# Psychophysical Effects of Nasal and Oral Inflammation

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**Olfactory disorders are common in "nasal inflammation" even though the term is comprehensive and subsumes different kinds of entities which have to be differentiated. The most common cause of olfactory disorders are sinonasal disorders, which are defined as secondary smell disorders caused by diseases/pathologies in the nose/paranasal sinuses. According to the literature, sinonasal disorders represent—depending on the examined population—up to 72% of all olfactory disorders. In general, noninflammatory and inflammatory disorders are differentiated. Inflammatory disorders can be further classified into infectious or noninfectious disorders, both forms in which olfactory disorders can be present. For the clinician examining patients, the exact classification of the olfactory disorder is mandatory in order to choose appropriate treatment and counseling. Among the most common inflammatory disorders are acute rhinitis, allergic rhinitis, post-upper respiratory tract infection and chronic rhinosinusitis, which are discussed in detail. In contrast to nasal inflammation, only little is known about oral inflammation and its psychophysical effects on taste function. Taste disorders following oral inflammation are briefly discussed.**

*Key words:* **inflammation; psychophysical; smell; taste**

Nasal inflammation in all its variations—viral, bacterial, or allergic—is probably the most common cause of olfactory disorders in patients.[1–3] Onset and underlying pathophysiological mechanisms of the different forms of nasal inflammation differ; however, these conditions can not only reduce olfactory function but can also induce permanent anosmia. In contrast to nasal inflammations, oral inflammations causing taste disorders are less common even though they routinely develop in patients receiving radiotherapy. The most important inflammatory nasal diseases causing olfactory disorders are discussed in detail. A more precise analysis and differentiation of these disorders might contribute to counseling (Fig. 1) and giving correct advice to the patient about the prognosis of the existing disorder.

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## Acute Rhinitis

Acute rhinitis is a common, usually self-limiting primarily viral nasal inflammation. Among the most common viruses are rhinovirus, respiratory synticial virus, adenovirus, coronavirus, and influenza and parainfluenza viruses. Three pathophysiological phases are distinguished: a prodromal phase (white, pale mucosa), a catharactic phase (mucosa red, swollen, hypersecretion), and a viscuous phase (thickening of secretion, decrease of symptoms). Although acute rhinitis is among the most common causes of olfactory disorders,[4] there is little known about functional changes. In their experimentally induced common cold study, Akerlund *et al.*[5] observed an impaired olfactory function correlating with nasal congestion, suggesting a relationship between olfactory function and nasal congestion but not between olfactory function and nasal discharge. In another experiment, Hummel *et al.*[6] examined subjects suffering from a common cold using

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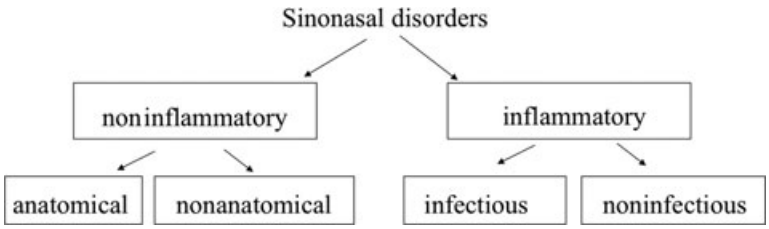


**Figure 1.** Depicts a guideline of how to classify sinonasal olfactory disorders.

the Sniffin Sticks Test and observed an increase of olfactory threshold and a decrease of N1 amplitudes in recordings of olfactory event-related potentials even when the nasal obstruction and discharge was controlled by the application of oxymetazoline.[7] These results imply that the observed olfactory changes in acute rhinitis may be independent of nasal congestion. Because the disease is self-limiting and olfactory function usually recovers, patients rarely seek medical advice concerning the smell disorder during acute rhinitis. When the acute rhinitis subsides and the olfactory disorder persists, it is called a "postviral olfactory disorder" or an olfactory disorder "post-upper respiratory tract infection" (post-URTI).

## Post-URTI Disorders

Post-URTI disorders are olfactory disorders following an acute upper respiratory tract disorder and are therefore usually considered as their own entity of olfactory disorders. Typically in the patients' history there is a close temporal connection to the infection/inflammation which is often recalled as "severe" or "more severe" than usual. The prevalence of post-URTI disorders is between 11% and 40%[1,8,9] and usually women are more affected than men.[10,11] The clinical examination is uneventful and psychophysical examination reveals hyposmia or anosmia.[12] Parosmia is a common feature.[13] Approximately one-third of the patients suffering from a post-URTI disorder experience spontaneous recovery within the first 2 years.[14] Histopathologically replacement of the olfactory epithelium by respiratory epithelium has been demonstrated, as well as a reduction in the number of olfactory receptors, but the number of studies is limited.[12]

## Allergic Rhinitis

Allergic rhinitis is clinically defined as a symptomatic disorder of the nose induced by an IgE-mediated inflammation after allergen exposure of the membranes lining the nose. Symptoms of rhinitis include rhinorrhea, nasal obstruction, nasal itching, and sneezing, which are reversible spontaneously or under treatment. It is subdivided into "intermittent" or "persistent" disease. The severity of allergic rhinitis can be classified as "mild" or "moderate-severe."[15] Although olfactory disorders are not part of the definition, they can be present—often temporarily—in up to 23% of the cases.[16] In patients with pure allergic rhinitis—seasonal or perennial—without any signs of accompanying sinusitis or nasal polyposis, olfactory disorders are less severe than in patients suffering from other forms of rhinitis.[17] A decrease in olfactory threshold after allergen exposure has been shown to be related to hypersecretion.[18] A clinical examination typically shows mucosal swelling, mostly in the lower turbinate, hypersecretion, and with ongoing disease and increasing disease duration, a reddening of the mucosa as a sign of the inflammatory processes in the mucosa. However, the visibility of the olfactory cleft and olfactory function do not correlate.[19] Topical steroids are used to treat allergic rhinitis and its effect on olfactory function remains contradictory. In studies testing olfactory function, two

studies described an improvement in either olfactory identification[20] or threshold[21] whereas another study found no improvement in olfactory function at all.[22] Because the olfactory disorder is mostly temporary, it is usually not the main symptom making the patient seek medical advice.

### Rhinosinusitis (Acute or Chronic, with or without Polyps)

Rhinosinusitis is defined as inflammation of the nose and the paranasal sinuses characterized by two or more of the following symptoms, one of which should be either nasal blockage/obstruction/congestion or nasal discharge (anterior/posterior nasal drip):

1. ± facial pain/pressure;
2. ± reduction or loss of smell;
3. and endoscopic signs of
   A. polyps, and/or
   B. mucopurulent discharge primarily from middle meatus, and/or
   C. edema/mucosal obstruction primarily in middle meatus, and/or computer tomogram changes;
   D. mucosal changes within ostiomeatal complex and/or sinuses.

This definition according to the European Position paper on Rhinosinusitis and Nasal Polyps[23] includes olfactory disorder as a symptom, which reveals that olfactory disorder is a very common symptom in this entity, especially in chronic rhinosinusitis (CRS), which is defined as a disorder lasting more than 12 consecutive weeks.[24] Although CRS is a very common disease, its exact etiology still remains unclear. Therefore, the exact pathophysiology of the accompanying olfactory disorder is difficult to determine. It is tempting to classify the olfactory disorder in CRS with polyps as conductive alone. This hypothesis was supported by the fact that (1) these deficits rapidly improve after oral steroids; (2) the olfactory epithelium was believed to be an "immunological privileged" site incapable of mounting a normal immune response to foreign proteins; and (3) biopsies revealed at least some normal appearing olfactory receptor neurons.[25] Recent evidence has shown, that at least partly, olfactory disorders are caused by an increase in olfactory receptor neuron apoptosis due to an extensive caspase-3 activity in CRS.[26] From the clinical point of view, it is important to remember that up to one-quarter of patients with CRS are unaware of their decreased olfactory ability.[27,28] Together with the fact that subjective disturbance and measured olfactory function in patients only show a moderate correlation,[29] olfactory testing is mandatory especially prior to surgical intervention. The clinical endoscopic picture is diverse; polyps, mucous discharge, mucosal reddening and swelling, and purulent discharge may be present. Once again, the visibility of the olfactory cleft from an endoscopic view and olfactory function do not correlate. Psychophysical test results reveal alternations between hyposmia and anosmia because olfactory disorder develops slowly and therefore only a portion of the affected patients are aware of the disorder.[30,31] So far no study in these patients has been able to document an association between olfactory test scores and intranasal airway access factors whether measured by rhinomanometry, acoustic rhinometry, or rhinoscopy. On the other hand, there seems to be growing evidence that the severity of histopathological changes in the olfactory mucosa, which can be seen in CRS, seems to be correlated with decreased olfactory function.[32]

## Oral Inflammation

Oral inflammation can influence taste function in several ways. Inflammation can influence the saliva itself, destruct the taste buds, damage neural pathways or cause systemic disturbances, which then alter taste function. Although numerous diseases are known to change taste function,[33] larger studies systematically

examining taste function and deficits in oral inflammatory diseases are lacking.

The most frequently examined disorders are diseases with reduced salivary function, such as Sjögrens disease or taste disorders following radiotherapy or radio-chemotherapy. In Sjögrens syndrome, a syndrome which is defined by lymphocytic infiltration, immune complex deposition and destruction of exocrine glands, patients usually complain of a dry mouth. In these patients, taste sensitivity is reduced; however, there is a poor correlation between salivary flow and taste function.[34] Radiotherapy alone or in combination with chemotherapy leads to an inflammation of the oral mucosa (mucositis) usually followed by xerostomia. During the acute phase of the inflammation, patients usually suffer from pain and the taste disorder is one minor problem among others. As the oral mucositis decreases the consecutive xerostomia becomes apparent. At this stage, usually 1–2 months after therapy, taste function is still reduced and the patients especially noticed a reduction of the perception of bitter and salty quality.[35] The disorder can persist up to 2 years.[35] Studies using confocal microscopy have shown that after radiotherapy, taste pores are covered with epithelia cells, which might be one of the reasons why taste function is reduced.[36]

## Conflicts of Interest

The author declares no conflicts of interest.

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