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ORIGINAL COMMUNICATION

Chemosensory decrease in different forms of olfactory dysfunction

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Abstract

The aim of this study is to investigate the effect of olfactory dysfunction (OD) on the two other chemical senses, namely gustation and the intranasal trigeminal system. Taste and trigeminal function were analyzed in a retrospective cross-sectional study of 178 participants with OD ($n=78$ posttraumatic, $n=42$ idiopathic, $n=27$ post-infectious and $n=31$ chronic rhinosinusitis (CRS) OD). All patients had been investigated for OD at our smell and taste outpatient clinic. Evaluation of olfaction was performed by means of the Sniffin' Sticks test (odor threshold, odor discrimination and odor identification), whereas gustatory function was assessed with the Taste Strips test and the intranasal trigeminal sensitivity by means of the lateralization task. The degree of olfactory impairment was found to depend on the cause of OD, but not on patients' age. Patients with posttraumatic OD showed lower olfactory function than patients with idiopathic, post-infectious and CRS OD ($p=0.01$). Gustatory and trigeminal sensitivity in turn depended on age rather than the cause of olfactory dysfunction. Partial correlations between olfactory, gustatory, and trigeminal scores, with age as covariate, were significant, showing a decrease of taste and trigeminal function proportional to the OD ($p<0.05$). The present data suggest that the three chemical senses are closely connected for humans underlining that in case of OD the remaining chemical senses (taste, trigeminal function) tend to decrease rather than compensate as this is seen for sensory loss in other modalities. This finding has direct clinical implications and importance when dealing with smell and taste disorders.

Keywords Olfactory · Gustatory · Trigeminal · Chemosensory interaction · Chemical senses

Introduction

Humans have three senses enabling them to decode their molecular environment. These are olfaction, taste, and intranasal trigeminal function. Related to their stereo-chemical functioning with molecule–receptor interaction, they are often summarized as the chemical senses. In daily life, but also on a cerebral level, these three senses are often simultaneously activated and share common brain areas for processing [28]. Flavor perception is mainly the result of the integration from these three different chemosensory channels. Perception of breathing is the result of olfactory and trigeminal inputs generated at the nasal level. Amongst the chemical senses, olfaction exerts a key function in oral or nasal perception as the system with the widest range of different possible perceptions, whereas taste and trigeminal inputs considerably modulate olfactory interpretation. Olfaction, taste and trigeminal function interact mutually in healthy individuals [3, 7, 25].

In contrast to other sensory systems, the chemical senses seem to interact differently. While sensory loss (e.g., blindness) is typically associated with crossmodal compensation (e.g., heightened auditory acuity) [31], reduced function in one of the chemical senses is typically associated with

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reduced sensitivity in the other chemosensory systems as well [21].

Olfactory dysfunction (OD; anosmia, hyposmia) affects up to 20% of the general population and is the most common form of impaired chemosensory function [20]. OD is therefore much more common than ageusia and absence of trigeminal perception, which are very rare conditions [15]. Most frequent causes relate to (1) sinonasal problems (2) upper respiratory tract infections (3) head trauma or (4) idiopathic, meaning that no underlying cause could be found [15].

A clinically relevant question is how OD influences the remaining chemical senses, taste and trigeminal function. Many patients with OD suffer from poor recognition of their condition and struggle with medico-legal and insurance issues [22]. A simplistic but still widespread concept is that patients with olfactory loss should be able to recognize taste or trigeminal stimuli as healthy subjects do. In the contrary case this is often interpreted as malingering by health professionals. Finally, general impairment in chemical perception that is not only restricted to OD makes these patients more prone to be a victim of hazardous events such as eating spoiled food or not detecting burnt cooking [27]. The current literature on consequences of OD on the other chemical senses has mostly focused on one, but not both remaining senses, and rarely distinguished between different causes of OD [12, 21, 32]. Therefore we set out to investigate this issue more closely by analyzing all three chemical functions in a group of patients with OD. As all four major forms of OD (namely posttraumatic, following a viral infection of the upper respiratory tract, linked to chronic rhinosinusitis, and idiopathic) show very different reductions of olfactory sensitivity, we aimed to see if taste and trigeminal function were affected proportionally in OD patients.

Materials and methods

This retrospective cross-sectional study was carried out in the smell and taste outpatient clinic of the department of Otorhinolaryngology of Geneva University Hospitals. The study was approved by the institutional ethics review board and conducted according to the Declaration of Helsinki on Biomedical Research Involving Human Subjects (IRB approval No: 13–161).

Participants

The chemosensory data of one hundred seventy-eight patients with olfactory dysfunction were included (82 women, mean age of 48.22 ± 17,41; 96 men, mean age of 45.51 ± 15.97) ranging from 15 to 84 years old. The etiology of olfactory dysfunction was posttraumatic ($n = 78$), idiopathic ($n = 42$), post-infectious ($n = 27$) and chronic rhinosinusitis ($n = 31$). Olfactory diagnosis was established according to the current recommendations for smell impairment [16]. All patients had full ENT examination including nasal endoscopy. Of the 178 included patients, full data set for all modalities was available for 124 (posttraumatic, $n = 67$; idiopathic, $n = 32$; post-infectious, $n = 16$; chronic rhinosinusitis, $n = 9$). For the remainder ($n = 54$) taste was not assessed.

Chemosensory testing

Olfactory function: all patients had been tested by means of a well established and validated orthonasal psychophysical smell test (Sniffin'Sticks, Burghart, Wedel, Germany) [26]. All patients underwent the extended test battery comprising olfactory threshold, discrimination, and identification. The composite score of these three tests, called TDI score (for threshold, discrimination, and identification), allows for classification into anosmia (TDI score below 16), hyposmia (TDI score between 16 and 30.5) or normosmia (TDI score above 30.5) [26]. A total of 44 patients were tested for both sides separately. For these cases, we computed an overall TDI score by the summing the results of the better score for each nostril, according to established procedures [1, 10, 26].

Gustatory function was assessed by means of the Taste Strips test (Burghart, Wedel, Germany), described previously [23]. The test is based on filter paper strips which are soaked with four tastes (sweet, sour, salty, and bitter) in four different concentrations. A total of 32 taste strips were randomly presented on the left and right side of the anterior third of the extended tongue. Participant had to identify the tastes with the help of a list of four descriptors (sweet, sour, salty, and bitter; forced choice). The summation of the correct answers was used to obtain an estimate of gustatory function (taste score) [23]; scores can range between 0 and 32.

Intranasal trigeminal function was assessed using the lateralization task, according to methods described previously [14]. Two identical squeeze bottles (total volume 250 ml) were presented simultaneously to each patient's nostrils. One bottle contained the target odor (30 ml of eucalyptol, Sigma-Aldrich, Switzerland); the other bottle contained 30 ml of odorless propylene glycol. Two air puffs were delivered by pressing the two bottles at the same time, with one entering one nostril, and the other entering the other nostril. The patient's task was to indicate to which nostril the stimulus had been presented. A total of 40 pseudo-randomized stimuli were applied at an interval of 30–40 s between each stimulation; patients were blindfolded to not have any visual cues. After each stimulation, participants were asked to identify the nostril to which the target had been presented (forced choice). Each correct answer was counted as point. The sum

of correct identifications was used to estimate trigeminal sensitivity; scores can range between 0 and 40. Since each nostril was stimulated 20 times, we were able to compute scores for individual nostrils as well.

Statistical analysis

Data analyses were carried out using SPSS 25.0 (SPSS inc., Chicago, IL, USA).

Participants were separated into four major OD groups: posttraumatic OD, idiopathic OD, post-infectious OD, and chronic rhinosinusitis-related OD. First, we performed a repeated-measures ANOVA on scores with *group* (4 levels; posttraumatic, idiopathic, post-infectious, CRS) and *sex* (2 levels; women, men) as within-subject factors and *modality* (3 levels; olfaction, gustation, trigeminal system) as within-subject factor and *age* as a covariate. To disentangle interactions, we subsequently performed three univariate ANOVA (one per modality), with *group* (4 levels; posttraumatic, idiopathic, post-infectious, CRS) as within-subject factor and *age* as a covariate. For significant effects we ran post-hoc *t* tests with Bonferroni–Holm corrections for multiple comparisons.

Second, we examined whether scores for the different modalities were correlated by computing Pearson's partial correlations between scores for olfactory, gustatory, and trigeminal function with age as covariate.

Third, in patients for whom results for individual nostrils were available, we examined whether scores for individual nostrils for olfactory function (TDI) / olfactory threshold and lateralisation test were correlated by computing Pearson's partial correlations with age as covariate. The alpha value was set to 0.05.

Results

Descriptive statistics: average scores and standard deviation for the three modalities are presented for the four groups separately in Table 1 and Fig. 1.

The repeated-measures ANOVA yielded significant main effects of *cause* [$F(3,115) = 3.955$; $p = 0.010$], *modality* [$F(1,907) = 27.802$; $p < 0.001$] and *age* [$F(1,115) = 10.823$; $p = 0.001$]. We further observed significant interactions of *modality* and *cause* [$F(5,722) = 2.917$; $p = 0.010$] and of *modality* and *age* [$F(1,907) = 5.073$; $p = 0.008$]. There was no effect of sex.

To disentangle the interaction between *modality* and *cause*, we carried out three separate univariate ANOVA, one for each modality. For the olfactory score, we observed a significant main effect of *cause* [$F(3,173) = 13.067$; $p < 0.001$], but no effect of *age*. Post-hoc comparisons indicated that patients from the posttraumatic group scored significantly lower (vs post-infectious $p < 0.001$; vs CRS: $p < 0.001$; vs idiopathic: $p = 0.012$) than the other three groups; we did not observe any other group difference. For the gustatory score we observed a significant effect of *age* [$F(1,119) = 4.598$; $p = 0.034$], but no effect of *cause*. Finally, for the trigeminal score, we observed a significant effect of *age* [$F(1,172) = 16.942$; $p < 0.001$] but no effect of *cause*.

Table 1 Descriptive statistics, average scores, and standard deviation for the three modalities for the four groups

	N	Mean	Std. deviation
TDI score			
Posttraumatic	78	14.37	7.33
Idiopathic	42	19.52	9.48
Post-infectious	27	23.21	7.86
CRS	31	24.35	10.66
Total	178	18.67	9.45
Lateralisation score			
Posttraumatic	78	28.59	8.37
Idiopathic	42	29.40	7.84
Post-infectious	27	31.15	5.63
CRS	30	32.20	6.26
Total	177	29.95	7.59
Taste strips score			
Posttraumatic	67	18.00	7.68
Idiopathic	32	19.75	6.98
Post-infectious	16	19.44	6.51
CRS	9	23.00	3.75
Total	124	18.98	7.18

We next computed partial correlations between olfactory, trigeminal and gustatory scores, with age as covariate. All three scores were significantly correlated to each other (Table 2).

Finally, we examined whether one-sided olfactory and trigeminal scores were correlated in those patients that were tested for each nostril separately (subset of 44 patients only). TDI (threshold) scores were correlated between left and right nostril (all $p < 0.001$), as were lateralization scores for left and right nostril ($p = 0.001$). However, TDI (threshold) scores and lateralization scores were not correlated for a given nostril.

Discussion

The main finding of the present study is that the degree of olfactory impairment in patients with acquired OD is correlated with both gustatory and trigeminal function. Thus, taste and intranasal trigeminal sensitivity seem to decrease proportionally to the degree of olfactory dysfunction regardless of the underlying cause (Fig. 1). A second finding is

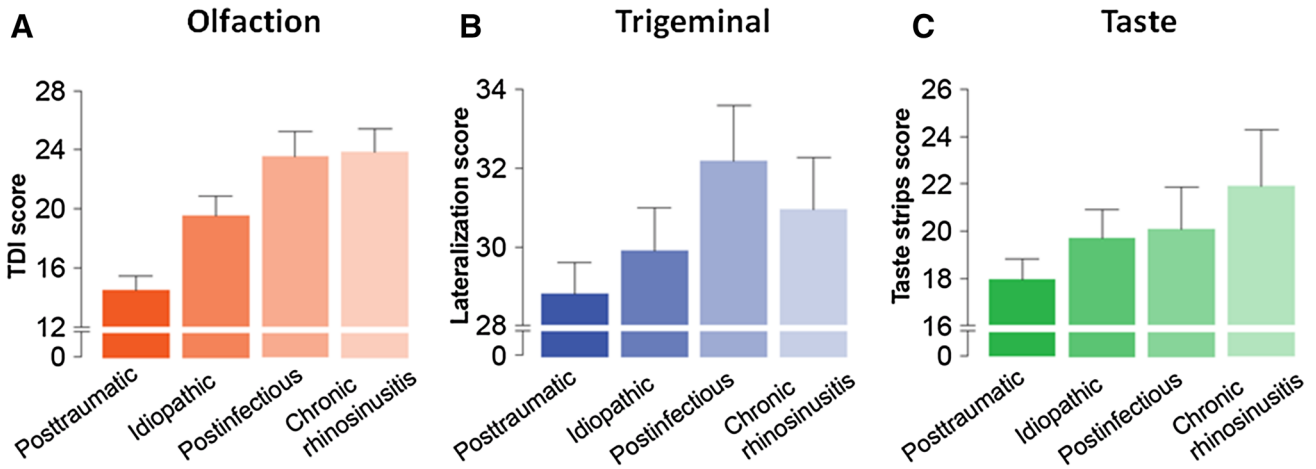

Fig. 1 Psychophysical test results (mean and standard deviation) according to the underlying OD causes for **a** olfaction **b** trigeminal, and **c** taste function: **a** mean values of TDI scores for posttraumatic OD, idiopathic OD, post-infectious OD and CRS OD. **b** Mean values of lateralisation scores for posttraumatic OD, idiopathic OD, post-infectious OD and CRS OD. **c** Mean values of taste scores for post-traumatic OD, idiopathic OD, post-infectious OD and CRS OD

Table 2 Correlation between olfactory, trigeminal, and gustatory scores, with age as covariate

	Gustatory score	Trigeminal score (lateralization score)
Olfactory score (TDI score)		
r	0.193	0.245
p	0.033	0.006
Gustatory score (taste strips score)		
r		0.357
p		< 0.001

that severity of olfactory dysfunction depends largely on the underlying cause. This has already been reported previously and confirmed by our data, showing that trauma produces a large olfactory impairment followed by idiopathic and to a lesser extent post-infectious and sinonasal-related origins [9]. Trigeminal and gustatory sensitivity, however, depend less on the cause of olfactory dysfunction, but rather on age. This is also in line with previous reports [14, 23].

The present data support and are in line with the majority of reports on acquired olfactory impairment and its effect on the other two chemical senses [12, 13, 21]. These studies investigated either trigeminal function or taste function separately in patients with olfactory dysfunction. The present study extends this by comparing all three modalities in a large sample size. Although crossmodal impairment is not found in all reports [32], it appears to be a rather constant finding when investigating patients with an acquired chemosensory dysfunction. This is in contrast to patients with congenital anosmia who exhibit unchanged trigeminal and gustatory function [11, 24].

Interactions between the chemical senses, and more specifically amplifying effects between smell, taste and trigeminal function in healthy individuals have been known for decades [2]. Different factors such as congruency and context [7, 17] determine whether input in one sense enhances or suppresses information in the other. Together with the present finding it is a further argument for the hypothesis that the chemical senses rarely function individually but share a lot of common processing [25].

The present results are cross-sectional and obtained in a clinical population with acquired dysfunction. Our findings therefore do not provide any elements for the location where the interaction takes place. Also, our data do not give insight to the mechanism leading to reduced gustatory and trigeminal sensitivity. Different hypotheses can be put forward: endings of the trigeminal nerve can be found in the olfactory mucosa [6]; in rodents some have been found to re-enter the central nervous system and to terminate within the olfactory bulb [29], giving rise to potential interaction between both systems [6]. Further, all three sensory systems provide input to the orbitofrontal cortex and the insula [28], which therefore may be alternative or additional sites of interaction. It is therefore still unclear whether the mutual interaction is due to peripheral or central effects or even both.

Our findings are particularly important for clinicians dealing with olfactory dysfunction. They should be aware that olfaction, taste and trigeminal function are connected and that olfactory dysfunction may affect the other chemosensory systems as well. This has significant consequences: patients who suffer from olfactory dysfunction may have a lower taste and trigeminal perception, which may impact

their quality of life. Further, some patients are evaluated in a legal context, e.g., for insurance claims. Considering the present data, reporting of reduced taste or trigeminal function in OD patients may no longer be a clear argument for suspecting the patient to malinger or aggravate. Even though the observed mutual decrease is rather small and may therefore be subclinical, it is significant, clearly present and overall related to the degree of olfactory impairment. Recent clinical findings of reduced taste sensitivity in posttraumatic [4] and idiopathic Parkinson's disease patients [5] seem to further confirm the present findings.

Reduced trigeminal sensitivity may also be relevant in our understanding how reduced olfactory function is connected to the perception of reduced nasal airflow [19]. In fact, menthol normally enhances the airflow perception leading to a feeling of unblocked nose, but olfactory dysfunction is associated with a decreased perception of coolness caused by menthol [30]. Further, decreased perception of intranasal airflow leading to a feeling of nasal obstruction, also called empty nose syndrome, is associated with decreased olfactory function [18]. Thus, crossmodal effects may be more frequently related to clinical complaints than previously thought.

The reasons of why chemical senses decrease mutually remains open. In contrast to the compensatory interaction found for vision, audition and touch, other mechanisms seem to take place amongst the chemical senses. This might be related to the close functional and anatomical relation between the senses. Structural impairment or reduced input to the orbitofrontal regions due to OD might impair the neuronal substrates necessary for taste and chemical trigeminal function. In case of trauma patients who exhibit the most marked dysfunction it might be argued that the trauma itself might have damaged the orbitofrontal cortex [8] or affected taste and trigeminal afferents.

A primary limitation of the study is the absence of a control group consisting of individuals with normal olfactory function, which is due to the retrospective cross-sectional study character. The second limitation is the absence of a group of patients suffering from neurodegenerative disorders like Parkinson's. This group as well was not included due to the retrospective cross-sectional study character. Previous studies have determined that patients with Parkinson's disease have a decrease in smell but not of trigeminal function [33]. Taste function has been reported to be decreased in patients with Parkinson's disease, suggesting that also in OD due to neurodegenerative disease chemosensory interaction may occur.

This study sheds light on common decrease in chemosensory sensitivity in different forms of olfactory dysfunction. It suggests that the degree of olfactory impairment influences the remaining chemical senses. Our findings have a clinical and legal impact and also raise questions about the neural correlates of chemosensory interactions. In future studies, the impact of chemosensory interaction and its decrease in disease state on quality of life of concerned patients needs to be addressed.

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Compliance with ethical standards

Conflicts of interest The authors declare that they have no conflict of interest.

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