

Letter from the Desk of David Challinor  
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In Washington, D.C., where I live, an abrupt and highly visible change in local bird composition occurred in 2002. During the previous five years, our crow population had exploded, and from my zoo office I enjoyed watching, through my binoculars, young crows fledging from a nest high in a white pine across Olmstead Walk. During my noontime perambulations around the Bird House, crows were always if not in sight, at least in sound. My golfing spouse reported them rampant at her course and so aggressive that they rummaged through unoccupied golf carts to loot sandwiches casually left exposed. The ubiquitous congeries of crows virtually vanished in 2002 from their deadly susceptibility to West Nile virus (WNV), a relatively recent introduction from the old world to the new. It is spread among birds in several ways, i.e. by contact, such as feeding or grooming; by being bitten by a mosquito carrying the virus; or by eating an infected mosquito. Not only have hundreds of thousands—perhaps millions—of birds succumbed to this virus, but so have creatures as disparate as elephants, humans and alligators. This month's letter discusses WNV and mad cow disease, two plagues that have jumped from animals to humans, and the role that humans continue to play in their dissemination.

The first human cases of WNV in the eastern U.S. were diagnosed in the summer of 1999 in Queens, Long Island. The summer had been dry, which contrary to popular theory created ideal breeding conditions for the urban house mosquito (*Culex pipiens*). After biting a bird, her preferred but not exclusive blood source, the female mosquito lays her eggs in nutrient-rich water. Sewer water in drains unflushed by rain is an ideal site for mosquito larvae. When they metamorphose to their winged stage, they remain in the cool sewers until the first rain washes them out to feast on their preferred hosts. The virus lives in the female mosquito but needs a warm-blooded host to replicate itself. Whenever the infected mosquito bites another bird, the virus gains a new host. Thus the virus spread rapidly from its initial outbreak in Queens in the summer of 1999 to Washington, D.C. and environs by 2002.

*C. pipiens* also bites people, and the first human victim of WNV died within two weeks of the August rain that flowed through the Queens storm sewers. The human deaths that followed were all described at that time as being caused by St. Louis encephalitis, a viral disease heretofore unknown in New York City. Earlier that summer thousands of crows were found dead, including at least 40 in and around New York's Bronx Zoo across the East River from Queens. The Zoo lost a bald eagle, a cormorant and three Chilean flamingoes to the West Nile virus.

Enter the Zoo's professional staff. Tracey McNamara, the Zoo's veterinarian, was skeptical that St. Louis encephalitis was causing such avian mortality because this virus strain usually strikes mammals, particularly horses, rather than birds. She thought

the virus might be eastern equine encephalitis, which attacks the brains of horses, people and birds. However, emus (an Australian ostrich-like ratite) are particularly susceptible to this virus, yet none of the Zoo's emus were ill. McNamara's assistant accidentally stuck herself with a needle while drawing blood from a dying bird, so the veterinarian logically contacted the Center for Disease Control (CDC) to test the blood samples from both her colleague and the dead bird. CDC refused, for bureaucratic reasons, to do the test, but another laboratory was willing and found an unusual virus in both samples. After numerous subsequent tests, the CDC finally agreed that it was indeed a virus new to the U.S. and after comparing it to other viruses of similar genetic strains, they found it matched one from the brain of a dead goose in Israel.

Although West Nile virus was first identified in the 1930's in an infected woman on the west bank of the Nile in Uganda (hence its name), the virus is widely spread by infected birds, most of which are resistant to it, along their migration routes from Africa to Europe. Still not understood is how this virus, heretofore confined to the old world, jumped the Atlantic and fatally infected a man in Queens. We may never learn the answer, but the odds are reasonable that it may have been carried by an escaped African bird imported through Kennedy airport. Once the virus arrived, however, it rapidly and fatally infected 7 of 59 people who were hospitalized with it.

By 2001, the virus was already in the bird populations of ten states from Massachusetts to Florida. What made it so hard to control was that more than forty species of mosquitoes can carry the virus and more than 200 bird species, as well as some mammals, are all WNV vectors. The highest mortality from this virus at the National Zoo was in 2002. Of the hundreds of dead crows found on the Zoo grounds, almost all tested positive for WNV; we also lost six flamingo chicks and about 20 other birds from our collection. A vigorous vaccination program, plus strict mosquito control, has helped curb the outbreak and with the gradual development of immunity in the wild (unvaccinated) bird population, there were no deaths at the Zoo from WNV in 2003. The virus, nonetheless, is now endemic in the U.S.; quarantine and immunization when practicable will be necessary to control the spread of the virus.

Not so successful have been the efforts to control another plague that has also jumped from beast to man. Mad cow disease, scientifically known as bovine spongiform encephalopathy (BSE), was first identified in the U.S. in a cow in Washington State. The name refers to the sponge-like appearance of an infected cow's brain. This disease is now closely linked to a human condition with similar symptoms called Creutzfeldt-Jakob disease (CJD), named after the two physicians who first identified it in 1920. The disease is hard to diagnose in humans for it has many of the same early symptoms as Alzheimers.

Creutzfeldt-Jakob disease (CJD), mad cow disease (BSE) and kuru, the fatal neurological disease of the Foré group in New Guinea, are now referred to as transmissible spongiform encephalopathies (TSE's). The first of this group to be

identified in animals was scrapie, a disease of sheep and goats, in 1732. Although long suspected of being infectious, it was not clinically shown to be so until 1936. It was confined to Europe until 1947, when it showed up in a mink farm in Wisconsin where the animals were fed with a mixture of sheep and goat entrails from evidently infected carcasses.

In 1984, BSE started infecting cows throughout parts of England and 10 years later (1993), about 1,000 new cases a week were recorded in the UK. In 1989, a 36-year-old woman was diagnosed with CJD; she had been associated with a farm on which BSE had been present. A government-sponsored scientific group discounted a connection between CJD and BSE. However, three years later, a dairy farmer whose herd had been infected with BSE also developed CJD; the nervous government still dismissed any linkage between the two diseases. By 1993, however, the connection was becoming more evident when not only beef and dairy farmers were coming down with CJD, but young beef-eating people who had never been near a farm with BSE-infected animals also were stricken with CJD. It was not until April 1996 that the British Secretary of State for Health publicly admitted that the ten beef-eating young people who died had all contracted what was to be called variant CJD, or mad cow disease in humans. Now, the search for the spread of the disease began to focus on the mechanism for infection.

In 1980, scrapie jumped from sheep to cow, and in Britain, where most of the research was done to identify this disease, scientists learned that the root cause was a nutrient supplement fed to cattle. It was made from bones and entrails of slaughtered livestock. Despite the intensive boiling and grinding used to prepare this nutrient supplement, the disease-causing pathogen survived; not until the 1980's was it identified and named. It turned out not to be a virus, although it behaves like one; furthermore, it had no DNA—and thus was not even alive. To “kill” it, the pathogen has to be soaked in a bucket of lye for an hour, or autoclaved at 134°C for about the same time. The infecting agent was named prion. A prion is a protein, one of our body's normal molecular constituents, but prions are connected to each other in a lethal way. Prions do not reproduce, but once present in a susceptible animal they force normal proteins into abnormal arrangements, often leading to the animal's death.

Evidently, mad cow disease started in England, caused by a series of unintentional human-initiated practices. First, the number of sheep raised in Britain increased during the eighties, from 31 to 44 million. That resulted in many more sheep, including scrapie-infected ones, being sent for slaughter and eventually to rendering plants. Their byproducts were fed to cows as a protein supplement. Cows, however, had not evolved to eat meat, even if reconstituted as a nutrient supplement. Scientists are not certain that this new diet was the precise cause of BSE in cows, but the evidence is strong. The use of animal protein in cow feed is now prohibited worldwide.

Variant Creutzfeldt-Jakob disease has a ten-year incubation period, so at my age it is not a high personal priority for concern, but my heart goes out to those innocent people who may already be infected and are unknowingly incubating this scourge. Control measures are being sought, but one can grasp the difficulty in trying to “kill” a prion when it is not alive. Human and veterinary medicine, I am confident, are up to the challenge. Just as they have curbed, but not obliterated, WNV, so, I believe, they can do the same for transmissible spongiform encephalopathies-scrapias, mad cow, kuru and Creutzfeldt-Jakob diseases.

Beef and dairy cattle, sadly, are not the only victims of prions, for in the last few decades deer and elk in the Rockies have been hit by Chronic Wasting Disease (CWD). It was first observed in Colorado mule deer in 1967, but not until a decade later was a prion determined to be the cause. Although there is yet no hard evidence that CWD can jump from deer to humans, it is a possibility. The disease has now spread in deer across Mississippi, and we can only speculate with trepidation what could happen should it reach the deer-crowded east coast.

Despite the medical fascination of these and other new (to us) and potentially threatening diseases, it is important to remember that the relatively trivial number of fatal cases scarcely justifies the publicity and research investment they have spawned. Do not forget that millions of children die every decade from diarrhea, malaria and influenza—all treatable under prosperous conditions. Balancing research and public health priorities is difficult and demands the wisdom and judgment of Solomon.

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P.S. Much of the information in this letter I gleaned from the following sources:

- 1) Walters, Mark J. 2003. *Six Modern Plagues and How We are Causing Them*. Shearwater Books, Washington
- 2) Mara, Peter P. *et al.* 2004. *West Nile Virus and Wildlife*. *Bioscience*. 54 (5):393-402
- 3) Brown, Paul. 2004. *Mad Cow Disease* lecture at Cosmos Club, Wash., DC 11 May