SARS-CoV-2 in saliva may be deactivated with fatty acids or emulsifiers

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Long-chain unsaturated fatty acids and medium-chain saturated fatty acids are effective in deactivating enveloped viruses. Short-chain (butyric, caproic, and caprylic) and long-chain saturated (palmitic and stearic) fatty acids had no or a very small antiviral effect. Monocaprin, the 1-monoglyceride of capric acid, and lauric acid caused a >1,000-fold reduction in the viral titer of herpes simplex virus in 1 min at a concentration of 20 mM. Unsaturated free fatty acids such as oleic acid, arachidonic acid and linoleic acid inactivate enveloped viruses such as herpes, influenza within minutes of contact at micromolar concentrations. Electronmicrographs of enveloped treated viruses indicate that the inactivation is associated with disintegration of the virus envelope. As SARS-CoV-2 is an enveloped virus, it may also be vulnerable to deactivation by these fatty acids. These fatty acids were effective especially in their monoglyceride form, which acts as emulsifiers. Emulsifiers belong to the chemical class of surfactants. The Japanese Ministry of Economy, Trade and Industry recently confirmed that SARS-CoV-2 was deactivated by several diluted surfactants. Emulsifiers and fatty acids are widely used as food additives and are harmless, even if swallowed. Persons affected with SARS-CoV-2 may be able to reduce the infectivity of their saliva or saliva-derived droplets by inclusion of those in their mouth. Saliva is used for PCR based tests to detect COVID-19. However, it can be difficult to collect samples from elderly people with low saliva production. Flavored foods and liquids can stimulate salivation. Therefore, if it is confirmed that emulsifiers and/or fatty acids deactivate SARS-CoV-2 in the saliva and do not adversely affect RNA stability; then there may be a benefit in their inclusion in the mouth at the time of specimen collection, both to stimulate salivation and to reduce the risk to the individual collecting samples. Moreover, the presence of emulsifiers and/or fatty acids in the mouth before or during speech, or dental treatment may reduce the infectivity of the saliva and saliva-derived droplets. If we assume that SARS-CoV-2 infects and replicates in the oral mucosa because angiotensin converting enzyme-2, the host receptor for target cell entry, is highly expressed on the epithelial cells of oral mucosa, then repeated introduction of emulsifiers and/or fatty acids in the mouth may prevent aggravation of the disease due to entry of the virus into the respiratory tract. In vitro and in vivo research to test this hypothesis are desired.

References


