



Public Health Brief

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Was Grandma Right?

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Grandma's adage that you can [catch the flu](#) from the cold weather is not without merit. This association is undoubtedly evident for [COVID-19](#), whose incidence is [predicted](#) to continue to increase exponentially as [winter approaches](#).

Indeed, it is not the cold weather per se that causes these [respiratory diseases](#), but rather, pathogenic organisms. SARS-CoV-2 spreads [mainly through contact](#) (via droplets and aerosols), and longer-range and airborne transmission can occur via aerosols, especially in enclosed spaces with inadequate ventilation. But what is the mechanism by which cold temperatures facilitate the spread of respiratory infections?

Seasonal [variation](#) in the incidence of respiratory infections is well-established. Similar to the [1918 H1N1 influenza pandemic](#) (the "Spanish flu"), the deadliest in recent history, we are witnessing a rapidly accelerating increase in COVID-19 as we move into the cold season. Nonetheless, there is no firm consensus in the scientific community as to why this seasonal variability occurs. A number of plausible explanations have been proffered, particularly in the context of the common flu, which helped to inform public health measures against SARS-CoV-2.

SARS-CoV-2 shares key transmission characteristics with the influenza virus, albeit there are major differences. The epidemiological [triad](#), a seminal public health model for understanding disease causation and spread, posits that risk and [severity of infection](#) is a function of the interaction of the agent or causal organism, the host (person susceptible to infection), and the environment (setting or context in which infection occurs). Each of these factors can be influenced by other characteristics (such as temperature and humidity) which can modify infection dynamics.

Like the [influenza virus](#), SARS-CoV-2 is an enveloped virus. That is, it has an outer fatty or lipid membrane, which maintains the structural integrity of the virus allowing for the protection and



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transmission of its RNA to appropriate host cells, where it replicates, and initiates a new infection. Research on the influenza virus has shown that this outer fatty membrane solidifies into a gel at temperatures slightly above freezing and below. This thickened membrane or rubbery outer coat may allow the virus to survive in cooler temperatures and move from person to person.

So, it is biologically plausible that SARS-CoV-2 will adapt similarly to the cold weather, developing a thicker outer membrane, making the virus more hardy and hence potentially more available to spread. This increases the probability that a potential host or susceptible person will be exposed to the virus.

The primary environmental drivers of the winter spike in influenza cases are [temperature and humidity](#). This has also been [reported for COVID-19](#), with the virus surviving longer in cold, dry air, and low humidity, as in temperate winters. Low humidity also increases evaporation of respiratory viral droplets into smaller aerosol particles that can linger longer in the air, increasing the risk of airborne transmission in the winter. It is plausible that this can also increase exposure to a higher viral load during the winter.

Other [environmental factors](#) driving accelerated spread of respiratory infections [including COVID-19](#), are increased indoor dwelling and overcrowding, which are more likely to occur during the winter season. Low humidity during the winter enables the influenza virus to live longer indoors, and this together with spending more time indoors and in closer contact, significantly increases the risk of transmission and infection.

In general, a broad spectrum of host characteristics can moderate the risk and severity of respiratory infections, including biological, psychological, and sociological factors. But specific to the winter season [is vitamin D deficiency](#). Vitamin D is produced in the skin during exposure to sunlight, and has been shown to have a [protective effect](#) against respiratory tract infections by being positively correlated with immune health. The level of vitamin D may be depleted during this COVID-19 pandemic and the winter due to increased indoor activity and reduced exposure to sunlight. Another winter-related host factor is drying of the [nasal passage](#) due to low humidity, and this can increase susceptibility to infection by damaging the [nasal mucosa](#), making it easier for the virus to invade the body.

SARS-CoV-2 is a novel coronavirus, and our knowledge of its transmission dynamics is still evolving. These factors, along with a high level of transmissibility (including asymptomatic transmission) and virulence of SARS-CoV-2, create a perfect recipe for an even more explosive pandemic. The vast majority of the population remains unexposed and therefore susceptible to infection. This COVID-19 pandemic has the potential to parallel the 1918 flu pandemic if we fail to comply with the protective measures recommended by [public health authorities](#).

<http://alpinecountyca.gov/516/COVID-19>

<https://covid19.ca.gov/>

<https://www.cdc.gov/coronavirus/2019-nCoV/index.html>



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