Seasonality of SARS-CoV-2: Will COVID-19 go away on its own in warmer weather?

Myth 2: The "common cold" coronaviruses are seasonal, with little transmission in the summer, so SARS-CoV-2 will be too.

Predicting how a novel virus will behave based on how others behave is always speculative, but sometimes we have to do so when we have little else to go on. So the first problem with this myth is that we don't know whether those coronaviruses, which go by the evocative names like OC43, HKU1, 229E, and NL63, are good analogies for this virus. Still, it is worth considering the analogy especially to OC43 and HKU1, which are SARS-CoV2's closest relatives among the seasonal coronaviruses. The other reason this is a myth is that seasonal viruses that have been in the population for a long time (like OC43 and HKU1) behave differently from viruses that are newly introduced into the population.

To understand why, it helps to understand what we know about why many respiratory viruses are winter-seasonal in temperate regions like most of the USA. Scientists have identified four factors that contribute to this phenomenon. For some viruses, we have evidence for which factors are most important, for others, we have to extrapolate.

Factor 1: The environment.
In the winter, the outdoor air is colder, and the air is dryer usually both indoors and out. For influenza, it has been elegantly shown in the lab that absolute humidity — the quantity of water vapor in the air — strongly affects flu transmission, with drier conditions being more favorable. Subsequently it has been shown that epidemiological patterns are consistent with this lab data in the US and in Vietnam, among other study sites. Notably the Vietnam study looked at influenza-like illness, without distinguishing influenza from other types of pathogens. This hints that similar mechanisms may be at work for other respiratory viruses, but to my knowledge are no specific studies of the role of humidity for coronaviruses or other respiratory viruses besides flu. Also important: there may be some very humid conditions that also favor flu transmission, especially relevant in the tropics. Still it is safe to say that in temperate countries, dry cold air = favorable conditions for flu transmission. For coronaviruses, the relevance of this factor is unknown. A recent preprint on which I collaborated suggests that transmission is possible in many different climates, and points out that Singapore, for example, which lies nearly on the equator, has had significant transmission. This is one piece of evidence, but as my colleague Dr. Eli Perencevich has pointed out there are many differences between Singapore in February and a temperate zone in summer — different day length, ultraviolet radiation, and other factors that may be important for coronavirus – we simply don't know.

Factor 2: Human behavior.
In the winter humans spend more time indoors with less ventilation and less personal space than outdoors in the summer. In particular, schools are a site of much infectious disease transmission. School terms have been strongly identified as periods of higher transmission for respiratory viruses including those causing chicken pox, measles, and flu (here and here). The 2009 pandemic flu in the United States was very much decreased during the summer, and then came back rapidly in September.

The relevance of school terms is important but unknown for the SARS-CoV-2. Few children have been identified as cases. This may mean they do not get easily infected and don't do much transmitting. Or it may mean only that they don't get severe symptoms when they are infected, and transmit nonetheless. Or something in between. Understanding this is key if we want to know whether school closures can help control COVID-19 spread, as well as to anticipate how much summer vacation may help reduce spread.

Factor 3: The host's immune system.
It is possible that the condition of the average person's immune system is systematically worse in winter than summer. One hypothesis has focused on melatonin which has some immune effects and is modulated by the photoperiod, which varies seasonally. Another with more evidence is that vitamin D levels, which depend in part on ultraviolet light exposure (higher in summer) modulate our immune system in a positive way. The best evidence for the relevance of this hypothesis is that vitamin D supplementation reduces the incidence of acute respiratory infection, according to a meta-analysis of randomized trials. On the other hand, we found that this effect was unlikely to be a large factor in the variation in influenza incidence between summer and winter. This is a promising area for more study but at present its relevance seems uncertain.

Factor 4: Depletion of susceptible hosts.
Even without any seasonal variability, infectious disease epidemics rise exponentially, level off, and decline because when many individuals are susceptible, each case infects more than one new case ($R_{\text{eff}} > 1$). Then as the proportion of susceptible contacts declines, the epidemic peaks ($R_{\text{eff}} = 1$), and eventually declines ($R_{\text{eff}} < 1$). When there is some factor (like any or all of factors 1, 2, or 3) varying seasonally, and when new susceptibles appear in the population over time (for example through births) this process interacts with the seasonal factors to produce recurrent epidemics typically at the same time each year.

This leads us to the last point: **Even seasonal infections can happen "out of season" when they are new.**
New viruses have a temporary but important advantage – few or no individuals in the population are immune to them. Old viruses, which have been in the population for longer, operate on a thinner margin — most individuals are immune, and they have to make do with transmitting among the few who aren't. In simple terms, viruses that have been around for a long time can make a living — spread through the population — only when the conditions are the most favorable, in this case in winter.

The consequence is that new viruses — like pandemic influenza — can spread outside the normal season for their longer-established cousins. For example in 2009, the pandemic started in april-may (well outside of flu season), quieted in the summer (perhaps because of the importance of children in transmission of flu), and then rebounded in september-october, before the start of normal flu season. Seasonality does not constrain pandemic viruses the way it does old ones. This pattern is common for flu pandemics.