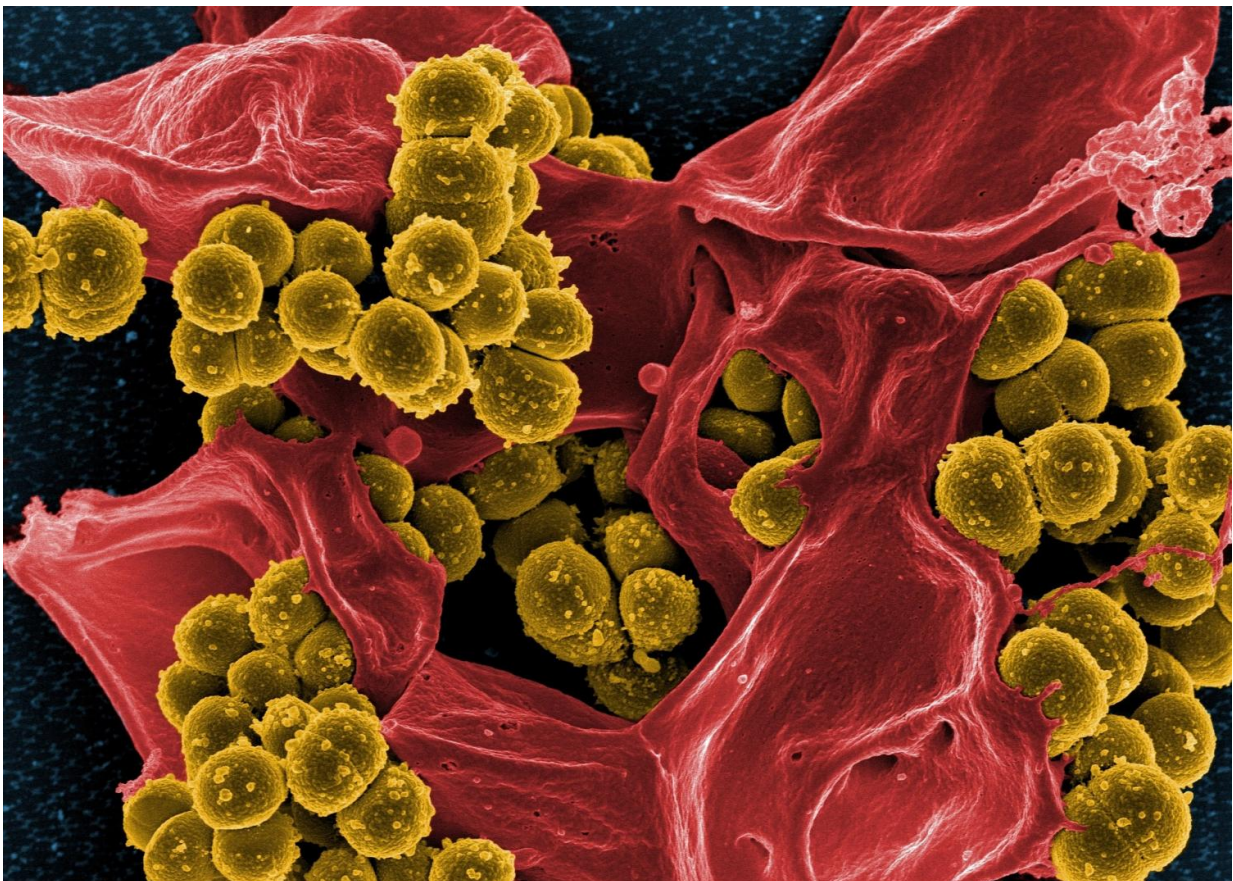


Bacteria found to produce compounds that activate sweet taste receptors in the sinuses allowing infections

September 7 2017, by Bob Yirka



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(Phys.org)—A team of researchers affiliated with several institutions in

the U.S. has found that a certain type of bacteria produces compounds that cause sweet taste receptors in the sinuses to activate—this in turn shuts off an immune response allowing the bacteria to thrive. In their paper published in the journal *Science Signaling*, the group describes their study of bacteria that cause chronic sinus infections and what they learned about them.

Researchers have found that as many as 10 percent of the people living in America suffer from chronic sinus infections, leading to suffering due to the pain they cause and burdensome [health care costs](#). Current treatments generally involve using antibacterial agents and/or drugs that reduce inflammation. But as the researchers note, such remedies are not always effective, and they do not prevent reoccurrence. Health scientists would prefer to find a way to prevent such infections rather than fighting them once established. To that end, research is ongoing regarding the nature of such infections and why some people are more susceptible to them than others. In this new effort, the researchers focused on interactions between [bacteria](#) and taste receptors, which are similar to those on the tongue, but which also exist on the surfaces of sinuses.

Prior research has shown that when [bitter taste receptors](#) in the sinus are activated (when sensing the presence of bacteria), the immune system kicks in and starts fighting the bacteria. In contrast, when sweet taste receptors are activated, the opposite occurs.

To learn more about what happens when Staphylococcus bacteria, frequently responsible for sinus infections, make their way into the sinuses, the group enlisted the assistance of patients with [chronic infections](#), taking nasal swabs from each. The mucus from the swabs was then cultured in a lab where the bacteria was tested to see which types of amino acids it produced. The team found two sweet amino acids—D-Leu and D-Phe. The team did the same with Pseudomonas aeruginosa and found that neither of the two acids were produced. This suggested

the two acids were used by the *Staphylococcus* bacteria to dampen an immune response. The team next grew human sinus cells in a petri dish and treated them with a chemical that stimulated the bitter [taste receptors](#)—they then added D-Leu and D-Phe. The team found that the acids prevented the receptors from sending out an alarm to instigate an immune response. They conclude by suggesting that *Staphylococcus* bacteria use the two sweet amino acids to dampen an [immune response](#), allowing them to thrive in the sinuses.

More information: Robert J. Lee et al. Bacterial d-amino acids suppress sinonasal innate immunity through sweet taste receptors in solitary chemosensory cells, *Science Signaling* (2017). [DOI: 10.1126/scisignal.aam7703](#)

Abstract

In the upper respiratory epithelium, bitter and sweet taste receptors present in solitary chemosensory cells influence antimicrobial innate immune defense responses. Whereas activation of bitter taste receptors (T2Rs) stimulates surrounding epithelial cells to release antimicrobial peptides, activation of the sweet taste receptor (T1R) in the same cells inhibits this response. This mechanism is thought to control the magnitude of antimicrobial peptide release based on the sugar content of airway surface liquid. We hypothesized that D-amino acids, which are produced by various bacteria and activate T1R in taste receptor cells in the mouth, may also activate T1R in the airway. We showed that both the T1R2 and T1R3 subunits of the sweet taste receptor (T1R2/3) were present in the same chemosensory cells of primary human sinonasal epithelial cultures. Respiratory isolates of *Staphylococcus* species, but not *Pseudomonas aeruginosa*, produced at least two D-amino acids that activate the sweet taste receptor. In addition to inhibiting *P. aeruginosa* biofilm formation, D-amino acids derived from *Staphylococcus* inhibited T2R-mediated signaling and defensin secretion in sinonasal cells by activating T1R2/3. D-Amino acid-mediated activation of

T1R2/3 also enhanced epithelial cell death during challenge with *Staphylococcus aureus* in the presence of the bitter receptor-activating compound denatonium benzoate. These data establish a potential mechanism for interkingdom signaling in the airway mediated by bacterial D-amino acids and the mammalian sweet taste receptor in airway chemosensory cells.

[Press release](#)

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