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Taste and Smell Loss in COVID-19: Possible Mechanisms, Challenges and Clinical Implications

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Description

The novel coronavirus SARS-CoV-2 is a highly pathogenic, single-strand, positive-sense RNA virus responsible for the present outbreak of COVID-19. The virus emerged in Wuhan, China, at the end of 2019 and rapidly spread worldwide, causing a global pandemic [1]. Several reports demonstrate the high prevalence of smell and taste loss in COVID-19 positive patients [2,3]. Approximately 50% of COVID-19 patients experience smell or taste disturbances during the infection, in particular at early stages of the disease. Today, the loss of taste and/or smell is considered one of the most reliable symptoms of COVID-19 [4]. Although, the loss of smell and taste was found 3-4 times lower among the COVID-19 patients infected with Omicron strain compared to earlier strains of SARS-CoV-2 (eg: Wuhan, alpha, delta).

Many viruses, including coronaviruses, may cause transient loss or changes in olfactory function, primarily due to inflammatory responses [5]. While it is well known that coronaviruses can cause chemosensory dysfunction, the underlying pathophysiological mechanisms remain unknown and may be distinct for each chemosensory system. The SARS-CoV-2 RNA genome is covered by an envelope composed of spike proteins and a lipid membrane. The infection cycle is initiated by the attachment of the spike proteins of SARS-CoV-2 to Angiotensin I Converting Enzyme 2 (ACE2), a host cell membrane protein that serves as a receptor, and TMPRSS2 (transmembrane protease), which serves as a co-receptor for viral entry by endocytosis.

One possible explanation for SARS-CoV-2 induced smell and taste loss is that the virus infects cells through interactions between its spike protein and the ACE2 receptor on target cells. Cultured human taste (HBO) cells and human Olfactory Epithelial (hOE) cells containing ACE2 and/or TMPRSS2 are prone to SARS-CoV-2 infection [6]. Although ACE2 receptor express on the taste cell membrane, the virus probably does not cause taste loss through direct infection of these cells. Instead, taste buds might be damaged by inflammation caused by the infection. Alternatively, taste loss (ageusia) may result from taste nerve damage following central nervous infection by SARS-CoV-2. Another possible explanation for taste impairment is cytokine storms caused by SARS-CoV-2 in taste cells and surrounding tongue epithelial cells. A cytokine storm could make taste cells permissive to SARS-CoV-2.

Specific signaling events can cause a cytokine storm and lead to three forms of cell death signaling following a viral infection, including apoptosis, pyroptosis, and necroptosis. The cytokine-cytokine receptor interaction pathway has been identified as an important cross-talk pathway using a pathway-pathway interaction analysis for SARS-CoV-2 infection. ACE2 has been shown to be over expressed in the presence of IFN [7]. In a recent study, increased ACE2 relative expression was observed in SARS-CoV-2 infected olfactory and taste cells [5].

During SARS-CoV-2 infection, altered olfactory and taste functions were observed but the etiology remains unknown for both conditions. SARS-CoV-2infection of non-neuronal cell types leads to olfactory dysfunction in COVID-19 patients. Persistent loss of olfactory function is caused by the presence of SARS-CoV-2 transcripts in olfactory neuronal and supporting sustentacular cells. Independent studies have demonstrated elevated levels of inflammatory cytokines in the Olfactory Epithelium (OE) of infected patients. Inflammatory intermediates have been suggested to indirectly lower the expression of Odorant Receptor (OR) genes and of key genes of the OR signaling pathway, which could cause significant changes in odor perception. This work also shows that OR expression levels return to normal after cessation of the inflammatory insult [5-7].

Finally, chemosensory disorders causing health conditions including, but not limited, neurodegenerative diseases, upper respiratory infections (eg: Influenza, SARS-CoV-2), post-traumatic states, and normal aging have serious health and quality-of-life consequences for patients. However, the lack of awareness of taste or olfactory deficiency has drawn little attention from the public and the scientific community, though chemosensory dysfunction was thrust into the spotlight during the COVID-19 pandemic. Chemosensory dysfunctions have significant impacts on patients' quality of life, depression and anxiety, and cognition. Evaluation of the chemosensory system, specifically olfactory and taste function should be incorporated into routine physical exams by Primary Care Providers (PCP) and Ear, Nose and Throat (ENT) specialists. Furthermore, improved public health education and awareness of patients with chemosensory dysfunction may help primary care providers to identify those who may benefit from additional treatment and diagnosis and patients outcome.

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