

Mediators of chronic COVID-related smell and taste distortion

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The unprecedented COVID-19 pandemic caused by SARS-CoV-2 has now puzzled the biomedical research community both with its acute presentation and its long-term sequelae. Among the unique symptoms of COVID-19 are the abrupt and profound loss of smell and taste. While transient disruption of normal olfaction and taste is associated with many upper respiratory infections and chronic nasal inflammatory conditions, the sudden onset and degree of severity are key distinguishing symptoms of COVID-19. Importantly, up to 4% of patients who recover from COVID-19 have persistently distorted sense of smell and/or taste 6-12 months later.

Although current studies have identified mechanisms that might explain the acute loss of smell and taste due to SARS-CoV-2, the mechanistic determinants of persistent olfactory and gustatory dysfunction remain unclear. Through a registry of 214 patients with post-COVID dysosmia, we identified nasal dryness as an independent predictor of persistent smell disruption after COVID-19. In contrast, subjective symptoms indicative of nasal inflammation are not associated with post-COVID dysosmia. Furthermore, we find that ACE2, the entry receptor for SARS-COV2, is specifically enriched in the glandular/secretory cells in the nose that are responsible for secretion and/or transport of nasal lining fluid, a critical contributor to odorant transport. We propose to evaluate our >40 banked samples collected as part of an IRB-approved study of subjects with different degrees of post-COVID smell and taste disruption for transcriptional and compositional changes in the nasal mucosa as well as differences in inflammatory mediator, odorant binding and odorant biotransformation proteins.