Essential Oils and Coronaviruses

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On March 11, 2020, the World Health Organization (WHO) declared a pandemic caused by SARS-CoV-2 – a virus that was first seen in the Chinese city of Wuhan, and subsequently appeared in virtually every country in the world. The designation means that the epidemic of the virus and the disease it causes, COVID-19, has spread internationally, and through local transmission. Efforts to control the epidemic have failed. Pandemic declaration does not mean that the virus has become more infectious or more deadly, nor that individual risk has escalated (other than the risk of catching it). The classification of pandemic means that governments should shift from containment (epidemic control goal) to mitigation of effects (Fischer 2020).

The effects this virus has had on the lives of people around the world cannot be contested – from closed schools, to cancelled sports events and conferences to, in some cases, the quarantine of a whole country. This article will give you some background on the virus and its origins, the ways it impacts the human body, and what we know about essential oils and this type of virus.

What is a coronavirus?

Coronaviruses are so named because they possess spiked surface proteins, which give the virus surface a crown/corona appearance. In the biological tree, coronaviruses are part of the family Coronaviridae, in the order Nidovirales. Coronaviruses are enveloped viruses (influenza is too), which means they are covered by a membrane of cellular matter taken from their host cell, and coronavirus genetic content is organized as single stranded RNA. Bats are common reservoirs of coronaviruses, and most pass through an intermediate carrier species before humans are infected.

There are seven known coronaviruses that infect humans, including four that are responsible for about 15% of common colds. The four "cold" coronaviruses affect the upper respiratory tract and cause symptoms such as sore throat or runny nose. Three coronaviruses have caused major human disease: *SARS-CoV*, *MERS-CoV*, and *SARS-CoV-2* virus, or "the novel coronavirus" as it's commonly called. These three affect the lower respiratory tract – the lungs. It is unclear whether SARS-CoV, MERS-CoV, or SARS-CoV-2 could also affect the upper respiratory tract (Yang et al 2020).

A look back at SARS and MERS

SARS-CoV and MERS-CoV were the cause of two major outbreaks in the last two decades in South East Asia and the Middle East. (SARS stands for Severe Acute Respiratory Syndrome, while MERS is Middle Eastern Respiratory System). We can look at these past epidemics to understand how other coronaviruses behaved, and how we managed their spread.

SARS-CoV was first identified in the Guangdong province of China in 2002, and it spread to more than 30 countries. In the 2002/2003 outbreak, SARS-CoV had about a 10% fatality rate. The intermediate source was civet cats sold in a live meat market, which had been infected by horseshoe bats acting as SARS-CoV reservoirs (Tessini 2018, Luk et al 2019). SARS-CoV (the 2002/2003 virus) was successfully contained through adequate personal protective equipment for health workers, fever clinics that screened for abnormalities in white blood cell count (signaling infection) and performed chest images, SARS-designated hospital wings with 1000-bed capabilities, closure of poorly maintained medical facilities, and accurate information dissemination (Yang et al 2020). Some 8,000 people were infected in the outbreak, with less than 800 fatalities across 11 countries (Luk et al 2019).

The MERS-CoV outbreak occurred in 2012, and all cases were people linked to residence or travel through the Middle East, with >80% in Saudi Arabia. The median age of MERS-CoV infected people was 56yo, and it was more severe in elderly patients and those with pre-existing conditions. MERS-CoV is spread through direct contact, respiratory droplets, and aerosols. The intermediate carrier is likely dromedary camels, but the mechanism of transmission to humans is unknown. The fatality rate is about 35%, and in about 21% of infected people there are mild or no symptoms. The 2012 outbreak was contained early, with a total of 2,494 infected people, but a small number of cases still occur each year (WHO).

COVID-19 and the novel coronavirus

The novel coronavirus is officially called SARS-CoV-2, and the resulting disease is called COVID-19. The name was picked by the WHO and is an abbreviation for corona virus disease. COVID-19 cases were infected initially via contact with a wet market in Wuhan, China, in November and December 2019 (Tessini 2020). The closest sequence homology, or the closest genetic relative, for SARS-CoV-2 was initially a virus found in the Chinese chrysanthemum bat. Recently a virus found in pangolins was shown to be 99% sequence identical, so pangolins may be the intermediate species (Yang et al 2020). SARS-CoV-2 is spread via large respiratory droplets, but might spread via fecal-oral routes, surfaces infected by respiratory droplets, and aerosols. Incubation times have been estimated to be 1-14 days. Initial fatality estimates from China were 2.3%, while SARS was 10% and MERS was 35% (Tessini 2020). However, it is important to note that COVID-19 fatality rates are still unclear, as they fluctuate from country to country and currently range from 0.9% in South Korea to 7% in Italy, but fatality rates are heavily influenced by the amount of testing performed. With the focus currently on very ill patients, it is impossible to accurately determine how many people are infected because many may show mild symptoms but are not tested.

COVID-19 symptoms include fever, cough, and difficulty breathing. More severe cases can have lymphopenia (abnormally low level of lymphocytes in the blood) and chest imaging that looks like pneumonia (Tessini 2020). Critical patients may progress rapidly to acute respiratory distress syndrome (ARDS), septic shock, metabolic acidosis, coagulation dysfunction, and even death (Yang et al 2020). SARS-CoV-2 causes damage to the airway epithelial cells, which means that they are unable to clear the lungs of dirt or mucus, and this can lead to pneumonia. Patients also

show evidence of a “cytokine storm”, which are dramatic and damaging increases in levels of chemokine and cytokine proinflammatory molecules, often complicated further by pneumonia.

Treatments to address the “cytokine storm” seen in some patients are being investigated (Brüssow et al 2020). Some people with COVID-19 may have few to no symptoms, but it is unclear because unless someone has had contact with positive patients, they currently are not tested for SARS-CoV-2 in the United States. This approach will very likely lead to untested community-infected individuals spreading the virus further. The elderly and people with pre-existing conditions, such as heart disease or respiratory disease, are more likely to experience severe symptoms, and have a higher risk of dying.

Once the virus infects the respiratory system, the SARS-CoV-2 spike proteins bind host cell ACE2 receptors, just like SARS-CoV (Hoffman et al 2020), and the receptor-bound virus particle enters the host cell inside an endosome, much like influenza (Fung and Liu 2019). For a visual depiction of the process, scroll down through this New York Times graphic.

What about antiviral essential oils?

At the time of writing this article, there is no known cure for COVID-19 (although many possibilities are being tested), and there is currently no research into the effect essential oils may have on this particular virus. We are therefore not suggesting any treatment for SARS-CoV-2 infection with essential oils. Current standard of care for COVID-19 is supportive only.

Essential oils considered to be “antiviral” are not universal virus killers. Before we explain the existing research on “antiviral essential oils”, it is important to clarify the difference between virucidal and antiviral. “Antiviral” means that a compound inhibits the proliferation of a virus, while “virucidal” means a virus is destroyed or deactivated. In many instances, essential oils may be effective in killing one specific virus, but not another. Tea tree (*Melaleuca alternifolia*) essential oil inhibits the proliferation of influenza viruses inside cells (making it antiviral), but only modestly inhibits HSV-1 and HSV-2 (Garozzo et al 2009). Tea tree essential oil was not able to inhibit proliferation of the non-enveloped viruses poliovirus 1, adenovirus 2, echovirus 9, and Coxsackie B1 (Garozzo et al 2009). Much of the existing research on antiviral essential oils is on viruses that cause skin disease (herpes simplex I and II: HSV-1 and HSV-2), which has little relevance to viruses that cause respiratory tract infections.

Eucalyptus essential oils may be useful for managing minor symptoms

In addition, finding research for antiviral activity does not equate to finding an “essential oil recipe for the virus”. For example, an *in vitro/in vivo* study examining an avian coronavirus that causes upper respiratory infections in chickens and other small birds worldwide showed that a proprietary compound showed virucidal activity and was effective in controlling coronavirus-related bronchitis in chickens (Jackwood et al 2010). The manufacturers report the compound contains botanical oleoresins and essential oils, but do not reveal the formulation. Another *in vitro* study examining the same avian coronavirus found that ethanol extracted plant material could interfere with the coronavirus infection. The most successful were ethanol extractions (not

distilled essential oils) from peppermint (*Mentha piperita*), thyme (*Thymus vulgaris*), and a plant called showy tick-trefoil (*Desmodium canadense*) (Lelešius et al 2019). However, as mentioned above, this does NOT translate into any proven effect.

The closest to an applicable study is an *in vitro* study examining SARS-CoV (the 2002/2003 outbreak virus) and the effect of several essential oils. The authors report that a distilled oil extracted from *Laurus nobilis* berries was an effective virucidal against SARS-CoV (Loizzo et al 2008). The *L. nobilis* berries were sourced from a region in Lebanon, and the essential oil contained β-ocimene, 1,8-cineole, α-pinene, and β-pinene as the main constituents. This essential oil also contained eremanthin and dehydrocostus lactone as minor constituents at 3.65% and 7.57%, respectively (Liozzo et al 2008). These compounds are somewhat unusual in essential oils, but at least one *in vitro* study found that dehydrocostus lactone had activity against hepatitis B virus, an enveloped DNA virus (Chen et al 1995). However, laurel berry essential oil is not commercially available, and laurel leaf essential oil is not the same substance. It is possible that the oil used was a combination of essential oil and fatty oil made from Laurel berries by traditional methods, since only 56% of volatile compounds were identified (Tisserand and Young 2012, p322).

Research into coronaviruses may eventually address the antiviral efficacy of essential oils, but to date, *in vitro* data on the antiviral and virucidal activity of essential oils is limited, and *in vivo* human data is non-existent. As such, the specific mechanisms that coronaviruses use is poorly understood. SARS-CoV-2 is the most recent outbreak, which means even less is known about this coronavirus.

What can we learn about essential oils from research on other viruses?

The limited research available suggests that enveloped viruses are inactivated by some essential oils and their constituents, while non-enveloped viruses like Coxsackie B1 virus and human papilloma viruses (HPV), are not. Because there is **no clinical research** looking at coronaviruses and essential oils, using influenza as a proxy may provide some insights. Like influenza, coronaviruses are enveloped viruses and upon endosome-mediated cell entry, must be uncoated to enter the cytoplasm. You can think about this like a safe. In order to get to the money inside, you must first know the combination to the lock. Enveloped viruses must have that envelope removed for the inner part of the viral particle to enter the cytoplasm of the host cell, where it can reproduce. This mechanism (viral uncoating) has been well characterized for influenza, and you can read more about the essential oils and chemical constituents that affect influenza HERE. Coronaviruses affect the respiratory tract as does influenza, and also cause “cytokine storms”, followed by pneumonia, and sometimes death.

Suggestions in the Becker 2017 influenza article were based on research into the biology of influenza, and the interactions of essential oils and chemical constituents with multiple viral mechanisms of infection (Becker 2017). As discussed in Becker 2017, influenza viral uncoating occurs as the pH drops inside the endosome and the receptor-bound influenza protein undergoes

a conformational change, allowing the virus contents to enter the host cell cytoplasm. In the case of influenza, a number of essential oils and their constituents interfere with this process *in vitro*, but the significance for influenza patients is unlikely (Becker 2017). The mechanism for uncoating of coronaviruses is unclear, but it differs from the mechanism used by influenza due to the difference in viral protein content, and we cannot assume that the same compounds interfere with coronaviruses at this step. The proteins involved in influenza host cell exit (namely NA) are not found on SARS-CoV-2.

The host's innate immunity is provoked upon influenza infection, and a cascade of inflammatory pathways are initiated. In addition, a process called autophagy is triggered within the host cell. Autophagy is a mechanism that recycles cellular content. It can be triggered when a cell is stressed, or when a cell detects damaged proteins that need to be degraded (Jackson 2015). Autophagy is involved in cell death and interacts with inflammatory systems (Wang et al 2018). SARS-CoV (the 2002 outbreak) and MERS-CoV also interact with autophagy (Fung and Liu 2019).

Like influenza, SARS-CoV-2 infection often results in "cytokine storms", which are dramatic and damaging increases in levels of chemokine and cytokine proinflammatory molecules, often complicated further by pneumonia (Hayashi et al 2007, Li et al 2012, Wu et al 2012, Dai et al 2013, Brussow et al 2020). Becker 2017 suggestions to mitigate the "cytokine storm", inflammation, lung injury, and pneumonia were based on *in vitro* and *in vivo* research involving clove bud (*Syzygium aromaticum*) essential oil, eugenol, patchoulol, and *trans*-cinnamaldehyde (Hayashi et al 2007, Li et al 2012, Wu et al 2012, Dai et al 2013), but there are no studies in humans examining this. It's important to note that prevention and treatment are not the same thing. An essential oil that kills virus particles in the air or on the skin is a different concept to one that interferes with the actions of a virus once it has entered the body. **At this time, we cannot suggest any aromatherapy formulations to either prevent or treat SARS-CoV-2 infection.**

Symptom management and psychological wellbeing

Because there is no known cure for COVID-19, medical interventions focus on symptom management, and in severe cases necesitate respiratory support via ventilators. Under no circumstances should you try to address a severe case of the disease, and once shortness of breath occurs, you must seek medical help immediately. If only mild symptoms are present, you could use essential oils to assist in their alleviation – chest rubs containing pinene or cineole rich essential oils such as Eucalyptus (*Eucalyptus globulus*) or Rosemary (*Rosmarinus officinalis*), steam inhalation with the same oils, etc. You could also use inhalation to combat the inevitable stress and anxiety. The use of lavender (*Lavandula angustifolia*) essential oil with citrus essential oils, often sweet orange (*Citrus sinensis*) or lemon (*Citrus limon*), has been effective in reducing situational anxiety, as well as chronic anxiety (Lehrner et al 2005, Perry and Perry 2006, Goes et al 2012).

By far the best thing you can do to mitigate the pandemic is to follow the recommendations of the WHO and other public health authorities – social distancing and hand hygiene.

Summary

Viruses depend on the host cells that they infect in order to reproduce, and there are several stages to host cell viral infection. SARS-CoV-2 is an enveloped virus, like influenza A virus (IAV) and there is promising in vitro research on some essential oils and how they interfere with the mechanisms used by the IAV to infect cells. However, while some of these "antiviral" mechanisms may possibly translate to SARS-CoV-2, we don't yet know if the essential oils are clinically effective for influenza, nor do we have a clear idea on route of administration, dosage, safety, delivery systems and so on. And, we do know there are some fundamental differences between IAV and SARS-CoV-2. Therefore, making any assumptions at this point would be a massive stretch. While claims that essential oils will help protect you from, or treat COVID-19 are not based on evidence, this does not mean you cannot or should not make use of essential oils for respiratory and psychological support.

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