If viruses were capable of emotion, they would commemorate the day the World Health Organization (WHO) declared coronavirus 2019 (COVID-19) a pandemic and celebrated each time a country announced lockdown to contain the spread of the respiratory illness. For what SARS-CoV-2, or severe acute respiratory syndrome coronavirus 2, has achieved is no mean feat for its family.

Yes, there exist hundreds of coronaviruses out there. Till the early 21st century, they were mostly known to circulate among pigs, camels, bats and cats and cause mild form of common colds in humans. Virologists’ attention turned to it in 2002, when one member jumped from a horseshoe bat to a person, possibly via a civet cat, and went on to cause severe acute respiratory syndrome (SARS) among 8,500 people and killed over 900. Just like COVID-19, symptoms included fever, sore throat, shortness of breath and pneumonia. A decade later, another coronavirus, believed to have originated from bats but transmitted to humans from camel, caused a similar outbreak in Saudi Arabia. It was named Middle East...
respiratory Syndrome (MERS). Despite high fatality rates—9.5 per cent in case of SARS and 34 per cent for MERS—none of the coronaviruses could cause large-scale outbreaks. While SARS-CoV appears to have disappeared in 2004, MERS-CoV causes limited outbreaks. Now, call it a third time lucky or outcome of an evolutionary strategy, SARS-CoV-2, despite a low fatality rate of 2-5 per cent, has emerged as the most devastating pandemic since the 1918 Spanish flu.

The success of SARS-CoV-2 is also no mean feat when compared with other trillions of pathogens that naturally get transmitted between animals and humans but more often than not fail to establish a disease in human population, let alone cause major epidemics.

Most of these zoonotic pathogens, be it a virus, bacterium, fungus or parasites (protozoa and helminths), are believed to be host-specific. This means they usually restrict themselves to a limited number of species, such as bats, pigs, rats and chimpanzees, and prefer residing in them by creating a life cycle reservoir. This trait of pathogens is due to species barriers. Along with the human body’s resilience system against diseases, species barriers help us most of the time lead a life free of infections despite living in a pathogen-filled world. Crossing it is not easy as these barriers are determined by the level of human exposure to pathogens (directly through faeces or body fluids like saliva, blood and urine, or of an infected animal, or indirectly through areas where they live and roam, or contaminated surfaces) and their ability to infect a human and cope with the new host’s immune response. It thus requires the pathogen to undergo specific changes through mutation or genetic exchanges with the host.

However, these mutations are not always successful. Thus, says Abi Vanak, disease ecologist at the Ashoka Trust for Research in Ecology and the Environment (ATREE), Bengaluru, a vast majority of animal to human spillover likely result in dead-end for the virus (and other pathogens). This means the pathogen does not get transmitted beyond the contacted person, he adds.

There have been instances when the pathogen has managed to hop on to humans but does not cause mortality or morbidity in them. Early this year, researchers in Brazilian states of Tocantins and Amapa have identified one Ambidensovirus in patients with symptoms similar to dengue or Zika. "Viral species in this genus have been described only in insects, shellfish and other invertebrates; never in mammals," the researchers wrote in the March issue of journal PLoS ONE. They are, however, not sure if Ambidensovirus is responsible for the patients’ morbidity.

THEN WHO AILS AND WHY

The US Centres for Disease Control and Prevention recognises 1,407 human pathogens; 60 per cent of them are zoonotic.

These pathogens have managed to cross species barriers and establish diseases in human population. However, most of these pathogens maintain their life cycle

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### Viruses lead disease outbreaks

Viruses form just 14% of the total human pathogens. But among new and emerging pathogens, they account for 44%
Invincible mutant

In its journey from a bat to a pangolin or a snake and then to humans, SARS-CoV-2 mutated to iron out any glitches.

Angiotensin-converting enzyme 2 (ACE2) is an enzyme attached to the outer surface of human cells in the lungs, arteries, heart, kidney, and intestines. Both SARS CoV and SARS CoV2 use these cells as receptors for infecting humans.

But SARS-CoV-2 went through mutations which has enabled it to bind with ACE2 more efficiently, making it more successful at human to human transmission.

This invisible feature on the crown of SARS-CoV-2 is the reason why it has a death toll of 265,000 compared to 914 by its cousin, SARS-CoV.

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available for nine of these diseases.

HIV, which is responsible for a majority of HIV infections worldwide, is one such virus. It made the jump from African primates to human as a result of bushmeat eating in the 1970s. Subsequently, it has established a life cycle reservoir in humans. The Ebola virus, which causes a severe haemorrhagic fever with fatality rate up to 90 per cent, however, shows what a virus is capable of to ensure its transmission. Since its first detection in 1976 in Sudan and Democratic Republic of Congo, the virus has managed to cause outbreaks without establishing a natural reservoir in humans. It has rather mutated to develop the ability to transmit from human to human. This newfound ability of Ebola came to notice during the 2013 outbreak when it spilled over, possibly from a bat to a 18-month-old boy in Guinea. Within months, it became a global epidemic.

But the ongoing pandemic by SARS-COV-2 shows how zoonotic pathogens are constantly honing their genome to expand their realm. A study published in journal Antiviral Research in April, throws some light on this. Despite a genome sequence highly similar to that of other SARS-like coronaviruses, SARS-COV-2 differs from SARS-COV in its interaction with ACE2 (angiotensin-converting enzyme 20), a crucial enzyme that remains attached to the outer surface of human cells in the lungs, arteries, heart, kidney and intestines. Both SARS-COV and SARS-COV-2 use these cells as receptors for infecting humans. According to the study, somewhere in its journey from a bat to a pangolin or a snake and finally to humans—the pathway still remains shrouded in mystery—the SARS-COV-2 went through mutations which has enabled it to bind with ACE2 more efficiently, making it more successful at infecting a person and ensuring human to human transmission despite a low reproduction rate (2) and fatality (2-5 per cent). By comparison, SARS-COV had a reproduction rate of 2.8 and fatality rate of 9.5 per cent. In a way, these mutations are the reason SARS-COV-2 had a death toll of over 265,000 by the first week of May compared to its older cousin that killed just 914 over two years.

Emergence of such robust and intelligent pathogens are worrying for another reason. This global coup is largely led by viruses, particularly RNA viruses that can exploit all known mechanisms of genetic variation to cause epidemic spread.

VIRUSES LEAD THE COUP
Considered often a non-living entity, viruses can infect all life forms, including microbes like bacteria. This is probably because this submicroscopic particle, made either of RNA or DNA as its genetic material, can replicate or produce multiple copies of themselves only when it is inside a living host cell. Most of the jumps to humans are, however, made by RNA viruses that account for some 44 per cent of emerging
infectious diseases, says a study, published in *ilar Journal* in 2017. In fact, some of the biggest zoonoses like chikungunya, dengue, Zika, avian influenza, Lassa fever, Ebolavirus, MERS, SARS are all caused by RNA viruses.

These RNA viruses are considered to have recent evolutionary origins. Their mutation rate can be 1,00,000 times higher than DNA viruses. “RNA viruses show remarkable ability to adapt to new environments and confront different selective pressures they encounter. This not only include the host’s immune system and defense mechanisms but also the current artificial challenges devised by the biomedical community,” notes the *ilar Journal* study. This high rate of mutation of a RNA virus is because of the way it replicates. In DNA viruses, several proteins correct if there is any faulty genome replication. But RNA viruses replicate without this proofreading process to increases their mutation rates. But this has a downside: any undesirable mutation can negatively impact the fitness of the virus.

Among the RNA virus group, coronaviruses have been found to have overcome this tradeoff between mutation rate and incorrect replication. A study published in *Plos Pathogens* in May 2010 says SARS-CoV had mutated to produce an enzyme that diminishes the number of mutations. “The viruses might switch the proofreading mechanisms on or off depending on the context, allowing them to rapidly adapt to new environments without losing replicative fidelity,” the study says.

**OUR TRANSGRESSION**

Ultimately, it’s about humans and their interaction with other species, whether in the wild or in farming. It’s also about how we transgress into the habitat of wild species or “manufacture” food from domesticated animals. For instance, villages in the eastern foothills of the Western Ghats in India regularly experience outbreaks of...
Kyasanur forest disease (KFD), a viral haemorrhagic fever similar to Ebola and dengue that spreads by ticks, (Hemaphysalis spinigera) living on monkeys. People here mostly depend on forests for living (see 'Jungle stirs virus', p47). SARS and COVID-19 outbreaks have also been linked to exposure to the viruses in the Chinese wet markets. Interconnectedness of the world has just made the spread massive and instantaneous.

It’s not just interface with wildlife, livestock also plays a role. In the case of Spanish flu, it is widely held that the avian influenza virus jumped from a pig on a military farm in Kansas, US, to the first known human case. Though there are other theories about where the "jump" took place, from Europe to China, what’s clear is that the virus mutated from animals and was taken across the world by the movement of soldiers during the World War I. Ultimately “Spanish flu” killed more people than the war.

So, it is a combination of factors—movement of people, living conditions, population density and, of course, eating habits—that makes the virus more deadly in its new host. Ebola, for instance, was not new to parts of Africa even though outbreaks were reported way back in 1976. What changed between then and the outbreaks of 2013-14 was the demography in the affected countries, says Sanath Muliya, scientist at the Wildlife Institute of India, Dehradun. Between the 1960s and early 2010s, population density increased by 223 per cent in Guinea, 178 per cent in Sierra Leone and by 275 per cent in Liberia, particularly in urban parts that experienced high rural-to-urban migration. All major outbreaks occurred in such urbanised set up with high human densities, says Muliya.

A similar development in Indonesia in 1998-1999 resulted in the first outbreak of Nipah virus infection, but in neighbouring Malaysia. The virus is naturally harboured by pteropid fruit bats. But in the months before the outbreak, large-scale deforestation was going on in Indonesia for pulpwod. Palm oil industries had also prompted slash-and-burn of forests for setting up industrial plantations. While deforestation destroyed the bat’s habitat, the haze reduced flowering and fruiting of forest trees. Reduced rainfall caused by the severe 1997-1998 El Niño conditions exacerbated the situation, resulting in mass migration of pteropid bats to Malaysia, which was experiencing an upsurge of large-scale piggeries with fruit orchards on their edges. A combination of factors led to the spillover of a novel virus from the bat to the domestic pig and then to pig farmers.

The scope and scale of deforestation and the opening of new interfaces with forests and wildlife increase the chances of spillovers, says Prashanth N S of the Institute of Public Health, Bengaluru, adding, “The way in which we interact with our environment has increased the exposure to newer pathogens that would have otherwise not come into contact with large populations.”

**INDUSTRIAL CHURNING**

*Even as subsistence farmers and herders coped with zoonoses, the very nature of industrial food business has exacerbated the issue*

The influenza A(h1n1) virus—swine flu—is not transmitted from human to humans by eating pork, and that remains its saving grace. Today, it is widely accepted that swine flu was first found in human beings in La Gloria, a little town in Mexico. It is known that a young boy suffering from fever in March 2009 became the first confirmed victim of the outbreak, which then spread from country to country. But then, when the disease broke out, what was quickly lost in this tragedy was the location of the ill-fated town—right next to one of Mexico’s biggest hog factories, owned by the world’s largest pig processor Smithfield.
Foods. What was also not said that people in the town had repeatedly protested about water pollution, terrible stench and waste against the food giant. While this fact was never followed up or uncovered, what was reported was that food majors wanted WHO to change the name of the contagion so that pork eating would not be affected. Virologists at US CDC, however, based on genetic fingerprinting found that the strain of this swine flu is the same as first identified on industrial pig farms in North Carolina, the hub of industrial pig farms in USA.

The H1N1 strain is high on the evolutionary ladder. In 1998, when there was an outbreak of swine flu among pig herds in North Carolina, it was a triple hybrid—containing gene segments from human, bird and classical swine influenza viruses—that spread across pig herds of the integrated world. Then it mutated further. Today, it is believed that common flu virus infecting humans has got mixed with the hybrid, creating an altogether new human-animal virus.

In 1997, when the world first caught avian flu (H5N1), wild migratory birds that are natural carriers of the virus, had been widely indicted for the spread, but with little evidence. It was easier to blame wild birds with no defenders in agribusiness, than birds produced in poultry factory farms. The problem
stemmed from the model of growing chicken in an environment that is highly conducive for the virus. The birds are raised in tightly confined, often poorly ventilated enclosures with regular exposure to chemicals, blood and faecal matter. Diseases can spread, and spread fast, in such conditions. Since the birds also have lowered immunity because of their genetic uniformity, they are almost literally sitting ducks when disease hits.

But after avian flu hit Asia, the Food and Agriculture Organization (FAO) told governments that while it would be possible to tighten biosafety in commercial poultry farms, it would be impossible to do it for non-commercial enterprises, such as backyard production systems where flocks forage outdoors. It recommended animal production should move to larger farms where surveillance is possible. Danielle Nierenberg, who researches this sector at Washington-based Worldwatch Institute, reports that this led Vietnam in April 2005 to impose a ban on live poultry markets and asking farms to convert to factory-style methods. This then became the method across.

This is when, the need of the hour was to regulate the industrial processes of growing chicken so that the virus does not breed and does not grow. The business needed to improve the genetic stock of birds and raise their immunity against diseases, just the way traditional backyard poultry farmers do. But instead of reforming the poultry industry the containment of the flu ended up promoting the very industry and its practices and destroyed the livelihood of small and marginal farmers.

THE INEVITABLE
It’s time the world planned in a prudent manner as zoonotic pathogens are ever-increasing and becoming unpredictable.

Studies also show how viruses are ever-adapting and ever-expanding via new susceptible hosts and additional transmission routes. S Abdul Rahman, executive director, Commonwealth Veterinary Association, says, unlike the old diseases like cholera and pneumonia, which we know how to deal with, these diseases are highly unpredictable. “With factors like climate change, zoonoses are emerging as the single biggest threat to human health and we are not prepared, as is evident from COVID-19 pandemic,” he says.

But the pathogens are honing their genome and preparing for their next mutation, and there is no doubt about it. A study published in Nature in October 2015, titled “Spillover and pandemic properties of zoonotic viruses with high host plasticity”, says pathogens, present in animals belonging to 10 biological orders, are 12 times more likely to transmit from human to human than those found in only one animal order. This is because the evolutionary process which equips a virus to rapidly adapt to new hosts also makes it capable of interspecies transmission. Many viruses, like Ebola, SARS-CoV and MERS-CoV, before jumping to humans were limited only to animals. When all conditions were met, they made the jump. Small wonder, most jumps have been made by RNA viruses.

The next step would be finding the right transmission route for easier, faster and effective dispersals.

So far, oral, aerosols, direct contact, fomite and vectors have been the five primary routes of disease transmission for zoonotic pathogens. These routes are crucial for determining their contagiousness, which is measured through reproduction rate (R0) or the number of secondary cases one case would produce in a susceptible population. In a way, they are responsible for taking a pathogen from the level of transmission to the level of epidemic spread. Cholera, a water-borne zoonotic bacterial disease, has a very high R0 of 9.5. By comparison, the R0 of Zika is 4.2; R0 of COVID-19 is 2.

A reasons for this low R0 of COVID-19 is the virus is still only hitching rides on
droplets, expelled from the body through coughs and sneezes. Since respiratory droplets are heavy, they cannot travel more than 1 metre. At least, that’s what WHO believes as of now. However, with studies finding that SARS-CoV-2 can travel up to 8 metres, several virologists seem to disagree that it is not airborne.

As the jury is still out on how SARS-CoV-2 travels, the fact remains that airborne transmission is the most lethal of all routes that can make a virus most contagious. Pandit says multiple factors determine if a pathogen is able to transmit with airborne droplets. First, an infectious person should be able to create droplets that are of appropriate size so that they can become aerosolised droplets with the help of particulate matter in the air. Then enough viable infectious dose has to remain in the air for a significant time so that either wind or air currents transmit it to other places where it can infect another susceptible person.

Once in the air, the success of the virus to remain infections depends both on the virus and the particle. Environmental factors like temperature, ultraviolet radiation, relative and absolute humidity, and air movement are important drivers influencing virus viability. Factors like temperature and humidity also impact
the size of droplets which in term affect the viability of virus.

So far, Q fever among animals like goat, sheep and cattle, caused by zoonotic bacteria Coxiella burnetii is believed to be the only disease that gets airborne dispersal. While it remains to be seen how long other pathogens can resist this temptation to go airborne, a study published in the journal Cell in 2014, found evidence of airborne transmission of avian influenza among ferrets. The researchers discovered that the ability to go airborne only took five substitutions in the virus.

DON’T PANIC, PREVENT

The threats zoonoses pose to the public health, global economy and food security and geopolitics is well established.

In what may sound like prophesy now, a 2014 study published by the Bank of American Merrill Lynch, after extrapolating historic examples, estimated that, “a severe and prolonged global pandemic could kill 180-360 million and hit global GDP by as much as 5-10 per cent in the first year, with most industry sectors adversely affected.”

Pandemics aside, between 1997 and 2009, the economic cost of six major zoonoses outbreaks was estimated to be about US $80 billion by the World Bank, report People, Pathogens and our Planet: The Economics of One Health, 2012. The cost would have been much higher had these outbreaks metamorphosed into pandemics. A 2011 report by the Organisation for Economic Co-operation and Development showed that pandemics are a prime global catastrophic threat. Potential losses resulting from a severe influenza pandemic, for instance, that leads to 71 million human fatalities would be $3 trillion, or 4.8 per cent of the global GDP. Zoonoses such as leptospirosis causes an estimated 1.03 million human infections and 60,000 deaths annually across 34 countries, for which there is adequate surveillance data, says Bethan Purse, ecologist at the UK Centre for Ecology and Hydrology. In 2000, WHO estimated that over a billion people are at the risk of scrub typhus and over one million cases occur annually. Since then, South-Asian countries with good surveillance have shown a rising incidence of scrub typhus.

Muliya says zoonoses kill the most number of people second only to non-communicable diseases. In terms of years lost due to premature death or to disability for living with the health condition or its consequences, they are second to none.

Unfortunately, though most of the major disease outbreaks have been caused by zoonotic viruses, Pandit says viral infections in general are difficult to treat. Very few antiviral drugs are effective against them, unlike antibiotics which we use against bacterial infections as they are broadly effective. Besides, says Pandit, since emerging viruses are
A RECENT study identifies domesticated species, primates and bats to be harbouring more zoonotic viruses than other species. Three mammalian orders (rodents, bats and primates) have together been implicated as hosts for the majority (75.8 per cent) of zoonotic viruses, while about 88 per cent of the mammal species have not been recorded with a zoonotic virus, says the study published in the Proceedings of the Royal Society Biological Sciences in April this year. Species listed as endangered on IUCN due to exploitation of habitat have also been found to share more viruses with humans than others. Yet more than any other species, bats have always been implicated in the emergence of most zoonotic diseases.

There’s a reason. Bats are hosts to more zoonotic viruses per species than any other species, particularly those causing Ebola, SARS and now COVID-19. “Bats have a very good immune system and for their size, they live long, for around 30 years. They live in large colonies and are long distance migrants, which make it easy for the virus to propagate,” says Sanath Krishna Muliya, project scientist at the Wildlife Institute of India, Dehradun. Many bat species are gregarious and some live in dense aggregations. Some Mexican free-tailed bat colonies can reach densities of 3,000 bats per square metre, in populations of up to a million individuals per roost. “Roosting sites can house diverse assemblages of multiple bat species. High intra- and interspecific contact rates can facilitate rapid transmission of pathogens and large population sizes could sustain acute-immunising infections,” says a study published in Proceedings of Royal Society Biological Sciences in April 2013. It says since bats are ancient mammals in evolutionary terms, they might have coevolved with pathogens to become immune to them.

Another study has focused on the feeding habit of bats as a reason for the spread of viruses. “Bats’ feeding habits are constrained by the aerodynamics of flight, so they can’t ingest huge amounts of food. Yet many bats are frugivorous— that is, they meet their energy requirements by ingesting fruits. But they merely chew them to extract the sugars and higher energy components, and then spit out the partially digested fruits. Other animal species ingest these fruit remnants and may consequently become infected with virus particles in residual bat saliva,” says a study published in Science in 2005.

PLAN AND PREPARE

In a way, the COVID-19 pandemic is exactly what experts had been warning for a long time. The signs were all there. Yet, no one could say when it was going to hit.

The potential for future pandemics is vast. As many as 1.7 million unidentified viruses of the type known to infect people are believed to still exist in mammals novel, developing vaccines or antibodies related treatments take a lot of time.

“Dealing with zoonoses is tricky because they keep mutating, forcing us to restart the effort to control it anew,” says Muliya. This also makes it difficult to promote a permanent cure.

Another reason for difficulty in treatment is that many zoonotic outbreaks are underreported. Zoonoses mostly infect people living under poverty with little access to healthcare. who’s report on neglected diseases also find correlation between living in proximity with livestock and the emergence of zoonoses. “Although one or more of these diseases can be found in almost every livestock-keeping community in the developing world, they are often simply forgotten,” acknowledges a 2015 report, "The control of neglected zoonotic diseases", prepared by who,

Because these diseases are neglected, proper efforts are not made to curb them. Consider Ebola and Zika. Before they caught the global attention with outbreaks in 2013 and 2015 , the diseases were for a long time considered tropical neglected diseases. But despite the attention, vaccines have not been developed for them so far. Ironically, before the outbreak, two promising candidates, the adenovirus-vectored (Ad5-GP) and the vesicular stomatitis virus-vectored (vsvag/ebovgp) were tested on nonhuman primates in 2003 and 2005. Although the trials produced positive immunogenicity and safety data, Ad5-GP was not investigated further.
and water birds. Any one of these could be the next "Disease X", potentially more disruptive and lethal than COVID-19. Since such pandemics are a direct consequence of human activity, we need to act now, when we are in the middle of a pandemic, caused by a zoonosis.

Rampant deforestation, uncontrolled expansion of agriculture, mining and infrastructure development as well as unregulated trade in wild animals have created a "perfect storm" for the spillover of diseases from wildlife to people. Unfortunately, communities who live on the fringes of forests and are most vulnerable to such infectious diseases pay the price of resulting outbreaks.

A recent article published on the website of Intergovernmental Science-Policy Platform on Biodiversity and Ecosystem Services (IPBES), an independent intergovernmental body, thus suggests to ensure that the actions being taken to reduce the impacts of the current pandemic are not themselves amplifying the risks of future outbreaks and crises. First, ensure the strengthening and enforcement of environmental regulations and deploy only those stimulus packages that offer incentives for more sustainable and nature-positive activities. Second, recognise the complex interconnections among the health of people, animals, plants and our shared environment. Third, fund health systems and incentivise behaviour change on the frontlines of pandemic risk. Third, fund health systems and incentivise behaviour change on the frontlines of pandemic risk. It may be politically expedient at this time to relax environmental standards and to prop up industries such as intensive agriculture and fossil-fuel-dependent energy sectors, but doing so without requiring urgent and fundamental change, essentially subsidises the emergence of future pandemics, say IPBES experts.

WHO has already floated a globally recognised response framework for dealing with zoonoses. According to WHO, “One Health” is an approach to designing and implementing programmes, policies, legislation and research in which multiple sectors communicate and work together to achieve better public health outcomes. Purse says, “We need to first understand how people come into contact with zoonotic infections as they use ecosystems for their livelihoods, what are their priorities and means of coping with diseases. Only by taking this joined up approach, can we understand what changes in policy, behaviour or systems might be required to reduce risks of infection and mitigate impacts.”

There is also a need to pump in more funds for neglected infectious diseases. Surveillance programmes of species like bats that are known sources of zoonotic pathogens need to be initiated. It can help in pandemic preparedness. Studies also need to conducted to understand viruses that are now being released as the Arctic thaws. COVID-19 pandemic has offered us a chance to prepare for much bigger threats that are yet to come. Let’s not waste it.