre/fact_sheets/fs_20050802.htm.
- . 2006. Cumulative number of confirmed human cases of avian influenza A/(H5N1). July 4. www.wpro.who.int/NR/rdonlyres/F129EF8A-5DAB-4411-8E89-45E08B0657C0/0/AIWeekly53WPRO.pdf.
- World Health Organization and Office International des Epizooties. 1999. WHO Consultation on Public Health and Animal Transmissible Spongiform Encephalopathies: Epidemiology, risk, and research requirements. December 1–31.
- Worldwatch Institute. 2004. Meat: Now, it's not personal! But like it or not, meat-eating is becoming a problem for everyone on the planet. *World Watch*, July/August: 12–20.
- Yoo, D., P. Willson, Y. Pei, M.A. Hayes, A. Deckert, C.E. Dewey, R.M. Friendship, Y. Yoon, M. Gottschalk, C. Yason, and A. Giulivi. 2001. Prevalence of hepatitis E virus antibodies in Canadian swine herds and identification of a novel variant of swine hepatitis E virus. *Clinical and Diagnostic Laboratory Immunology* 8: 1213–1219.