Smallpox, rabies, Spanish flu, polio, AIDS and Ebola are all viruses that evoke great fear, having demonstrated catastrophic impacts on humans, including wiping out entire populations. The evolution and emergence of viral infectious diseases is without bounds. Viruses parasitize every living organism, including bacteria (phages) and plants. Human pathogenic viruses frequently require a zoonotic host. The original animal viruses subsequently evolve to become very communicable among animals, including humans. Humans find themselves living knee deep in an animal cesspool of ever-evolving viral diseases. According to WHO, the 2016 top 10 list of emerging infectious diseases are: Crimean-Congo hemorrhagic fever [1], Ebola [2], MERS CoV [3], SARS [4], Lassa fever [5], Nipah virus [6], Rift Valley fever [7], Chikungunya [8], severe fever with thrombocytopenia syndrome [9], and Zika [10], all of which are viruses.

Viruses are nothing more than a molecular sack of chemicals, molecular machines really. They are not alive in any sense of the term. They do not demonstrate metabolic activity or generate any of the energy molecules of life. They are not self-sustaining, and they do not reproduce outside a live host organism. Viruses are coated with proteins that adhere to their target cells simply by their chemical recognition and affinity properties. Depending on the specifics of the virus, they enter their target cells and commandeer the host cell’s energy systems, signaling systems and replication systems to make a zillion daughter viruses, which are ultimately released back into the host and from there the environment to start the cycle anew. Viruses come in two flavors depending on their genetic material, DNA or RNA.

I will now describe the infectious process for HIV, a special RNA virus called a retrovirus. HIV is a bag of fat molecules surrounding a water core. The fat bag is studded with special viral proteins,
GP120 sitting atop a stalk of protein GP41 (looks like a mushroom). It may also contain some host proteins captured from its originating host cells. The next layers consist of spheres of proteins (matrix and capsid structures) that encase the HIV genome (9 genes total), composed of two copies of noncovalently linked, unspliced, positive-sense, single-stranded RNA folded into several conserved secondary structure elements. Also, contained within the capsid vessel are the key enzymes and ancillary proteins, regulatory and accessory element proteins necessary for successful conquest of the target cells by HIV virions. Infection commences with adhesion of the GP120 protein to a couple of key human target proteins on the cell surface of human immune cells. The process of sticking to the cell triggers the injection of the virion contents, that RNA and all those key pirate proteins into the host cell where they promptly take charge and initiate the viral replication process. The RNA is copied as a complementary single strand DNA and then the viral RNA is degraded. The new single strand viral DNA is now converted to a double DNA strand. This feat is all managed by the HIV enzyme (a chemical) called reverse transcriptase. DNA is spliced into the host DNA by another special HIV enzyme, integrase. With encouragement of certain host and the ancillary HIV proteins, the integrated HIV DNA is then transcribed into HIV RNA. The HIV RNA is translated to all the key HIV proteins that make up an infectious HIV virion. This motley crew of proteins, along with two copies of single strands of HIV RNA, are repackaged by self-directed adhesion processes into new virions which, with the aid of host factors, bud from the cell surface and are shed to go forth and contaminate more cells, as well as enter the environment and ultimately a new host. Each infected cell produces 1000 to 10,000 virions in its lifetime. Beginning to end, the entire process is an exercise in pure organic chemistry. There is not a single magical moment of life for the virus. HIV is just chemical recognition, molecular adhesion and chemical reactions processes between molecules.

Using HIV-1 sequences preserved in human biological samples, along with estimates of viral mutation rates, scientists calculate that the jump from chimpanzee to human probably happened during the late 19th or early 20th century, a time of rapid urbanization and colonization in equatorial Africa. By 2015, 36.7 million people were infected by HIV. The total number of HIV-related deaths is 35 million. It was only because of the drug inventions arising from the laboratories of the pharmaceutical industry that the absolute death sentence from a HIV infection was successfully derailed and HIV became a manageable chronic disease. HIV is a classic dangerous, emerging, infectious viral disease with its origins in the zoonotic pool. So, what could be next?

Any of the viruses listed on the WHO Top 10 list above could become a big problem. However, a particularly virulent strain of the highly communicable influenza virus is most likely to be our next really big problem. Remember, the great 1918 Spanish flu infected 500 million people across the world, including people in the remote Pacific islands and the Arctic. This pandemic resulted in the deaths of 50 to 100 million (three to five percent of the world’s population), making it one of the deadliest natural disasters in human history. Lesser serious influenza outbreaks have occurred since that time, including the Asian flu (1957), Hong Kong flu (1968) and the Russian flu (1977). No doubt a new influenza strain will be our next natural plague. That said, the hemorrhagic viruses are the scariest. These include Ebola, dengue, Rift Valley fever, Lassa fever, Marburg, Hantavirus,
Crimean-Congo virus and Machupo virus, among others. Hemorrhagic diseases cause massive vascular leakage, that is blood leaking out of the circulatory system into the tissues. As you might imagine, that is not a good thing and a bit gruesome to boot. I will describe a very scary emerging hemorrhagic virus that anyone of us could encounter and might indeed become a big-time danger to humans around the world. That virus is the rodent borne Guanarito virus, the cause of highly lethal Venezuelan hemorrhagic fever.

Venezuelan hemorrhagic fever is a zoonotic human illness first identified in 1989. The disease is most prevalent in several rural areas of central Venezuela and is caused by the Guanarito virus. The short-tailed cane mouse is the main host. The virus is spread mostly by inhalation of aerosolized droplets of saliva, respiratory secretions, urine, or blood from infected rodents. Person-to-person spread is also possible. It causes fever and malaise, followed by hemorrhagic manifestations and convulsions. Some presentations of the virus are also characterized by vascular damage, bleeding diathesis, fever, and multiple organ involvement. The disease is fatal in 30% of cases. It is speculated that demographic and ecological changes in the rural areas increased the frequency of contact between humans and infected rodents such that VHF emerged. This virus was so dangerous and facilely transmitted, that the initial speculation on the disease origins was as a biological warfare agent. This disease is in the earliest phase of an emerging zoonotic disease threat. As the Guanarito virus continues to incubate and mutate in its rodent host, then jumps hosts and migrates north, it is possible that decades from now Venezuelan hemorrhagic fever could become a major lethal threat in the Americas and beyond.

The pool of viral dangers is very deep. For this synopsis, it is not possible to even begin to delineate the vast number of old and new viruses incubating in animal hosts. These viruses are continually reproducing, mutating and evolving characteristics that will enable these viruses to jump species into humans where they can wreak havoc.

Next: How to tame viruses