



Luke J. Janssen

Pandemics in Need of a Christian Response

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Humanity has survived many waves of attacks mounted by microscopic agents which we cannot see but which have left millions of our species dead, and hundreds of millions more enduring a great deal of pain and suffering, even life-long dysfunction. Pandemics have led to the downfall of whole populations and people groups; they have shaped policies and practices of the societies which survived. These encounters have taught us a great deal about Earth's biology, as well as our own physiology, resourcefulness, and potential. They have shone a spotlight on the essence of humanity: the good, the bad, and the ugly. We have now encountered another pandemic-producing agent – COVID-19 – which has disrupted human activities around the globe. All of this raises many important and even existential questions for humanity in general, and Christians in particular.

The Ongoing Global War between Humans and “Bugs”

In the first chapter of the book of Genesis, God, having just created humanity, gives us the mandate to rule over creation: “Be fruitful and multiply, and fill the earth and subdue it; and have dominion over the fish of the sea and over the birds of the air and over every living thing that moves upon the earth” (Gen. 1:28, RSV). A few verses later in this passage, we are shown God evaluating his creation: “God saw everything that he had made, and indeed, it was very good” (Gen. 1:31).

Today, we find humanity fleeing in fear and mass retreat from an infectious agent which one cannot even see without an electron microscope. Borders have been locked down. Schools and businesses have been closed, and economic activity has been reduced to Depression-era levels. Governments are struggling to manage their populations, keep the peace, and distribute limited resources. Budgets at all levels have been stretched out of all proportion. Groups of people debate, disagree, and divide (sometimes violently) over how to respond to this existential threat.

The medical and scientific community has refocused nearly all of its attention upon this infectious agent, in the hopes of learning all we can about its structure, “life-cycle,” mechanism of action, pathological consequences, and vulnerabilities. How can we control this threat? How do we help people who have become infected by it? What policies and health practices do we need to develop?

Theologians and believers are asking a whole other set of questions. Was the small piece of RNA now circulating throughout the planet, and which has drawn the attention of all humanity, part of God's original good creation, and how can we gain or regain “dominion” over it and others like it? Is it within God's perfect will for humans to endure so many deadly pandemics? Or are we the victims of our own doing? Is COVID-19 a divine agent sent as a corrective, along the same line as the personal and national

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tragedies described in the Old Testament as punishments for sinning against YHWH?¹ Or was it sent to provide a new perspective such as the personal tragedies apparently orchestrated to teach humanity an important theological lesson?² Does the divine mandate given in the first chapter of Genesis not apply to viruses because they are technically not “living things that move upon the earth”? What can we learn about ourselves, about the human condition, and about human nature? Most importantly, how should we respond in a Christ-like manner?

The ASA and its members strategically include both science and theological perspectives: what can we offer to this discussion?

Key Characteristics of Infectious Disease

Many diseases are caused by viruses, bacteria, and fungi invading the human body, usurping certain of its physiological functions, and otherwise directly damaging host cells and tissues. To better understand and control these diseases, it is useful to define certain characteristics of these infections.

Infectivity

Most of those infectious agents are easily and quickly identified and neutralized by a healthy host immune system. In those cases, only immunocompromised individuals are susceptible, and even close contact on the part of other healthy individuals usually does not lead to their getting infected. For example, a fungal pneumonia or rabies is generally not easily contagious: an infected individual can come into close contact with many healthy individuals with little or no likelihood of transmitting the pathogen to them. However, some microbes and viruses are more easily transmitted, such that one infected individual can on average infect another individual, leading to an essentially perpetual low-level persistence of the pathogen in the human population. When infectivity (closely related words are transmissibility and virulence) is so high that each infected individual transmits to several other individuals, there will be an explosive growth in the number of infected individuals, a situation often referred to as an epidemic. When this occurs over a large area (e.g., global) and involves many people, it is referred to as a pandemic. A great deal of attention has been paid within the current COVID-19 pandemic upon a measurement tool for transmissibility referred to as R. This quan-

tity is determined by four factors: (1) the duration of infection (how long infected individuals are shedding viral particles); (2) the opportunity of infection (related to social distancing, quarantining); (3) the transmission probability (separation behind glass, plexiglass, or plastic barriers; wearing protective personal equipment); and (4) the susceptibility of the uninfected population (including different subgroups among them).

Pathogenicity

In addition to the infectivity of a microbe or virus, another important characteristic is its pathogenicity (virulence is a closely related word): the negative effects that it exerts on infected individuals. Some infections can be nearly unnoticeable: innumerable species of bacteria colonize the outside of our bodies and digestive tract, and our genomes are riddled with the residue of all kinds of viral infections (some of which can be “re-awakened” when the host is stressed, as is the case in herpes simplex³). Other infections are quite noticeable but easily tolerated because the symptoms are relatively minor and/or quickly resolve (common cold, viral conjunctivitis). The most concerning kinds of infections, though, are those which can produce substantial morbidity and even lead to death (AIDS, Ebola).

Transmission and the Key Roles of Other Species

Another important consideration pertains to the details around the involvement of other nonhuman species. There are two very different ways in which this aspect becomes important: (1) the mechanism by which that pathogen is transmitted to humans; and (2) the pathogenicity of that pathogen to humans.

First, we will consider the mechanism of transmission. The life cycles of many pathogens that plague humanity require the participation of one or more other intermediate species. On the one hand, that intermediate can serve as a reservoir of the pathogen: the latter infects the nonhuman population without producing major symptoms, allowing the pathogen to propagate and survive until it finds opportunity to infect human hosts. On the other hand, other intermediate species can play an active role in the pathogen’s delivery mechanism into humans. Many infected intermediates introduce the pathogen into the human bloodstream through a bite. For example, malaria is caused by the parasite *Plasmodium* spread by infected mosquitos;⁴ Lyme disease, by the intro-

duction of the bacterium *Borrelia* through the bite of various species of ticks (*Ixodes*);⁵ and rabies, by lyssaviruses passed on through the bite of an infected animal (dogs, cats, skunks).⁶ Other infected organisms simply bring the pathogen into close contact with humans: hantavirus is a life-threatening disease acquired when we are exposed to or inhale the urine, droppings, or saliva of infected white-footed deer mice occupying our dwelling spaces.

The second species-related consideration referred to above pertains to evolutionary origins. Infectious pathogens have long been coevolving in tandem with their hosts. The process of evolution always “seeks” to optimize the characteristics of each organism to maximize its survival and reproduction. In the specific case of infectious pathogens, it is generally not optimal for that agent to become increasingly and highly lethal; otherwise, it quickly destroys the host population upon which it depends for survival and, as a result, it then disappears from the ecosystem. Instead, it is optimal for it to lessen its pathogenicity while increasing its transmissibility, so that there will always be a fresh supply of new hosts. The hosts will develop some level of immunity and other protective mechanisms to which the infectious agent then re-adjusts (through successive generations over evolutionary time-frames), and an equilibrium is reached. A familiar example of this would be the common cold. We all get these every year, and put up with them for our entire lifetime because they do not greatly affect our well-being, let alone our survival.

We have evidence that birds, mammals, and coronaviruses (the family of viruses which give humans the common cold, as well as COVID-19) have been coevolving for 55 million years.⁷ But when an infectious agent suddenly finds itself able to infect an entirely new host species, the latter may not have the same defense mechanisms in place to accommodate that infection, or may have a different set of vulnerabilities to the pathogen (for example, humans require a much longer time for maturation to full reproductive potential than birds, bats, pigs, or primates). In this case, the pathogen that coevolved with those nonhuman species, but suddenly develops the ability to infect humans, can be lethal to humans. An example of this would be HIV-AIDS, which is believed to have originally been a simian immunodeficiency virus (SIV) which “jumped” into humans (see below).

Knowledge of the means by which agents infect us can explain many of the features of the diseases they produce, but it can also offer strategic opportunities for controlling that disease. These infectious agents first encounter the protective epithelial barrier of our skin through direct environmental contact, of our lungs following inhalation of aspirated particles, or of our digestive tract through ingestion, and then employ one or more mechanisms to breach that epithelial barrier. That breaching may involve the bite of an intermediate organism (as mentioned above), or can involve the pathogen itself physically penetrating the overlying mucous layer (cholera) and/or the epithelial layer (Trichinosis roundworms) and then innate immune cells (*Yersinia pestis*, or plague). In yet other cases, the pathogen employs a nondestructive molecular mechanism which is intrinsic to the host cells: distinctive markers on the surface of the pathogen bind to specific surface markers on the host cell and trigger a carefully orchestrated internalization process referred to as endocytosis (Ebola, influenza, coronavirus, AIDS). Once inside the cell, the internalized pathogen is then unpackaged and initiates a cascade of normal physiological molecular events which result in infection (usurping of the synthetic machinery of the host cell, insertion into the host genome).⁸

Global Pandemics throughout Human History

The greatest concern for humanity comes from pathogens which are highly infectious (transmissible) and also highly virulent (pathogenic), including grave morbidity and high mortality. Human history is checkered with many such pandemics.

Smallpox

Rashes observed on the skin of three Egyptian mummies suggest that smallpox may have afflicted humanity at least as far back as the third century BCE.⁹ The Antonine Plague (AD 165–180), brought to Rome by soldiers returning home from a campaign against Parthia, appears to have been smallpox: it is said to have killed over 5 million people and contributed to the collapse of the Roman Empire. There are also written descriptions of a disease which appears to have been smallpox from fourth-century China, seventh-century India, and tenth-century Asia Minor. European explorers introduced a variety of novel diseases, including smallpox, to the immunologically naïve indigenous populations of

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the Western Hemisphere, resulting in the American Plagues (sixteenth century) that killed 90% of the latter and contributed to the collapse of the Mayan, Aztec, and Incan Empires. It has been estimated that smallpox has killed 300 million people during the twentieth century.¹⁰

Smallpox is caused by one of two variants of variola virus of the genus *Orthopoxvirus*. Its genome is a single length of double-stranded DNA which codes for 118 early, 53 intermediate, and 38 late genes enveloped in a host-derived lipid shell studied with at least 20 proteins which recognize and bind to host markers and, in that way, gain entry.¹¹ Once inside, the virus disrupts the innate immune system by interfering with interferon signaling.¹² The virion is spread through contact with bodily fluids (pus exuded from the skin sores), aspirated droplets (cough, sneeze), or even sloughed skin (on bedding or clothing) of infected individuals. Human-to-human contact appears to be the only means of transmission: there is no evidence that it is spread by insects or animals. Initial symptoms include those that are common to many viral illnesses: fever, muscle pain, fatigue, headache, nausea, and vomiting. Skin lesions can manifest during the next two weeks; these exude a pus and produce scabs, both of which help spread the disease.

In the fifteenth century, the Chinese recognized that deliberate introduction of pus and/or scab from smallpox sores to unaffected individuals could confer a low level of illness and then immunity against the disease (in the unlucky few, the exposure was lethal). In the late eighteenth century, European doctors began to develop this further into what is now known as vaccination strategy, which eventually set the stage for globally directed smallpox eradication campaigns by the World Health Organization starting in 1959. On May 9, 1980, the 33rd World Health Assembly officially declared the world free of this disease.¹³

Plague

The Plague has frequently ravaged humanity.¹⁴ One outbreak claimed up to 10% of the world's population when it struck the Byzantine Empire in the middle of the sixth century. In the middle of the fourteenth century, the Black Death (1346–1353) emerged from Asia and ultimately wiped out over half of Europe's population, and arguably changed the course of Western civilization. The Great Plague

of London (1665–1666), the Great Plague of Marseille (1720–1723), and the Russian Plague (1770–1772) each claimed approximately 100,000 lives.

Plague is mediated by various strains of *Yersinia pestis* bacteria carried by fleas on infected rodents which serve as a reservoir. The bacteria are transmitted through the bite of those fleas, by coming into contact with the tissues or body fluids of an infected animal: for example, hunters skinning infected rabbits or other prey, or a domestic cat which has eaten an infected rodent, or breathing aspirated droplets from another infected human. The Marseilles pandemic seems to have been introduced by a single cargo ship arriving from the Mediterranean, while the Russian pandemic was introduced by prisoners of war and booty brought back by troops returning from war in what is now Romania.

Plague presents clinically with a familiar spectrum of symptoms: fever, chills, headache, and weakness accompanied by either painful swollen lymph nodes near the bite which infected the person (bubonic plague), or internal bleeding and blackened skin (septicemic plague), or by rapidly developing pneumonia (pneumonic plague). Antibiotics are the best treatment, and the degree and time-course of recovery are determined by how quickly those antibiotics are given.

In order to gain a strong foothold within the host before an effective immune response is mounted, this bacterium employs a syringe-like projection (the type-III pathway) to pierce the cell membrane and inject various toxins into the host cell, thereby killing it.¹⁵ The bacteria primarily attack cells of the innate immune system: the macrophages, neutrophils, and dendritic cells. The latter are important for instructing the T- and B-cells of the acquired immune system to identify and neutralize the invading organism, a process which generally takes 8–10 days, but the host is generally overwhelmed and dies before that can happen.

Ebola

The Plague of Athens (430 BC) lasted five years and claimed 100,000 lives with reported symptoms that suggest it might have been Ebola (or typhoid fever);¹⁶ overcrowding caused by the war with Sparta appears to have been a major contributing factor. The first outbreak of Ebola virus disease (EVD) to come to the attention of the modern global medical community occurred in 1976 in two different parts

of Africa (South Sudan and Democratic Republic of Congo). Since then, there have been twenty-six outbreaks of EVD throughout tropical regions of Sub-Saharan Africa. Signs and symptoms can begin as early as 2 days and as long as 3 weeks after exposure, and manifest first with flu-like symptoms (fever, headache, sore throat, joint pain, nausea, vomiting, diarrhea, abdominal pain), which can produce severe dehydration and shortness of breath. However, this initial phase is followed one week later by hematological disruption: all infected patients show decreased blood clotting, and roughly half experience internal and external bleeding (in saliva or stools, into the skin [bruising], in the eyes). Recovery may begin 2 or 3 weeks later; death, if it occurs, is primarily due to low blood pressure from fluid loss. EVD mortality in the many outbreaks that have occurred ranges from 25–90%. At this time, there have been almost 30,000 infections and over 11,000 confirmed deaths due to Ebola.

EVD is caused by a negative sense, single-stranded RNA virus within the genus *Ebolavirus*, of which there are five types.¹⁷ It is surprising, given the massive destruction that EVD causes, that the viral genome contains only seven genes (including, of course, RNA polymerase).¹⁸ Fruit bats appear to be a natural reservoir of Ebola virus. The latter is then transmitted to humans either directly or through other animals (pigs, dogs, primates), often by eating improperly cooked meat. Human-to-human transmission occurs easily via contact with many different bodily fluids (blood, saliva, vomit, tears, urine, semen, breast milk), but apparently not through aerosolized particles (produced by coughing or sneezing), sweat, or mosquito bites. It can infect almost all human cells using different surface markers for each cell type, and enters through several cellular uptake mechanisms, including the endocytosis pathway described above.¹⁹ However, it usually first infects immune cells (compromising immune function) and then endothelial cells (leading to fluid loss and bleeding).

Influenza

There are four main types of influenza virus (A, B, C, and D).²⁰ Types A and B are responsible for the annual seasonal flu epidemic. There are many different subtypes of influenza A (belonging to the family *Orthomyxoviridae*), distinguished in part on the basis of the subtypes of two different proteins which make up the protective external shell of the

virus: hemagglutinin or “H” (of which there are 18 different subtypes) and neuraminidase or “N” (11 different subtypes). There are 198 different possible combinations of these 18 hemagglutinin and 11 neuraminidase proteins—although only 131 of these have been detected in the wild—but then there can also be differences within the genetic package of the influenza virus. This variety explains, in part, why the annual flu shot can be a hit-or-miss affair. Several different domesticated farm animals (poultry, pigs) and wild waterfowl can serve as reservoirs for the influenza virus, and through them be transmitted to humans.

The influenza A and B viruses comprise a genetic package (eight negative-sense strands of RNA which encode ten essential viral proteins and several other accessory proteins) contained within a host-derived lipid envelope, decorated by a variety of proteins, including H and N referred to above. When the virion particles encounter the host respiratory epithelium, the proteins on the viral envelope interact with markers on the host cell membranes (sialic acid sugars).²¹ Cleavage of H by a cellular protease then triggers endocytosis, which introduces the genetic package into the host cells. Viral shedding is essentially the reverse of this process.

One influenza outbreak in 1889–1890 appeared first in Russia but spanned the entire globe within a few months (despite air travel being nonexistent at that time), leaving 1 million people dead in its wake. The “Spanish Flu” (1918–1920),²² an H1N1 avian virus, infected half a billion people around the world and killed a fifth of those (wiping out numerous indigenous people groups); its propagation was likely facilitated by the cramped and dirty conditions that soldiers experienced in the Great War (later renamed World War I) and by poor global nutrition. The 1968 Pandemic (1967) was brought on by an H3N2 avian flu that originated in the United States, but ultimately killed over a million people worldwide. The Swine Flu pandemic (2009–2010) originated in Mexico, infecting almost 1.5 billion people around the globe and killing 150–575 thousand of them. It was caused by a novel form of H1N1 avian virus. The US Centers for Disease Control and Prevention (CDC) has estimated that the annual burden of influenza during the decade spanning 2010 to 2019 has been 8–45 million infections, 140–810 thousand hospitalizations, and 12–61 thousand deaths (again, these are average *annual* numbers over that decade period).²³

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Human Immunodeficiency Virus (HIV)

HIV comprises two retroviral species of the genus *Lentivirus*.²⁴ It is transmitted through certain bodily fluids (blood, semen, rectal fluids, vaginal fluids, and breast milk), or through direct injection into the bloodstream using a contaminated syringe needle. HIV is believed to be a mutated version of simian immunodeficiency virus (SIV), probably transmitted to humans through hunting and eating of an infected chimpanzee, possibly as far back as the late 1800s. It comprises a strand of positive-sense RNA which codes for nine genes, including reverse transcriptase, enveloped in a host-derived lipid shell studded with proteins which recognize a specific protein (the CD4 antigen) found on the surface of a subgroup of cells involved in the immune response: helper T-cells, macrophages, and dendritic cells.

Binding to the CD4 antigen leads to uptake of the virion into the host cells, whereupon it is reverse transcribed and incorporated into the host cell genome. HIV can then lie dormant for an indeterminate period of time (up to a decade), producing no obvious symptoms. Eventually, however, the latent viral genome is “awakened” when the infected T-cell becomes activated to fight an infection. HIV then usurps cell function to produce more HIV particles and ultimately kills those cells, thus knocking out the immune system (hence, the name of this syndrome: acquired immunodeficiency syndrome, or AIDS). These immunocompromised individuals then go on to succumb to secondary opportunistic infections and cancers, and ultimately to death.

At this time, there is still no vaccine or other treatment for HIV-AIDS. However, disease progression can be controlled by anti-retroviral drugs. Since it was first identified in 1981, HIV-AIDS has taken approximately 35 million lives.

Other Pandemics

There have been many other global pandemics in human history, but space constraints prevent us from detailing all of them within this article. Many of them have been far less pathogenic or destructive, including measles. The common cold could also be included in this category, but we will address the family to which it belongs in detail below, given that the latter has now taken center stage on the global scene.

Polio virus is highly infectious, but a vast majority (72%) of those infected will not experience any vis-

ible symptoms, and the other quarter will experience flu-like symptoms; only a few percent of the latter group will also suffer horrific neurological problems (paresthesia [pins and needles in the legs], meningitis, paralysis).²⁵

Other epidemics have affected much smaller populations in more localized regions, including Zika virus. Ebola virus might otherwise have been relegated to this category within this article, but for its exceptional pathogenicity. The same might be said of two epidemics which were caused by coronaviruses: Severe Acute Respiratory Syndrome (SARS-CoV)²⁶ and Middle East Respiratory Syndrome (MERS-CoV). SARS-CoV will be discussed below in the context of the global pandemic which now threatens humanity.

Yet many other pandemics have been—and still are—as widespread and destructive as the ones described in detail above: cholera, typhus, tuberculosis, leprosy, malaria, yellow fever, and the list goes on. Each of these has a unique story and history, and can also involve other species within the great Web of Life. What place do these infectious agents and these pandemics occupy within God’s good creation? Do these represent a break from his perfect will for humanity? Or have we humans somehow brought these upon ourselves against his will? Questions like these are particularly relevant, given the great looming shadow cast by a microscopic pathogen—which some have dubbed “the invisible enemy”—across the globe today.

COVID-19 Knocks Us Down

Coronaviruses comprise a large group of RNA viruses of the subfamily *Orthocoronavirinae*. Computer modeling suggests that the common ancestor of all coronaviruses goes back 55 million years, which would suggest that it has been coevolving together with the bat and avian species which serve as reservoirs for this pathogen.²⁷ These infect the respiratory and/or digestive tracts and produce illness that can range from mild (the common cold) to lethal (see below). The virion comprises a positive-sense RNA genome (26,000–32,000 base pairs) enveloped within a host-derived lipid shell studded with club-shaped spikes which give the appearance in electron micrographs of a crown or a wreath (hence the name, derived from the Latin *corona*).

Coronaviruses first came to the attention of modern medical science when a particularly lethal strain (mortality rate of 40–90%) infected domestic chickens in the 1930s.²⁸ Other forms which affect other animals continued to be discovered: human coronaviruses were first identified in the 1960s.²⁹ These viruses enter the cells using the typical endocytotic pathway described several times above (binding of viral coat proteins with host cell surface membrane leading to invagination and entry, viral shedding being the reverse of this). However, the target proteins can vary between the strains of coronaviruses: for example, a porcine form infects the digestive tract epithelium by targeting the alanine aminopeptidase receptor protein,³⁰ while a human form infects the respiratory epithelium by targeting angiotensin-converting enzyme type 2 (ACE2).³¹

The sheer number of different types of coronaviruses, owing to constant changes in coat proteins and genomic content, account in part for why humans have not yet been able to develop immunity to infection and also why these infections are continuously present all year, every year. Until the COVID-19 crisis drew the attention of the entire world toward developing a vaccine for that particular subtype of coronavirus (see below), there had never been a viable vaccine for any form of coronavirus infection, including the last deadly coronavirus that struck the world almost two decades ago (SARS). To discuss here all the subtypes of coronaviruses is beyond the scope of this article. Instead, we will consider in detail only SARS-CoV, the severe and lethal global pandemic which was the predecessor of the one that is now dominating the attention of the world at this time: COVID-19 or SARS-CoV-2.³²

In November of 2002, SARS broke out in the city of Foshan, in the province of Guangdong, China. All patients reported fever, which could also often be accompanied by other flu-like symptoms such as muscle pain, lethargy, cough, and sore throat. About 10% of patients declined around day 7, with symptoms progressing to viral pneumonia or leading to a secondary bacterial pneumonia, both of which cause shortness of breath requiring supplemental oxygen and mechanical ventilatory assistance. As the name of this disease implies, it can lead to severe acute respiratory distress and fatality.

For weeks, local officials did not inform their general public, but eventually when they found that they could not control the outbreak, they began to tell

the public that people were being infected by exposure to live animals at the local market. However, as infections continued to rise and human-to-human transmission was increasingly being detected, Chinese government officials finally took the decision to be more forthcoming and to share information with World Health Organization (WHO) officials. This delay and secrecy greatly contributed to the virus spreading easily and widely: within months, it had spread to more than two dozen countries worldwide.

The reader will be forgiven for thinking that the preceding paragraphs were describing COVID-19, which is presently sweeping the globe: all of the details are eerily reminiscent of recent events. The reason is that COVID-19 is caused by a genetic sibling of SARS. There may be hope for us in the way later events transpired for SARS. It seemed that medical containment efforts quickly brought it under control, and the WHO declared SARS contained on July 5, 2003. However, another much smaller outbreak was reported in early 2004. By the time SARS had run its full course, it had infected over 8,000 people worldwide and killed almost 800, mostly in Asian countries. Both the numbers of cases and of deaths were greater in Canada than in the United States and were centered almost exclusively in southern Ontario. Treatment of SARS involves antivirals and antipyretics (to moderate the fever), and also supplemental oxygen and mechanical ventilation when required. It is also important to quarantine infected individuals.

Government labs in the United States (CDC) and Canada (National Microbiology Laboratory) obtained the genome for SARS in April 2003, and showed it to be caused by a coronavirus (SARS-CoV) which is spread through a respiratory route (droplets coming into contact with mucous membranes) rather than the fecal-oral route.³³ Intensive efforts to locate the source of this virus found horseshoe bats from a remote cave in Yunnan province as the natural reservoir:³⁴ the virus is then transmitted to humans either directly or through live animals in local markets, primarily Asian palm civets (a somewhat raccoon-like mammal). SARS-CoV can also appear in raccoon dogs, ferret badgers, and domestic cats. The authors of this investigative work also found other subtypes of coronaviruses in the bats of this cave, and considerable genetic recombination occurring between them, and stated ominously that,

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This cave could be regarded as a rich gene pool of bat SARSr-CoVs, wherein concurrent circulation of a high diversity of SARSr-CoV strains has led to an unusually diverse assemblage of SARSr-CoVs ... Various SARSr-CoVs capable of using human ACE2 are still circulating among bats in this region. Thus, the risk of spillover into people and emergence of a disease similar to SARS is possible. This is particularly important given that the nearest village to the bat cave we surveyed is only 1.1 km away, which indicates a potential risk of exposure to bats for the local residents. Thus, we propose that monitoring of SARSr-CoV evolution at this and other sites should continue, as well as examination of human behavioral risk for infection and serological surveys of people, to determine if spillover is already occurring at these sites and to design intervention strategies to avoid future disease emergence.³⁵

Two years later, this prophetic warning materialized in the form of COVID-19. The latter was first reported in the Chinese media in January of 2020.³⁶ The source of the outbreak has been traced to a market in Wuhan; bats are believed to be the reservoir hosts, and evidence suggests that COVID-19 was transmitted to humans through pangolins.³⁷ In fact, the evidence suggests that there was recombinant genetic exchange between bat and pangolin coronaviruses which eventually gave them the ability to infect humans by binding to ACE2.³⁸

At present, the medical/scientific community are wondering if COVID-19 should be viewed more as a blood-related disease than a respiratory disease. It does indeed use ACE2 to gain entry through the respiratory epithelium, and does wreak havoc on the lungs and gas exchange. But there is increasing evidence that, once inside the body, it also enters inflammatory cells through ACE2 to then initiate an inflammatory response and changes in platelets (possibly through changes in the megakaryocytes which produce the platelets), ultimately resulting in disrupted regulation of blood-clot formation.³⁹ Destructive, dangerous, and potentially deadly clots can then cause perfusion defects in the lung (leading to destruction and fibrosis), brain (stroke), heart (heart attack), kidneys, skin (rashes seen particularly in children), and many other organs. Health experts are now warning that even those who survive a COVID-19 infection may suffer life-long damage with tremendously negative impact on well-being, including cognitive defects and impaired lung and kidney function.

Although the development of a novel vaccine has otherwise always taken several years, the present situation with COVID-19 has focused all the world's immunological resources on this one task, and many different strategies were developed in parallel. Even optimists were pleasantly surprised when several candidates with exceptionally high efficacy (>90%) were developed within six months. In this "Pandemics" special issue, Rebecca Dielschneider describes this accomplishment in more detail as she focuses on another somewhat unforeseen obstacle which has come up in this strategy to protect the world against COVID-19: vaccine hesitancy.⁴⁰ Several polls have indicated that a significant fraction of the population would be reluctant to receive a vaccine.⁴¹ In addition to those in the anti-vaccine movement who would outrightly refuse any vaccine as a matter of principle (this is their *raison d'être*), many have expressed concerns about personal safety (e.g., the surprisingly common view that vaccines cause autism), especially about a strategy which involves direct introduction of foreign genetic material into one's genome, and a growing mistrust of governments and healthcare institutions. A lack of universal cooperation on any global vaccination effort is certain, given the present lack of compliance on the part of the general public (and even some government leaders) with respect to calls for wearing of facemasks, social distancing, testing, contact tracing, and quarantining. There are also massive logistical considerations in unrolling a global vaccination program, as well as ethical questions about deciding which subgroups of people should get priority for limited vaccine doses.

Also, in this issue, Mark Strand shares his experience in educating the general public about the science of COVID-19.⁴² The readers of *PSCF* can learn from his example and experience, and use our resources, abilities, and contacts to reach out to the broader community and improve public understanding of this particular issue, and many others (physical distancing, mask wearing, herd immunity).

COVID-19 Brings Us to the Discussion Table

Pandemics keep appearing in human history in new forms, but they share many features in common. Notwithstanding the morbidity and mortality which attend these blights on humanity, another important common feature is that they frequently play out in a series of "waves": in fact, this article is being published during a third (or fourth?) wave of COVID-19.

Over the course of these pandemics' destruction, they expose humanity's dark side (selfishness, greed, carelessness, hatred, defiance, xenophobia) and bright side (love, altruism, self-sacrifice, compassion, collaboration): they put a spotlight on the human condition. They force us to reconsider what is important, what is virtuous, and how we want to be remembered. Their effect on humanity is often a mixture of societal collapse and rebuilding. They tend to initiate a great deal of policy formulation and refining, often related to medical care (healthcare systems), hygiene (water sanitation, sewage control, hand washing, social distancing), vaccination (development, implementation, enforcement), and other chemical/biological weapons against the pathogens (toxins, phages, genetic vectors).

Given this backdrop of pandemics occurring frequently all through recorded human history, and the current state of alarm over COVID-19 coming two years after the quasi-prophetic statement from the authors of an investigation of the origins of the SARS pandemic only 15 years prior, now is the time for wide-ranging research, discussion, and planning for other potential pandemics, including the potential second, third, and fourth waves of COVID-19. The ASA/CSCA—comprising scientifically trained members with an inclination toward Christian faith—can make a unique and important contribution to this discussion. Again in this issue, Mark Strand offers an example and template,⁴³ and Rebecca Dielschneider identifies a timely and strategic focal point.⁴⁴

There are not only scientific, medical, and psychological questions, but also sociological, ethical, and economic considerations. How do we prepare for and respond to these looming threats? What kinds of questions need to be prioritized, and what policies and practices need to be developed? There are also theological and philosophical questions. Christopher Southgate has provided an excellent starting point,⁴⁵ and this journal will feature other starting points in future issues. How do we make sense of this event within Christian theology? What are the responsibilities of Christians in this? Are Christians called to put themselves into harm's way by "touching the leper" as Jesus did? Do we adopt the attitude that we are specially equipped with the awareness and knowledge that science provides, and also motivated by the Great Commission of the Gospel of Christ? What can we contribute in the way of advocacy and public education, admonition and leadership of the church body, new research directions and medical

strategies, suggestions for public policy, and setting examples of following recommendations from government and health experts? The ball is in your court as the challenge continues. ⇔

Notes

¹The sin of Achan (Joshua 7), or the fall of the houses of Eli (1 Samuel 2 and 3) or of Saul (1 Samuel 15; 2 Samuel 1; 1 Chronicles 10), all of which brought on national military defeat to Israel: the nation of Israel was taken into captivity.

²The testing of Job (Job 1:13–2:8), or the man born blind from birth (John 9:1–3).

³University of North Carolina Health Care, "Discovery Shows How Herpes Simplex Virus Reactivates in Neurons to Trigger Disease: About 90 Percent of Us Live with HSV inside Brain Cells," *ScienceDaily* (December 9, 2015), accessed June 29, 2020, www.sciencedaily.com/releases/2015/12/151209143105.htm.

⁴Centers for Disease Control and Prevention, "Frequently Asked Questions (FAQs)," CDC>Malaria>About Malaria, last reviewed September 20, 2020, accessed January 4, 2021, <https://www.cdc.gov/malaria/about/faqs.html>.

⁵Centers for Disease Control and Prevention, "Lyme Disease," last reviewed November 5, 2020, accessed January 4, 2021, <https://www.cdc.gov/Lyme>.

⁶Centers for Disease Control and Prevention, "Rabies," last reviewed September 25, 2020, accessed January 4, 2021, <https://www.cdc.gov/rabies/>.

⁷Joel O. Wertheim et al., "A Case for the Ancient Origin of Coronaviruses," *Journal of Virology* 87, no. 12 (June 2013): 7039–45, <https://doi.org/10.1128/JVI.03273-12>.

⁸For a useful overview of how this works in the specific case of influenza, see Thomas O. Edinger, Marie O. Pohl, and Silke Stertz, "Entry of Influenza A Virus: Host Factors and Antiviral Targets," *Journal of General Virology* 95, no. 2 (2014): 263–77, <https://doi.org/10.1099/vir.0.059477-0>.

⁹Centers for Disease Control and Prevention, "What Is Smallpox?," last reviewed June 7, 2016, accessed June 29, 2020, <https://www.cdc.gov/smallpox/about/index.html>; World Health Organization, "Smallpox: Historical Significance," accessed June 30, 2020, <https://web.archive.org/web/20070921235036/http://www.who.int/mediacentre/factsheets/smallpox/en/>; and J. N. Hays, *Epidemics and Pandemics: Their Impacts on Human History* (Santa Barbara, CA: ABC-CLIO, 2005), 151–52.

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¹²Maria del Mar Fernandez de Marco et al., "The Highly Virulent Variola and Monkeypox Viruses Express Secreted Inhibitors of Type I Interferon," *The Federation of American Societies for Experimental Biology Journal* 24, no. 5 (2010): 1479–88, <https://doi.org/10.1096/fj.09-144733>.

¹³Frank Fenner et al., *Smallpox and Its Eradication* (Geneva: World Health Organization, 1988).

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