

From Chimps to Bats: Human Social Evolution and Pandemic Potential over 15,000 Years

The ongoing COVID-19 pandemic, like past pandemics and those yet to come, is self-inflicted. For most of human history, our ancestors lived much like chimpanzees, in small, mobile groups and hosted few viruses as a result. With the evolution of farming, cities, and ever-improving transport networks, our more recent ancestors lived more like bats, in tightly packed clusters that encouraged the transmission, reproduction, and mutation of viruses and other potential pathogens. Unlike those of bats, human immune systems are not calibrated by some 64 million years of evolutionary adaptation to heavy viral loads. So our potential as hosts for global pandemics is strong.

The ongoing COVID-19 pandemic is a self-inflicted calamity in both a narrow and a very broad sense.¹ It is self-inflicted in a narrow sense because prompt and decisive action on the part of Chinese authorities in December of 2019 might have confined the outbreak to Wuhan; and because those parts of the world that suffer most from it – thus far Europe, Russia, the USA, India, and Brazil – grossly mismanaged their pandemic response. In most cases, ignorant and callous leaders, hoping to boost their political fortunes, magnified the suffering needlessly. These leaders undermined pandemic preparedness before COVID-19 hit, and when it did they chose not to listen to the advice of experts who warned of what SARS-CoV-2 (the virus behind the pandemic) might do.

But the pandemic is self-inflicted in a much broader sense, one for which no one is responsible. As a species, we have followed a path of social and cultural evolution that makes us extremely well suited to infectious pathogens such as the SARS-Cov-2 virus and respiratory viruses in general. That evolution was a slow process that no one planned or intended.

Chimps and Bats

Chimpanzees live in bands of roughly 25-80 individuals, and never more than 150. Their overall population today is somewhere between 170,000 and 300,000.

¹ Thanks to Ramachandra Guha, Andrew Meshnick, and the community of environmental historians at Georgetown University for their advice on an earlier draft.

While in the past their population was surely larger, because chimps only ever lived in African moist tropical forests, it probably never reached one million and certainly not ten million. Chimpanzees make poor habitat for viruses because they are mobile and almost never spend two nights in a row in the same nest, and because their group size and their overall numbers are small. The structure of their society is such that they do not offer infectious agents – pathogens – endless chains for transmission. As far as is known, they host only about 20 virus varieties.

For most of human history – about 300,000 years – our ancestors lived in social groups that approximated those of chimpanzees, our closest relatives in the animal kingdom. Our hominin ancestors diverged from chimpanzees, in terms of evolutionary biology, about 7 million years ago. But like chimpanzees, they lived in small bands, foraging, scavenging, and hunting in groups of 15-80. Their total population, until 15,000 years ago, was likely under one million and certainly less than ten million. They too made poor habitat for viruses and probably hosted very few.²

Bats, on the other hand, make superb habitat for viruses. About 1,400 bat species are known to science, accounting for roughly 20-25% of all known mammal species. Bats live three to four times longer than other mammals of the same weight. Some live past age 30. Thus they have more time than most creatures to catch and spread viruses. Although some bat species live solitary lives, most bats live in colonies, usually in caves, and spend much of their time roosting upside-down, tightly packed together, literally cheek by jowl. As many as 3,000 bats can squeeze into a square meter, allowing easy transmission of pathogens from bat to bat. Bats live all over the world, which exposes them to a broad spectrum of viruses. Their ranges sometimes overlap and – uniquely among mammals – they can fly and visit distant colonies enabling bat viruses to spread quickly over long distances. Most bats eat insects, although some prefer fruit and a very few species can eat the flesh or drink the blood of other animals. Taken together, the roughly 1,400 species of bats are omnivorous, and the variety of food they ingest also exposes them to a wide range of viruses. They host at least 1,000 varieties of coronavirus alone.

The overall population of bats, which cannot be known with accuracy, might be about one billion. A single cave in Texas hosts 20 million bats each summer.³ The sheer number of bats, the variety of bat species, and their long lifespans means bat viruses have abundant opportunity to mutate into new varieties. In sum, bats are extremely good at acquiring, hosting, sharing, and altering viruses.⁴

² We cannot know much about their health, because most of them lived in warm and moist environments in which both skeletal remains and DNA evidence – our best sources for ancient disease history – degrade easily.

³ <https://www.worldatlas.com/articles/bat-species.html> (retrieved 2 February 2021).

⁴ DUTHEIL et al. 2021; CYLITA et al. 2019.

Bats are also supremely good at tolerating viruses. They have immune systems that are superb at interfering with viral reproduction but (unlike human immune systems) without producing runaway inflammation. It is not entirely clear why that is so, but bats have roughly 64 million years of practice, or, put more precisely, of evolutionary adaptation to the wide range of viruses that infest their colonies. One hypothesis holds that the extreme effort involved in flying causes bats to secrete chemicals that limit inflammation.⁵

Viruses routinely use the habitat provided by bats to mutate into forms that can thrive in other animals. Several of the more dangerous human pathogens come originally from bats (diseases that jump from animals to humans are called zoonoses). Rabies migrated from bats to humans long ago. The viruses behind Ebola, Marburg, Nipah, SARS, MERS all made the jump to humans in the 20th century. Ebola has done so about 25 different times since 1970. Most recently, it appears SARS-Cov-2 has followed that well-worn path. Rodents and herd animals, which also lived in tightly packed communities and make good viral habitat, have also donated many viruses to humankind, although probably fewer than bats. Humans now host about 250 distinct viruses, probably about an order of magnitude higher than the number that circulated among our remote ancestors.⁶

Agriculture and Cities

The foremost reason why we have become so suitable to bat viruses, or their close relatives, is that we now live more like bats and less like chimps. The first steps in this long saga of social and cultural evolution were the emergence of farming villages and then of cities.

The first transition to agriculture, as far as is known, began in Southwest Asia beginning about 12,000 years ago. Others, entirely independent of the first, occurred in China, New Guinea, Africa, South America, and North America – all before 2000 BCE and sometimes long before. All were gradual, and all seeded further transitions in neighboring lands. Neighboring peoples sometimes learned agriculture from those who practiced it. Sometimes those who practiced it took the lands of those who didn't. One way or another, farming spread widely over thousands of years becoming the way of life of the majority of humankind.⁷

When people took up farming they often settled down. Some farming peoples, especially those operating in forest biomes, practiced rotational forms of farming, often called shifting agriculture or swidden farming. That could entail moving every few years. But in most cases, farming meant settlement and village

⁵ IRVING et al. 2021; SKIRMUNTT et al. 2020.

⁶ MOLLENTZE & STREICKER 2020; OLIVAL et al. 2015; CYLITA et al. 2019.

⁷ For a recent summary, see: BOGUCKI 2019: 83-117.

life. Farming villages might number a few hundred people, or, eventually, a few thousand. Now, for the first time, people commonly lived in communities larger than those of chimpanzees. Larger interactive social groups offered slightly better opportunities for infectious pathogens.

So did larger populations. Farming allowed faster population growth, because people could produce food in greater quantities than they could gather or hunt it. As a rough guide, population densities among farmers rose ten times higher than those among foraging and hunting peoples. With larger populations, people encountered one another more often, and shared pathogens more frequently. At the same time, pathogens, having more human bodies in which to reproduce, increased their populations and their mutation frequencies. While most mutations were without consequence, every now and then a mutation proved advantageous to a pathogen's chances at survival and reproduction. With enough such changes, new pathogens emerged adding to the variety hosted by human beings. The more human bodies, the more mutations; and the more mutations, the more novel pathogens evolved.

In addition, in several settings where people took up farming, they also domesticated herd animals soon thereafter. In Africa and Asia, farming people came to live amid goats, sheep, cattle, pigs, chickens and other animals. This created microbial stewpots in which pathogens from several species had the opportunity to mutate and jump to other species.⁸ Farming, in short, created communities, human and non-human mixed together, that proved welcoming to viruses and pathogens in general. Where villages were numerous enough, epidemics that affected tens of thousands of people and ranged over thousands of square kilometers now became possible.

While farming and village life were spreading, and population growing, social evolution took another fateful turn: the emergence of urban life. The first cities appeared in what is now southern Iraq – Mesopotamia – about 3500 BCE. Cities appeared in the Indus Valley by 2600 BCE, in China by 1700 BCE, in Mesoamerica by 800 BCE, and in sub-Saharan Africa by 800 CE. The first cities were small of course, with only a few thousand people. It was probably not until the final centuries BCE that any city acquired a population of 250,000. Chang'an in China and Rome were likely the first to do so. In 1000 CE about 2-4% of people around the world lived in cities. By 1800, perhaps 3-5% did. Urban life remained rare until recently.

But then, with the advent of fossil fuels and industrialization some of the constraints on city living relaxed. By 1900, some 10-15% of people lived in cities, and by 1960 34% did. Today the urban proportion is about 55% and growing steadily.⁹

⁸ Details are summarized in: SCOTT 2017.

⁹ Useful data appear in Our World in Data: <https://ourworldindata.org/grapher/urbanization-last-500-years> (retrieved 2 February 2021).

Urbanization proved highly favorable from the point of view of viruses and infectious pathogens generally. Packing people tightly together, almost like bat colonies, accelerated the transmission of pathogens, especially respiratory viruses. In particular, city life made it possible for acute respiratory viruses to stay in circulation indefinitely. Viruses such as measles, smallpox, or mumps require a certain minimum interactive population to survive because they either kill or immunize their hosts. They need a lot of potential hosts in order to reliably get from one to another. In the case of measles, one of the most easily transmissible of human viruses, that minimum number is about 250,000. Without cities of that size, or closely clustered villages collectively of that size, measles cannot survive for long.

One recent interpretation of the genomic evidence is that measles appeared as a human disease, separating itself from its closest relative, the cattle disease rinderpest, at about 500-100 BCE, which is when Chang'an and Rome attained a size sufficient to keep measles going. That hypothesis is far from secure. The uncertainties are legion and the quantity of evidence is small. In any case, the simultaneity might be coincidence; networks of interacting villages with total populations of 250,000 probably would have sufficed.¹⁰

Networks of Cities

Cities as a form of social organization in Eurasia spread at first mainly at temperate latitudes from origins in Southwest Asia, northern India, and northern China. By 2200 BCE they had begun to form networks, the first of which connected Egypt, the Levant, Mesopotamia and the Indus Valley. By 100 CE, a vast network of interlinked cities spanned the Eurasian landmass, from Spain to Korea, and included cities on the North African shores of the Mediterranean Sea. Each of these cities was in turn connected to a hinterland of villages that supplied urban populations with food and fuel. By the time of the Roman and Han empires, which flourished in the first and second centuries CE, epidemics affecting millions of people became possible for the first time. (The world's total population in 100 CE was probably about 200 million.)

It is only after about 100 CE that the historical record begins to feature accounts of major epidemics that perhaps killed tens of millions. In every case, the magnitude of the epidemic is uncertain. The ancient textual sources usually offer no numbers, and when they do include numbers, they are rarely worth trusting. So it is impossible to say with confidence just how widespread, for example, was the so-called Antonine plague of the 160s CE, described by the ancient Greek medical writer Galen. It is often diagnosed as either measles or smallpox, or perhaps multiple infections at once, but no reliable identification is yet possible. By some estimates it killed a

¹⁰ DÜX et al. 2020.

few million in the Roman Empire alone, and it may have extended to China, for which there are records of deadly epidemics in the 160s. It is also impossible to specify the cause or the magnitude of the plague of Cyprian, ca. 250-270 CE, which contributed to the travails of the Roman Empire in its lengthy 3rd-century crisis.¹¹ The plague of Justinian in the 540s CE can now be more confidently identified as plague (*Yersinia pestis*), but controversy reigns concerning its dimensions. Some say it may have killed a quarter of the population of the Byzantine Empire, while others suggest its impact was trivial outside of the Empire's capital of Constantinople.¹²

While the Eurasian network of interlinked cities and villages extended from the Mediterranean to China's Pacific shore, smaller networks of linked cities, and their village hinterlands, developed in the Americas and in the African Sahel. Both of these networks were forming by 800 CE if not before, but only slowly. They involved far fewer people, far smaller territories, and were less tightly integrated than the Eurasian network of interlinked cities. And in the Americas, the presence of domesticated animals before Columbus's voyages of the 1490s was very limited, reducing the opportunity for pathogens to circulate among flocks and herds and, from time to time, to infect humans with zoonoses.

The multiple networks of interlinked cities on various continents fused into a global network by 1700 or 1800CE. The first major step along this path was the emergence of an inter-connected Indian Ocean world. Sailors from every shore between East Africa and the islands that are now Indonesia forged links among hundreds of port cities around the Indian Ocean in a slow process already underway in ancient times. River and caravan routes increasingly bound these port-city networks to the inland settlements of Eurasia. By 1400 CE, this Indian Ocean world was well integrated, providing opportunities for infections from any of its coastal regions to spread to all others. This development in effect enlarged the Eurasian network by adding to it several cities along the East African coast as far south as Sofala, in today's Mozambique.

In a similar expansion – the second major step on the path to a global network -- the cities emerging in the West African Sahel became connected to those of Mediterranean North Africa. In this case, it was not sailors that forged the links in question, but camel-drivers who learned caravan routes across the Sahara, beginning in a small way by perhaps 500 CE, and in a regular, sustained way by 800 CE. The strength of these trans-Saharan connections only grew over time.

This enlarged network hosted the greatest disease disaster in all human history in the middle of the 14th century. Bubonic plague killed tens of millions between

¹¹ HUEBNER 2021; HARPER 2015.

¹² For various perspectives, see: HARPER 2017; MORDECHAI et al. 2019: 25546-25554; MORDECHAI & EISENBERG 2019: 3-50; EICHENBERG & MORDECHAI 2020.

1347 and 1355 in Europe, North Africa, Southwest Asia, and possibly in West Africa, around the shores of the Indian Ocean, and in China. Where the documentation is strongest, in Europe and the Arab lands, it killed one-third to one-half the population. The shipping and caravan routes linking the major population centers made it possible for this single epidemic, commonly called the Black Death, to reach a large proportion, perhaps even the majority, of humankind. It did not, however, affect the Americas, Australia, or Oceania.

The third major step on the path to a global network began in the 1490s. Oceanic voyaging undertaken first by Iberian sailors knitted almost the whole inhabited world together. This step, like its predecessors, took time. It was not until the late 18th century that regular connections were forged between the wider world and the Pacific coasts of either North America or Australia. But with each passing decade after Columbus, tighter and tighter links developed binding peoples on every continent.

In the same centuries in which sailors began to crisscross the Atlantic and Pacific, Russians and Cossacks built connections to formerly isolated peoples in Siberia. By the mid-17th century they, like the indigenous populations of the Americas, were now in systematic contact with Europe, and indirectly with every major center of population on Earth.

These new connections brought terrible epidemics of measles, smallpox, influenza and other acute infections to the Americas, Siberia, and Australia. Indigenous populations fell by 50% in some places, and 95% in others. By far the greatest losses occurred in the Americas, where total population in 1492 was probably between 40 million and 70 million – and by 1650 only about 8 to 15 million (of whom many, unlike in 1492, were of European or African birth or ancestry). In proportional terms, the losses in Siberia and Australia were nearly as great, but the totals involved were far smaller, no more than 2 or 3 million in all.¹³

Global Pandemics

So between 800 and 1800, the various networks of cities around the world gradually fused into a single global-scale network. Infectious diseases could now spread among populations on every continent and almost every inhabited island. And they did so, intermittently. Their transmission was often checked by the length of voyages across the oceans.

Consider the challenges to transatlantic travel in the 16th century from the perspective of the smallpox virus. To get across the ocean from Europe or Africa required some unlikely coincidences: first, on the day of departure, in Seville

¹³ These epidemics are summarized in: CROSBY 1986; ALCHON 2003; LIVI-BACCI 2007.

or Lisbon or El Mina or Goree Island, at least one infected sailor or passenger, showing no symptoms (symptomatic cases would hardly be allowed on shipboard) had to embark. That means someone in the first week or two of infection, because smallpox symptoms such as a rash become readily visible usually within ten days. Second, several other people capable of hosting the virus had to be on board. Only a small proportion of adult Africans and Europeans had not already encountered smallpox and become immune to it for life (as all smallpox survivors do). So on most voyages, no infected people and no one capable of hosting the virus embarked and neither of these first two conditions was met. On those voyages when both conditions were met, the original infected individual had to breathe on at least one other susceptible person, who in turn had to convey the virus to a third susceptible person and so on. Transatlantic voyages in the 16th century took 6-7 weeks, often longer, while bodies infected with smallpox remained infectious for only a week or ten days. The probability of smallpox making it along a chain of susceptible hosts lengthy enough to arrive in the Americas was low. In most crews, all of the bodies into which a cough or sneeze might propel a load of virus were immune virus stoppers. So a successful transatlantic crossing for any smallpox virus required several unlikely circumstances. It likely took 26 years after Columbus's first voyage, until 1518, before smallpox established a beachhead in the Americas. Measles took even longer, probably until the 1530s, to cross the Atlantic.

Sea voyages across the Pacific took even longer during the age of sail, making it almost impossible to transmit acute viral diseases from Asia all the way to the Americas or vice-versa. However, ships' crews did transmit deadly diseases to the inhabitants of almost every Pacific Island by 1820, with horrific losses of population in every case.¹⁴

Global pandemics, by which I mean ones that affect people on every inhabited continent, became likely only after the age of sail gave way to the age of steam. In order for infections to reach people all over the world, infected people had to be able to move more quickly. With the advent of railroads and steamships by the 1830s, the opportunity for global pandemics magnified greatly. Within a few decades, regular steamship services united the major seaports of the world, while scheduled train service connected many cities, large and small, on each continent.

In addition, thanks to the improvement of transportation systems that could bring food and fuel to urban centers, cities could grow bigger than ever before. London's population surpassed 1 million in about 1800 and 3 million by 1850. A large proportion of Londoners lived in poverty and squalor, conditions well suited to the spread of infectious disease. Other cities, especially in the industrializing

¹⁴ RALLU 1990.

regions of the world, also grew rapidly, increasing the number of people exposed to a higher risk of disease transmission.

A further development raised the likelihood of global-scale pandemics as well. By the 19th century, the British Empire, and to a lesser extent other colonial seaborne empires, had become nearly global in reach. Naval ships and army detachments moved around the empire in accordance with military priorities, and often carried infections with them. In short, in the 19th century faster and more frequent commerce, migration, and military movements tightened the global network of ports and cities, sharply improving the odds of global pandemics.

It is possible that genuinely global pandemics of influenza, for example, existed before all these 19th-century changes in transport, urbanization, imperial reach, but there is no clear evidence one way or the other. There is abundant evidence, however, for a worldwide pandemic of cholera in 1832-34.

Cholera is a bacterial disease that originated in the warm, brackish, coastal and estuarine waters of the Bay of Bengal.¹⁵ It is transmitted from one victim to the next by contaminated food or water. Today it is easy to treat, but in the 19th century certain strains of *Vibrio cholerae* produced deadly infection that could kill people within hours of the onset of the first symptoms. Roughly half of all those who contracted cholera died of it. It apparently remained confined to South Asia until 1817. Between 1817 and 1823, it spread along the shipping routes of the Indian Ocean world, from the Persian Gulf to the Philippines. It also reached inland to Syria, Anatolia, and southern Russia. This was a giant regional epidemic, often called the first cholera pandemic. But it was not global. It never reached the Americas, Australia, or, as far as is known, most of Africa.

In 1830, cholera escaped Bengal once again. Travelers carried it to Russia and Europe in 1831. By 1832 it reached the Americas, from Quebec to Mexico and Cuba. It even reached Australia. A third cholera outbreak, in 1852-59, also reached pandemic proportions, infecting people on every continent. Three more followed by 1930, but by this point effective sanitation in the richer parts of the world, combined with international cooperation targeting disease transmission, reduced cholera's impact. By the mid-20th century antibiotics would reduce it further. The age of global cholera pandemics had passed, although the disease remained a menace in the poorer parts of the world where sanitation infrastructure was too expensive for available budgets – as it does today.

By 1832, the world had evolved so as to make global-scale cholera pandemics possible. It was difficult, however, for most other infectious disease to break out as global pandemics, despite the tightly connected world of the 19th-century age of steam. The reason was that most of the deadly infectious diseases had already

¹⁵ Cholera history is ably summarized in: HAMLIN 2009; and with respect to Africa in: ECHE-NBERG 2011.

slowly spread to many parts of the world and become endemic. That meant that almost everyone contracted them in childhood. In the case of most acute viral infections, those people who survived childhood acquired resistance and in many cases lifetime immunity.

That did not apply, however, to influenza. It flourishes in pigs and birds as well as humans, which helps the virus to mutate very rapidly. Influenza evolves new forms every year. In 1918 it mutated into a particularly virulent variant that spread rapidly around the world. It is not clear exactly where the new virus originated. But troop movements associated with the final stages of World War I, and the subsequent demobilization of millions of soldiers, assured rapid spread to every continent. The transport infrastructure that had enabled cholera to launch an age of global pandemics in 1832, was, by 1918, more widespread and efficient. Routes and regular service reached more cities and towns in more countries. Travel by train or ship was faster than in 1832. Moreover, the British Empire, and other European empires, had grown bigger by 1918 than in 1832, especially in Africa, India, and Oceania. That meant that the movement of military personnel affected more parts of the world than before, and of course in 1917-1918 the magnitude of military mobilization peaked, involving roughly 60 million men in all or about 3% of humankind. These changes, all of which were intensifications of patterns put in place in the 19th century, invited havoc once a highly transmissible, breath-borne virus, the strain of influenza called H1N1, evolved. The total death toll around the world came to somewhere between 16 million and 100 million—considerably more than the combat deaths of World War I.¹⁶

Conclusion

Since the 1918 influenza, the human population has quadrupled. The proportion of people living in cities has also quadrupled, so the total urban population is about 16 times as large as in 1918. Billions of us live packed close together, sharing our breath with dozens or hundreds of our fellow humans every day. Meanwhile, air travel, which became routine in many parts of the world between 1960 and 1980, sharply reduced transit times over long distances for both people and pathogens. Whereas the 1918 flu would have taken at least seven days to cross the Atlantic aboard troop ships, nowadays infections traveling inside the bodies of airplane passengers make the same trip in seven hours. There are roughly 40,000 airplanes in the world, and before COVID-19 there were about 100,000 flights every day. Thousands of cities around the world have airports. Global connections are faster and more numerous than ever before, and link populations that are bigger than

¹⁶ For conflicting estimates, see: PATTERSON & PYLE 1991: 4-21; JOHNSON & MÜLLER 2002: 105-115; and SPREEUWENBERG et al. 2018: 2561-2567.

ever before. In short, social and cultural evolution – history – has created a human world that in important respects resembles the world of bats.

For tens of millions of years, bats have flown from colony to colony, and, when not flying, huddled together in tightly packed crowds. Now most of us do the same. Over those tens of millions of years, bats evolved immune systems that allow them to host abundant varieties of viruses without harm to themselves. We have not had the time to do the same. Our immune systems are much the same, perhaps a little better, than they were 15,000 years ago when almost all of us lived like chimpanzees in small bands and moved around within limited ranges at walking speed. Hence we have created a mismatch between our immune systems and the environment we have built for the roughly 250 viruses and other infectious pathogens capable of replicating in our bodies. We have made ourselves exquisitely vulnerable to easily transmitted infections. That is why the current pandemic, like those that have come before, and those that are yet to come, is self-inflicted.

As historians of medicine have shown, in the past 150 years our species has enjoyed considerable success in building a surrogate for immune systems calibrated for our bat-like existence. Sanitation measures, vaccines, antibiotics and other drugs protect against a wide array of illnesses. But those novelties are still very unevenly available around the world. As yet, they make an incomplete substitute for a tight match between a species' immune system and its disease environment.

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Electronic documentation

Our World in Data: <https://ourworldindata.org/grapher/urbanization-last-500-years>

World Atlas: <https://www.worldatlas.com/articles/bat-species.html>

Od čimpanzi do šišmiša. Društvena evolucija i pandemijski potencijal tijekom 15.000 godina

Baš kao i dosadašnje pandemije, ali i one koja nam tek predstoje, aktualnu pandemiju bolesti COVID-19 prouzročio je sam čovjek. Glavni razlog tome je taj što je ljudska vrsta prigrllila način života neprikladan za ljudski imunosni sustav. Kroz veći dio povijesti čovječanstva, naši preci živjeli su uvelike poput čimpanza, u malim i pokretljivim skupinama, zbog čega se među njima udomaćilo malo virusa. Zbog toga se naš imunosni sustav prilagodio tek malom broju zaraza koje su najčešće pogađale naše pretke iz kamenog doba.

S razvojem poljoprivrede, gradova i sve boljih prometnih mreža, život naših suvremenijih predaka, baš kao i nas samih, sve je više nalikovao životu šišmiša, a sve manje onome čimpanza. Najčešće živimo u gusto zbijenim zajednicama koje su pogodovale prijenosu, reprodukciji i mutaciji virusa i drugih potencijalnih patogena. Na to kako nas bolesti pogađaju kao vrstu posebno su utjecala tri procesa. Prvi, koji je započeo prije više od 10 000 godina, odnosi se na prelazak na sjedilački način života i bavljenje poljoprivredom, što se često, mada ne i uvijek, odvijalo ruku pod ruku. Time se drastično povećala izloženost ljudi virusima i drugim patogenima povezanim s domaćim životinjama, iako samo u slučajevima kad je taj proces pratilo i udomaćivanje divljih životinjskih vrsta. Tako je porasla i izloženost ljudi patogenima povezanim s ljudskim otpadom. Drugi proces odnosi se na urbanizaciju i prenapučenost koju je donijela sa sobom. Time se drastično povećala izloženost ljudi patogenima koji se prenose dahom. Treći proces odnosi se na globalizaciju koja je otpočela uspostavom redovnih i stalnih interakcija među urbanim zajednicama, koje su se u početku odvijale uglavnom u Aziji. Proteklih 4000 godina obilježio je dugotrajan trend povećanja učestalosti i intenziteta interakcija među ljudskim zajednicama. U 15. i 16. stoljeću taj je proces poprimio istinski globalne razmjere zahvaljujući preoceanskoj plovidbi. U 19. i 20. stoljeću još se više ubrzao zahvaljujući tehnološkim pomacima u prometu,

poput željeznice, parobroda i zrakoplova. Globalizacija je jamstvo brzog širenja zaraznih patogena, što nam pandemija bolesti COVID-19 opetovano pokazuje, pri čemu je spetakar patogena kojima smo danas izloženi sve širi, a učestalost kojom se s njima susrećemo sve je veća.

Za razliku od imunosnog sustava šišmiša, ljudski imunosni sustav nema tu prednost da je prošao kroz 64 milijuna godina evolucijske prilagodbe velikim virusnim opterećenjima. Namjesto toga, evolucijska prilagodba poderala ga je tako da se može nositi tek s ograničenim spektrom zaraznih bolesti uobičajenih među našim hominidnim precima. Stoga je potencijal koji imamo kao domaćini uzročnika globalnih pandemija veći no ikad. Od ostvarenja tog potencijala mogu nas spasiti jedino djelotvorne javnozdravstvene mjere.

Ključne riječi: COVID-19, pandemija, šišmiši, povijesni razvoj, prometne mreže

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