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Article

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The Arrow of Disease

When Columbus and his successors invaded the Americas, the most potent weapon they carried was their germs. But why didn't deadly disease flow in the other direction, from the New World to the Old?

JARED DIAMOND

Learning Outcomes

After reading this article, you will be able to:

- Discuss the biological and social circumstances of disease transmission from animals to humans.
- Explain the "one-sidedness" to the disease transmission from Europeans to Native Americans.

The three people talking in the hospital room were already stressed out from having to cope with a mysterious illness, and it didn't help at all that they were having trouble communicating. One of them was the patient, a small, timid man, sick with pneumonia caused by an unidentified microbe and with only a limited command of the English language. The second, acting as translator, was his wife, worried about her husband's condition and frightened by the hospital environment. The third person in the trio was an inexperienced young doctor, trying to figure out what might have brought on the strange illness. Under the stress, the doctor was forgetting everything he had been taught about patient confidentiality. He committed the awful blunder of requesting the woman to ask her husband whether he'd had any sexual experiences that might have caused the infection.

As the young doctor watched, the husband turned red, pulled himself together so that he seemed even smaller, tried to disappear under his bed sheets, and stammered in a barely audible voice. His wife suddenly screamed in rage and drew herself up to tower over him. Before the doctor could stop her, she grabbed a heavy metal bottle, slammed it onto her husband's head, and stormed out of the room. It took a while for the doctor to elicit, through the man's broken English, what he had said to so enrage his wife. The answer slowly emerged: he had admitted to repeated intercourse with sheep on a recent visit to the family farm; perhaps that was how he had contracted the mysterious microbe.

This episode, related to me by a physician friend involved in the case, sounds so bizarrely one of a kind as to be of no possible broader significance. But in fact it illustrates a subject of great importance: human diseases of animal origins. Very few of us may love sheep in the carnal sense. But most of us platonically love our pet animals, like our dogs and cats; and as a society, we certainly appear to have an inordinate fondness for sheep and other livestock, to judge from the vast numbers of them that we keep.

Some of us—most often our children—pick up infectious diseases from our pets. Usually these illnesses remain no more than a nuisance, but a few have evolved into far more. The major killers of humanity throughout our recent history—smallpox, flu, tuberculosis, malaria, plague, measles, and cholera—are all infectious diseases that arose from diseases of animals. Until World War II more victims of war died of microbes than of gunshot or sword wounds. All those military histories glorifying Alexander the Great and Napoleon ignore the ego-deflating truth: the winners of past wars were not necessarily those armies with the best generals and weapons, but those bearing the worst germs with which to smite their enemies.

The grimmest example of the role of germs in history is much on our minds this month, as we recall the European conquest of the Americas that began with Columbus's voyage of 1492. Numerous as the Indian victims of the murderous Spanish conquistadores were, they were dwarfed in number by the victims of murderous Spanish microbes. These formidable conquerors killed an estimated 95 percent of the New World's pre-Columbian Indian population.

Why was the exchange of nasty germs between the Americas and Europe so unequal? Why didn't the reverse happen instead, with Indian diseases decimating the Spanish invaders, spreading back across the Atlantic, and causing a 95 percent decline in *Europe's* human population?

Similar questions arise regarding the decimation of many other native peoples by European germs, and regarding the decimation of would-be European conquistadores in the tropics of Africa and Asia.

Naturally, we're disposed to think about diseases from our own point of view: What can we do to save ourselves and to kill the microbes? Let's stamp out the scoundrels, and never mind what *their* motives are!

In life, though, one has to understand the enemy to beat him. So for a moment, let's consider disease from the microbes' point of view. Let's look beyond our anger at their making us sick in bizarre ways, like giving us genital sores or diarrhea, and ask why it is that they do such things. After all, microbes are as much a product of natural selection as we are, and so their actions must have come about because they confer some evolutionary benefit.

Basically, of course, evolution selects those individuals that are most effective at producing babies and at helping those babies find suitable places to live. Microbes are marvels at this latter requirement. They have evolved diverse ways of spreading from one person to another, and from animals to people. Many of our symptoms of disease actually represent ways in which some clever bug modifies our bodies or our behavior such that we become enlisted to spread bugs.

The most effortless way a bug can spread is by just waiting to be transmitted passively to the next victim. That's the strategy practiced by microbes that wait for one host to be eaten by the next—salmonella bacteria, for example, which we contract by eating already-infected eggs or meat; or the worm responsible for trichinosis, which waits for us to kill a pig and eat it without properly cooking it.

As a slight modification of this strategy; some microbes don't wait for the old host to die but instead hitchhike in the saliva of an insect that bites the old host and then flies to a new one. The free ride may be provided by mosquitoes, fleas, lice, or tsetse flies, which spread malaria, plague, typhus, and sleeping sickness, respectively. The dirtiest of all passive-carriage tricks is perpetrated by microbes that pass from a woman to her fetus—microbes such as the ones responsible for syphilis, rubella (German measles), and AIDS. By their cunning these microbes can already be infecting an infant before the moment of its birth.

Other bugs take matters into their own hands, figuratively speaking. They actively modify the anatomy or habits of their host to accelerate their transmission. From our perspective, the open genital sores caused by venereal diseases such as syphilis are a vile indignity. From the microbes' point of view, however, they're just a useful device to enlist a host's help in inoculating the body cavity of another host with microbes. The skin lesions caused by smallpox similarly spread microbes by direct or indirect body contact (occasionally very indirect, as when U.S. and Australian whites bent on wiping out "belligerent" native peoples sent them gifts of blankets previously used by smallpox patients).

More vigorous yet is the strategy practiced by the influenza, common cold, and pertussis (whooping cough) microbes, which induce the victim to cough or sneeze, thereby broadcasting the bugs toward prospective new hosts. Similarly the cholera bacterium induces a massive diarrhea that spreads bacteria into the water supplies of potential new victims. For modification of a host's behavior, though, nothing matches the rabies

virus, which not only gets into the saliva of an infected dog but drives the dog into a frenzy of biting and thereby infects many new victims.

Thus, from our viewpoint, genital sores, diarrhea, and coughing are "symptoms" of disease. From a bug's viewpoint, they're clever evolutionary strategies to broadcast the bug. That's why it's in the bug's interests to make us "sick." But what does it gain by killing us? That seems self-defeating, since a microbe that kills its host kills itself.

Though you may well think it's of little consolation, our death is really just an unintended by-product of host symptoms that promote the efficient transmission of microbes. Yes, an untreated cholera patient may eventually die from producing diarrheal fluid at a rate of several gallons a day. While the patient lasts, though, the cholera bacterium profits from being massively disseminated into the water supplies of its next victims. As long as each victim thereby infects, on average, more than one new victim, the bacteria will spread, even though the first host happens to die.

So much for the dispassionate examination of the bug's interests. Now let's get back to considering our own selfish interests: to stay alive and healthy, best done by killing the damned bugs. One common response to infection is to develop a fever. Again, we consider fever a "symptom" of disease, as if it developed inevitably without serving any function. But regulation of body temperature is under our genetic control, and a fever doesn't just happen by accident. Because some microbes are more sensitive to heat than our own bodies are, by raising our body temperature we in effect try to bake the bugs to death before we get baked ourselves.

We and our pathogens are now locked in an escalating evolutionary contest, with the death of one contestant the price of defeat, and with natural selection playing the role of umpire.

Another common response is to mobilize our immune system. White blood cells and other cells actively seek out and kill foreign microbes. The specific antibodies we gradually build up against a particular microbe make us less likely to get reinfected once we are cured. As we all know there are some illnesses, such as flu and the common cold, to which our resistance is only temporary; we can eventually contract the illness again. Against other illnesses, though—including measles, mumps, rubella, pertussis, and the now-defeated menace of smallpox—antibodies stimulated by one infection confer lifelong immunity. That's the principle behind vaccination—to stimulate our antibody production without our having to go through the actual experience of the disease.

Alas, some clever bugs don't just cave in to our immune defenses. Some have learned to trick us by changing their antigens, those molecular pieces of the microbe that our

antibodies recognize. The constant evolution or recycling of new strains of flu, with differing antigens, explains why the flu you got two years ago didn't protect you against the different strain that arrived this year. Sleeping sickness is an even more slippery customer in its ability to change its antigens rapidly.

Among the slipperiest of all is the virus that causes AIDS, which evolves new antigens even as it sits within an individual patient, until it eventually overwhelms the immune system.

Our slowest defensive response is through natural selection, which changes the relative frequency with which a gene appears from generation to generation. For almost any disease some people prove to be genetically more resistant than others. In an epidemic, those people with genes for resistance to that particular microbe are more likely to survive than are people lacking such genes. As a result, over the course of history human populations repeatedly exposed to a particular pathogen tend to be made up of individuals with genes that resist the appropriate microbe just because unfortunate individuals without those genes were less likely to survive to pass their genes on to their children.

Fat consolation, you may be thinking. This evolutionary response is not one that does the genetically susceptible dying individual any good. It does mean, though, that a human population as a whole becomes better protected.

In short, many bugs have had to evolve tricks to let them spread among potential victims. We've evolved counter-tricks, to which the bugs have responded by evolving counter-counter-tricks. We and our pathogens are now locked in an escalating evolutionary contest, with the death of one contestant the price of defeat, and with natural selection playing the role of umpire.

The form that this deadly contest takes varies with the pathogens: for some it is like a guerrilla war, while for others it is a blitzkrieg. With certain diseases, like malaria or hookworm, there's a more or less steady trickle of new cases in an affected area, and they will appear in any month of any year. Epidemic diseases, though, are different: they produce no cases for a long time, then a whole wave of cases, then no more cases again for a while.

Among such epidemic diseases, influenza is the most familiar to Americans, this year having been a particularly bad one for us (but a great year for the influenza virus). Cholera epidemics come at longer intervals, the 1991 Peruvian epidemic being the first one to reach the New World during the twentieth century. Frightening as today's influenza and cholera epidemics are, though, they pale beside the far more terrifying epidemics of the past, before the rise of modern medicine. The greatest single epidemic in human history was the influenza wave that killed 21 million people at the end of the First World War. The black death, or bubonic plague, killed one-quarter of Europe's population between 1346 and 1352, with death tolls up to 70 percent in some cities.

The infectious diseases that visit us as epidemics share several characteristics. First, they spread quickly and efficiently

from an infected person to nearby healthy people, with the result that the whole population gets exposed within a short time. Second, they're "acute" illnesses: within a short time, you either die or recover completely. Third, the fortunate ones of us who do recover develop antibodies that leave us immune against a recurrence of the disease for a long time, possibly our entire lives. Finally, these diseases tend to be restricted to humans; the bugs causing them tend not to live in the soil or in other animals. All four of these characteristics apply to what Americans think of as the once more-familiar acute epidemic diseases of childhood, including measles, rubella, mumps, pertussis, and smallpox.

It is easy to understand why the combination of those four characteristics tends to make a disease run in epidemics. The rapid spread of microbes and the rapid course of symptoms mean that everybody in a local human population is soon infected, and thereafter either dead or else recovered and immune. No one is left alive who could still be infected. But since the microbe can't survive except in the bodies of living people, the disease dies out until a new crop of babies reaches the susceptible age—and until an infectious person arrives from the outside to start a new epidemic.

A classic illustration of the process is given by the history of measles on the isolated Faeroe Islands in the North Atlantic. A severe epidemic of the disease reached the Faeroes in 1781, then died out, leaving the islands measles-free until an infected carpenter arrived on a ship from Denmark in 1846. Within three months almost the whole Faeroes population—7,782 people—had gotten measles and then either died or recovered, leaving the measles virus to disappear once again until the next epidemic. Studies show that measles is likely to die out in any human population numbering less than half a million people. Only in larger populations can measles shift from one local area to another, thereby persisting until enough babies have been born in the originally infected area to permit the disease's return.

Rubella in Australia provides a similar example, on a much larger scale. As of 1917 Australia's population was still only 5 million, with most people living in scattered rural areas. The sea voyage to Britain took two months, and land transport within Australia itself was slow. In effect, Australia didn't even consist of a population of 5 million, but of hundreds of much smaller populations. As a result, rubella hit Australia only as occasional epidemics, when an infected person happened to arrive from overseas and stayed in a densely populated area. By 1938, though, the city of Sydney alone had a population of over one million, and people moved frequently and quickly by air between London, Sydney, and other Australian cities. Around then, rubella for the first time was able to establish itself permanently in Australia.

What's true for rubella in Australia is true for most familiar acute infectious diseases throughout the world. To sustain themselves, they need a human population that is sufficiently numerous and densely packed that a new crop of susceptible children is available for infection by the time the disease would otherwise be waning. Hence the measles and other such diseases are also known as "crowd diseases."

Crowd diseases could not sustain themselves in small bands of hunter-gatherers and slash-and-burn farmers. As tragic recent experience with Amazonian Indians and Pacific Islanders confirms, almost an entire tribelet may be wiped out by an epidemic brought by an outside visitor, because no one in the tribelet has any antibodies against the microbe. In addition, measles and some other "childhood" diseases are more likely to kill infected adults than children, and all adults in the tribelet are susceptible. Having killed most of the tribelet, the epidemic then disappears. The small population size explains why tribelets can't sustain epidemics introduced from the outside; at the same time it explains why they could never evolve epidemic diseases of their own to give back to the visitors.

That's not to say that small human populations are free from all infectious diseases. Some of their infections are caused by microbes capable of maintaining themselves in animals or in soil, so the disease remains constantly available to infect people. For example, the yellow fever virus is carried by African wild monkeys and is constantly available to infect rural human populations of Africa. It was also available to be carried to New World monkeys and people by the transAtlantic slave trade.

Other infections of small human populations are chronic diseases, such as leprosy and yaws, that may take a very long time to kill a victim. The victim thus remains alive as a reservoir of microbes to infect other members of the tribelet. Finally, small human populations are susceptible to nonfatal infections against which we don't develop immunity, with the result that the same person can become reinfected after recovering. That's the case with hookworm and many other parasites.

All these types of diseases, characteristic of small, isolated populations, must be the oldest diseases of humanity. They were the ones that we could evolve and sustain through the early millions of years of our evolutionary history, when the total human population was tiny and fragmented. They are also shared with, or are similar to the diseases of, our closest wild relatives, the African great apes. In contrast, the evolution of our crowd diseases could only have occurred with the buildup of large, dense human populations, first made possible by the rise of agriculture about 10,000 years ago, then by the rise of cities several thousand years ago. Indeed, the first attested dates for many familiar infectious diseases are surprisingly recent: around 1600 B.C. for smallpox (as deduced from pockmarks on an Egyptian mummy), 400 B.C. for mumps, 1840 for polio, and 1959 for AIDS.

Agriculture sustains much higher human population densities than do hunting and gathering—on average, 10 to 100 times higher. In addition, hunter-gatherers frequently shift camp, leaving behind their piles of feces with their accumulated microbes and worm larvae. But farmers are sedentary and live amid their own sewage, providing microbes with a quick path from one person's body into another person's drinking water. Farmers also become surrounded by disease-transmitting rodents attracted by stored food.

The explosive increase in world travel by Americans, and in immigration to the United States, is turning us into another melting pot—this time of microbes that we'd dismissed as causing disease in far-off countries.

Some human populations make it even easier for their own bacteria and worms to infect new victims, by intentionally gathering their feces and urine and spreading it as fertilizer on the fields where people work. Irrigation agriculture and fish farming provide ideal living conditions for the snails carrying schistosomes, and for other flukes that burrow through our skin as we wade through the feces-laden water.

If the rise of farming was a boon for our microbes, the rise of cities was a veritable bonanza, as still more densely packed human populations festered under even worse sanitation conditions. (Not until the beginning of the twentieth century did urban populations finally become self-sustaining; until then, constant immigration of healthy peasants from the countryside was necessary to make good the constant deaths of city dwellers from crowd diseases.) Another bonanza was the development of world trade routes, which by late Roman times effectively joined the populations of Europe, Asia, and North Africa into one giant breeding ground for microbes. That's when smallpox finally reached Rome as the "plague of Antonius," which killed millions of Roman citizens between A.D. 165 and 180.

Similarly, bubonic plague first appeared in Europe as the plague of Justinian (A.D. 542–543). But plague didn't begin to hit Europe with full force, as the black death epidemics, until 1346, when new overland trading with China provided rapid transit for flea-infested furs from plague-ridden areas of Central Asia. Today our jet planes have made even the longest intercontinental flights briefer than the duration of any human infectious disease. That's how an Aerolíneas Argentinas airplane, stopping in Lima, Peru, earlier this year, managed to deliver dozens of cholera-infected people the same day to my city of Los Angeles, over 3,000 miles away. The explosive increase in world travel by Americans, and in immigration to the United States, is turning us into another melting pot—this time of microbes that we previously dismissed as just causing exotic diseases in far-off countries.

When the human population became sufficiently large and concentrated, we reached the stage in our history when we could at last sustain crowd diseases confined to our species. But that presents a paradox: such diseases could never have existed before. Instead they had to evolve as new diseases. Where did those new diseases come from?

Evidence emerges from studies of the disease-causing microbes themselves. In many cases molecular biologists have identified the microbe's closest relative. Those relatives also

prove to be agents of infectious crowd diseases—but ones confined to various species of domestic animals and pets! Among animals too, epidemic diseases require dense populations, and they're mainly confined to social animals that provide the necessary large populations. Hence when we domesticated social animals such as cows and pigs, they were already afflicted by epidemic diseases just waiting to be transferred to us.

For example, the measles virus is most closely related to the virus causing rinderpest, a nasty epidemic disease of cattle and many wild cud-chewing mammals. Rinderpest doesn't affect humans. Measles, in turn, doesn't affect cattle. The close similarity of the measles and rinderpest viruses suggests that the rinderpest virus transferred from cattle to humans, then became the measles virus by changing its properties to adapt to us. That transfer isn't surprising, considering how closely many peasant farmers live and sleep next to cows and their accompanying feces, urine, breath, sores, and blood. Our intimacy with cattle has been going on for 8,000 years since we domesticated them—ample time for the rinderpest virus to discover us nearby. Other familiar infectious diseases can similarly be traced back to diseases of our animal friends.

Given our proximity to the animals we love, we must constantly be getting bombarded by animal microbes. Those invaders get winnowed by natural selection, and only a few succeed in establishing themselves as human diseases. A quick survey of current diseases lets us trace four stages in the evolution of a specialized human disease from an animal precursor.

In the first stage, we pick up animal-borne microbes that are still at an early stage in their evolution into specialized human pathogens. They don't get transmitted directly from one person to another, and even their transfer from animals to us remains uncommon. There are dozens of diseases like this that we get directly from pets and domestic animals. They include cat scratch fever from cats, leptospirosis from dogs, psittacosis from chickens and parrots, and brucellosis from cattle. We're similarly susceptible to picking up diseases from wild animals, such as the tularemia that hunters occasionally get from skinning wild rabbits.

In the second stage, a former animal pathogen evolves to the point where it does get transmitted directly between people and causes epidemics. However, the epidemic dies out for several reasons—being cured by modern medicine, stopping when everybody has been infected and died, or stopping when everybody has been infected and become immune. For example, a previously unknown disease termed *o'nyong-nyong* fever appeared in East Africa in 1959 and infected several million Africans. It probably arose from a virus of monkeys and was transmitted to humans by mosquitoes. The fact that patients recovered quickly and became immune to further attack helped cause the new disease to die out quickly.

The annals of medicine are full of diseases that sound like no known disease today but that once caused terrifying epidemics before disappearing as mysteriously as they had come. Who alive today remembers the "English sweating sickness" that swept and terrified Europe between 1485 and 1578, or the "Picardy sweats" of eighteenth- and nineteenth-century France?

A third stage in the evolution of our major diseases is represented by former animal pathogens that establish themselves in humans and that do not die out; until they do, the question of whether they will become major killers of humanity remains up for grabs. The future is still very uncertain for Lassa fever, first observed in 1969 in Nigeria and caused by a virus probably derived from rodents. Better established is Lyme disease, caused by a spirochete that we get from the bite of a tick. Although the first known human cases in the United States appeared only as recently as 1962, Lyme disease is already reaching epidemic proportions in the Northeast, on the West Coast, and in the upper Midwest. The future of AIDS, derived from monkey viruses, is even more secure, from the virus's perspective.

The final stage of this evolution is represented by the major, long-established epidemic diseases confined to humans. These diseases must have been the evolutionary survivors of far more pathogens that tried to make the jump to us from animals—and mostly failed.

Diseases represent evolution in progress, as microbes adapt by natural selection to new hosts. Compared with cows' bodies, though, our bodies offer different immune defenses and different chemistry. In that new environment, a microbe must evolve new ways to live and propagate itself.

The best-studied example of microbes evolving these new ways involves myxomatosis, which hit Australian rabbits in 1950. The myxoma virus, native to a wild species of Brazilian rabbit, was known to cause a lethal epidemic in European domestic rabbits, which are a different species. The virus was intentionally introduced to Australia in the hopes of ridding the continent of its plague of European rabbits, foolishly introduced in the nineteenth century. In the first year, myxoma produced a gratifying (to Australian farmers) 99.8 percent mortality in infected rabbits. Fortunately for the rabbits and unfortunately for the farmers, the death rate then dropped in the second year to 90 percent and eventually to 25 percent, frustrating hopes of eradicating rabbits completely from Australia. The problem was that the myxoma virus evolved to serve its own interest, which differed from the farmers' interests and those of the rabbits. The virus changed to kill fewer rabbits and to permit lethally infected ones to live longer before dying. The result was bad for Australian farmers but good for the virus: a less lethal myxoma virus spreads baby viruses to more rabbits than did the original, highly virulent myxoma.

For a similar example in humans, consider the surprising evolution of syphilis. Today we associate syphilis with genital sores and a very slowly developing disease, leading to the death of untreated victims only after many years. However, when syphilis was first definitely recorded in Europe in 1495, its pustules often covered the body from the head to the knees, caused flesh to fall off people's faces, and led to death within a few months. By 1546 syphilis had evolved into the disease with the symptoms known to us today. Apparently, just as with myxomatosis, those syphilis spirochetes evolved to keep their victims alive longer in order to transmit their spirochete offspring into more victims.

How, then, does all this explain the outcome of 1492—that Europeans conquered and depopulated the New World, instead of Native Americans conquering and depopulating Europe?

In the century or two following Columbus's arrival in the New World, the Indian population declined by about 95 percent. The main killers were European germs, to which the Indians had never been exposed.

Part of the answer, of course, goes back to the invaders' technological advantages. European guns and steel swords were more effective weapons than Native American stone axes and wooden clubs. Only Europeans had ships capable of crossing the ocean and horses that could provide a decisive advantage in battle. But that's not the whole answer. Far more Native Americans died in bed than on the battlefield—the victims of germs, not of guns and swords. Those germs undermined Indian resistance by killing most Indians and their leaders and by demoralizing the survivors.

The role of disease in the Spanish conquests of the Aztec and Inca empires is especially well documented. In 1519 Cortés landed on the coast of Mexico with 600 Spaniards to conquer the fiercely militaristic Aztec Empire, which at the time had a population of many millions. That Cortés reached the Aztec capital of Tenochtitlán, escaped with the loss of "only" two-thirds of his force, and managed to fight his way back to the coast demonstrates both Spanish military advantages and the initial naïveté of the Aztecs. But when Cortés's next onslaught came, in 1521, the Aztecs were no longer naïve; they fought street by street with the utmost tenacity.

What gave the Spaniards a decisive advantage this time was smallpox, which reached Mexico in 1520 with the arrival of one infected slave from Spanish Cuba. The resulting epidemic proceeded to kill nearly half the Aztecs. The survivors were demoralized by the mysterious illness that killed Indians and spared Spaniards, as if advertising the Spaniards' invincibility. By 1618 Mexico's initial population of 20 million had plummeted to about 1.6 million.

Pizarro had similarly grim luck when he landed on the coast of Peru in 1531 with about 200 men to conquer the Inca Empire. Fortunately for Pizarro, and unfortunately for the Incas, smallpox had arrived overland around 1524, killing much of the Inca population, including both Emperor Huayna Capac and his son and designated successor, Ninan Cuyoche. Because of the vacant throne, two other sons of Huayna Capac, Atahualpa and Huáscar, became embroiled in a civil war that Pizarro exploited to conquer the divided Incas.

When we in the United States think of the most populous New World societies existing in 1492, only the Aztecs and Incas come to mind. We forget that North America also supported populous Indian societies in the Mississippi Valley. Sadly,

these societies too would disappear. But in this case conquistadores contributed nothing directly to the societies' destruction; the conquistadores' germs, spreading in advance, did everything. When De Soto marched through the Southeast in 1540, he came across Indian towns abandoned two years previously because nearly all the inhabitants had died in epidemics. However, he was still able to see some of the densely populated towns lining the lower Mississippi. By a century and a half later, though, when French settlers returned to the lower Mississippi, almost all those towns had vanished. Their relics are the great mound sites of the Mississippi Valley. Only recently have we come to realize that the mound-building societies were still largely intact when Columbus arrived, and that they collapsed between 1492 and the systematic European exploration of the Mississippi.

When I was a child in school, we were taught that North America had originally been occupied by about one million Indians. That low number helped justify the white conquest of what could then be viewed as an almost empty continent. However, archeological excavations and descriptions left by the first European explorers on our coasts now suggest an initial number of around 20 million. In the century or two following Columbus's arrival in the New World, the Indian population is estimated to have declined by about 95 percent.

The main killers were European germs, to which the Indians had never been exposed and against which they therefore had neither immunologic nor genetic resistance. Smallpox, measles, influenza, and typhus competed for top rank among the killers. As if those were not enough, pertussis, plague, tuberculosis, diphtheria, mumps, malaria, and yellow fever came close behind. In countless cases Europeans were actually there to witness the decimation that occurred when the germs arrived. For example, in 1837 the Mandan Indian tribe, with one of the most elaborate cultures in the Great Plains, contracted smallpox thanks to a steamboat traveling up the Missouri River from St. Louis. The population of one Mandan village crashed from 2,000 to less than 40 within a few weeks.

The one-sided exchange of lethal germs between the Old and New Worlds is among the most striking and consequence-laden facts of recent history. Whereas over a dozen major infectious diseases of Old World origins became established in the New World, not a single major killer reached Europe from the Americas. The sole possible exception is syphilis, whose area of origin still remains controversial.

That one-sidedness is more striking with the knowledge that large, dense human populations are a prerequisite for the evolution of crowd diseases. If recent reappraisals of the pre-Columbian New World population are correct, that population was not far below the contemporaneous population of Eurasia. Some New World cities, like Tenochtitlán, were among the world's most populous cities at the time. Yet Tenochtitlán didn't have awful germs waiting in store for the Spaniards. Why not?

One possible factor is the rise of dense human populations began somewhat later in the New World than in the Old. Another is that the three most populous American centers—the Andes, Mexico, and the Mississippi Valley—were never connected by regular fast trade into one gigantic breeding ground for microbes, in the way that Europe, North Africa, India, and China became connected in late Roman times.

The main reason becomes clear, however, if we ask a simple question: From what microbes could any crowd diseases of the Americas have evolved? We've seen that Eurasian crowd diseases evolved from diseases of domesticated herd animals. Significantly, there were many such animals in Eurasia. But there were only five animals that became domesticated in the Americas: the turkey in Mexico and parts of North America, the guinea pig and llama/alpaca (probably derived from the same original wild species) in the Andes, and Muscovy duck in tropical South America, and the dog throughout the Americas.

That extreme paucity of New World domestic animals reflects the paucity of wild starting material. About 80 percent of the big wild mammals of the Americas became extinct at the end of the last ice age, around 11,000 years ago, at approximately the same time that the first well-attested wave of Indian hunters spread over the Americas. Among the species that disappeared were ones that would have yielded useful domesticates, such as American horses and camels. Debate still rages as to whether those extinctions were due to climate changes or to the impact of Indian hunters on prey that had never seen humans. Whatever the reason, the extinctions removed most of the basis for Native American animal domestication—and for crowd diseases.

The few domesticates that remained were not likely sources of such diseases. Muscovy ducks and turkeys don't live in enormous flocks, and they're not naturally endearing species (like young lambs) with which we have much physical contact. Guinea pigs may have contributed a trypanosome infection like Chagas' disease or leishmaniasis to our catalog of woes, but that's uncertain.

Initially the most surprising absence is of any human disease derived from llamas (or alpacas), which are tempting to consider as the Andean equivalent of Eurasian livestock. However, llamas had three strikes against them as a source of human pathogens: their wild relatives don't occur in big herds as do wild sheep, goats, and pigs; their total numbers were never remotely as large as the Eurasian populations of domestic livestock, since llamas never spread beyond the Andes; and llamas aren't as cuddly as piglets and lambs and aren't kept in such close association with people. (You may not think of piglets as cuddly, but human mothers in the New Guinea highlands often nurse them, and they frequently live right in the huts of peasant farmers.)

The importance of animal-derived diseases for human history extends far beyond the Americas. Eurasian germs played a key role in decimating native peoples in many other parts of the world as well, including the Pacific islands, Australia, and southern Africa. Racist Europeans used to attribute those conquests to their supposedly better brains. But no evidence for such better brains has been forthcoming. Instead, the conquests were made possible by Europeans' nastier germs, and by the

technological advances and denser populations that Europeans ultimately acquired by means of their domesticated plants and animals.

So on this 500th anniversary of Columbus's discovery, let's try to regain our sense of perspective about his hotly debated achievements. There's no doubt that Columbus was a great visionary, seaman, and leader. There's also no doubt that he and his successors often behaved as bestial murderers. But those facts alone don't fully explain why it took so few European immigrants to initially conquer and ultimately supplant so much of the native population of the Americas. Without the germs Europeans brought with them—germs that were derived from their animals—such conquests might have been impossible.

Critical Thinking

1. What are the major killers of humanity and where do they come from? How did most victims of war die before World War II? What determined the winners of past wars? What percentage of the pre-Columbian Indian population was killed by Spanish microbes?
2. How might a bug such as salmonella be passively transmitted to its next victim? What examples does the author provide of microbes getting a "free ride" to its next victim? How might they modify the anatomy or habits of their host in order to accelerate their transmission?
3. How does the author, therefore, describe such "symptoms" of disease? Why is it in the bug's interest to make us "sick"? Is it in the bug's interest to kill us? How does the author explain death? How do bacteria survive even though the hosts die?
4. What "common responses" do we use to stay alive?
5. Explain the principle of vaccination. How do the bugs "trick us"? Why is the AIDS virus one of the "slipperiest of all"?
6. What is our slowest defense mechanism? How does the author describe the "escalating evolutionary context"?
7. Explain the difference between pathogens that fight a "guerrilla war," creating a steady trickle of new cases, versus those that conduct a "blitzkrieg" and cause epidemics.
8. What characteristics are shared by infectious diseases that visit us as epidemics?
9. Why does measles die out in populations of less than half a million people but persists in larger population?
10. Why can't such "crowd diseases" sustain themselves in small bands of hunter-gatherers and slash-and-burn farmers?
11. Does this mean that small human populations are free from all infectious diseases? Explain. Why does the author believe these to be the oldest diseases of humanity?
12. When did crowd diseases first develop and what are the various reasons for their increased frequency?
13. What is the paradox regarding these diseases? Where did they come from? Why does the author say we must be constantly bombarded by them?
14. Describe the four stages in the evolution of a specialized human disease from an animal precursor.

15. In what sense do diseases represent evolution in progress? How do the mixoma virus and the Australian rabbits provide an example?
16. How were Europeans technologically superior to Native Americans? What gave Cortez the decisive advantage over the Aztecs? Why were the surviving Aztecs demoralized? What was Pizarro's similar "grim luck"? What happened to the Mound Builders of the Mississippi Valley?
17. What was the author taught in school that originally justified conquest? What do we know now?
18. Why was there "one-sidedness" to the disease transmission in spite of the large Native American populations? Explain the author's reasoning.

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