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Coronaviruses (CoVs), a subfamily of the *Coronaviridae* family, are positive strand RNA viruses with the largest genome of all known RNA viruses. Coronaviruses are a family of viruses that can cause illnesses such as the common cold, severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS). In 2019, a new coronavirus was identified as the cause of a disease outbreak that originated in China.

The virus is now known as the severe acute respiratory syndrome coronavirus 2 (SARS-CoV. The disease it causes is called coronavirus disease 2019 (COVID-19)

Coronaviruses are enveloped, single-stranded RNA viruses, which means that their genome consists of a strand of RNA (rather than DNA) and that each viral particle is wrapped in a protein “envelope.” Viruses all do basically the same thing: invade a cell and co-opt some of its components to make many copies of themselves, which then infect other cells. But RNA replication typically lacks the error-correction mechanisms cells employ when copying DNA, so RNA viruses make mistakes during replication. Coronaviruses have the longest genomes of any RNA virus—consisting of 30,000 letters, or bases—and the more material a pathogen copies, the more opportunity there is for mistakes. The upshot is that these viruses mutate very rapidly. Some of these mutations may confer new properties, such as the ability to infect new cell types—or even new species. A coronavirus particle consists of four structural proteins: the nucleocapsid, envelope, membrane and spike. The nucleocapsid forms the genetic core, encapsulated in a ball formed by the envelope and membrane proteins. The spike protein forms club-shaped protrusions that stick out all over the ball, resembling a crown or the sun’s corona—hence the name. These protrusions bind to receptors on host cells, determining the cell types—and thus the range of species—that the virus can infect. The major difference between coronaviruses that cause a cold and those that cause a severe illness is that the former primarily infect the upper respiratory tract (the nose and throat), whereas the latter thrive in the lower respiratory tract (the lungs) and can lead to pneumonia. The SARS virus binds to a receptor called ACE2, and MERS binds to one called DPP4—both are found in lung cells, among other places. Differences in the distribution of these receptors in tissues and organs may account for differences between the two diseases, such as the fact that MERS is deadlier than SARS and features more prominent gastrointestinal symptoms. MERS is not hugely infectious, however, which may also be a receptor-related trait. “DPP4 is expressed [highly] in the lower bronchi [airways leading into the lungs], so you have to have a large number of viruses coming in, because our airways are very good at filtering out pathogens,” says virologist Christine Tait-Burkard of the University of Edinburgh. “You need prolonged, intense exposure [to reach the lungs], which is why we see people who work closely with camels getting sick.”

Conversely, because pathogens can get in and out of the upper airways more readily, viruses that replicate there are more infectious. In addition, “the ability to replicate in different temperatures makes a big difference, because the upper respiratory tract is cooler,” Tait-Burkard says. “If the virus is more stable at those temperatures, it doesn’t go to the lower respiratory tract.” The lower airways are also a more biochemically and immunologically hostile environment, she adds. Analysis of 2019-nCoV strongly suggests the new virus, like SARS, uses ACE2 to gain entry to cells. This observation would fit with the fact that it appears, so far, to be less deadly than MERS (the current estimated mortality rate for the new coronavirus appears to be about 2 percent, but that figure may change as the outbreak unfolds and more cases are detected).

The picture quickly becomes complex, though, because viruses that use the same receptor can result in drastically different illnesses. One human coronavirus called NL63 binds to the same receptor as SARS but only causes upper respiratory infections, whereas SARS primarily infects the lower respiratory tract. “Why that is, we don’t know,” Perlman says. Another curiosity is that the ACE2 receptor is prevalent in the heart, but SARS does not infect heart cells. “That was a clear indication that other receptors, or co-receptors, are also involved,” says molecular biologist Burtram Fielding of the University of the Western Cape in South Africa. The virus binding to a receptor is only the first step in the cell entry process. When a virus binds to a host cell, they start morphing together, and other viral proteins may bind to other receptors. “For the efficiency of entry, it’s not just the one main receptor,” Fielding says. “There could be others as well.”