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REVIEW

Impact of Fungiform *Papillae* Count on Taste Perception and Different Methods of Taste Assessment and their Clinical Applications

A comprehensive review

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أثر عَدّ الحليمات الكميئة على إدراك حاسَّةُ الذَّوق والطرق المختلفة لتقييم الذَّوق، وتطبيقاتها السريرية

مراجعة شاملة

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ABSTRACT: Fungiform *papillae* are raised lingual structures which contain taste buds and thus play an important role in taste perception. These structures vary in number due to their relative sensitivity to a range of systemic and local factors which affect the *dorsum* of the tongue. Taste sensation can be measured using both chemical and electrical methods; however, the number of fungiform *papillae* has a direct effect on chemogustometric and electrogustometric values during evaluation. This review provides a general overview of fungiform *papillae*, their quantification methods and the various factors which may affect these structures. In addition, numerous methods of recording taste sensation and their clinical applications are highlighted.

Keywords: Sensation; Taste; Taste Perception; Tongue; Taste Buds; Investigative Techniques.

الملخص: الحليمات الكمئية هي بنيات لسينية مرتفعة تحتوي على براعم الذوق، ولهذا فهي تؤدي دورا مهما في إدراك حاسَّةُ الذَّوق. وتتفاوت أعداد تلك البنيات نسبة للتفاوت في حساسيتها النسبية تجاه طيف من العوامل الجهازية والمحلية المؤثرة على ظهر اللسان. ويمكن قياس حاسَّةُ الذَّوق بطرق كيميائية وكهربائية. إلا أن لعَدّ الحليمات الكميئة أثرا مباشرا على قيم حاسَّةُ الذَّوق المقاسة كيميائيا، و كهربائيا، في غضون فترة التقييم. وتهدف هذه المراجعة إلى تقديم استعراض شامل للحليمات الكميئة، وطرق عدها، والعوامل المختلفة التي تؤثر على تركيبها. بالإضافة لذلك يتطرق المقال للطرق المختلفة التي يمكن بها قياس حاسة الذَّوق، واستعراض تطبيقاتها السريرية.

الكلمات المفتاحية: حس؛ ذَّوق؛ إدراك حاسَّةُ الذَّوق؛ السان؛ براعم الذوق؛ طرق الاستقصاء.

Fungiform *papillae* are mushroom-shaped structures located on the *dorsum* of the anterior two-thirds of the tongue.[1–3] Due to their rich capillary network, larger size and patchy distribution, fungiform *papillae* can be identified as reddish dots that contrast to the smaller and more numerous filiform *papillae*. According to cadaveric data, each individual has an estimated 200 fungiform *papillae* which are denser on the tip of the tongue in comparison to the middle region (29 versus 7–8 *papillae* per cm^2).[2,3] Each fungiform *papilla* carries between 0–20 taste buds, with an average of 2–4 buds; however, not all fungiform *papillae* contain taste buds and function as taste receptors at a given time. There are approximately 2,500 taste buds in the fungiform *papillae* of the anterior two-thirds of the tongue, although this has been reported to vary by approximately 18-fold between individuals.[1–3]

Based on their morphology, fungiform *papillae* are classified into four types that represent varying degrees of pathological severity.[4] Type 1 fungiform *papillae* are the healthiest and are egg-shaped or long and elliptical and devoid of surface thickness, while type 2 are slightly thicker. Type 3 are thick and have an irregular surface while type 4—which represent the most pathological state—are flat and have an atrophic surface.[4]

Most studies investigating taste function utilise fungiform *papillae* count as a tool to indicate either decreased or increased taste sensitivity.[5–7] To this end, fungiform *papillae* count has been correlated with various electrogustometric and chemogustometric thresholds. This review provides an overview of fungiform *papillae*, different methods for their quantification and the factors affecting these structures. In addition, various methods for recording taste sensation and their clinical applications are also discussed.

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Taste Receptors

The average human has a total of approximately 10,000 taste buds.[8] These are located in the *mucosa* of the *epiglottis*, palate and pharynx as well as in fungiform and circumvallate *papillae* on the tongue. Fungiform *papillae* are most numerous around the tip of the tongue, whereas circumvallate *papillae* are arranged in a 'V' pattern on the posterior third of the tongue immediately anterior to the *sulcus terminalis*.[8] The taste buds on the fungiform *papilla* are mostly embedded on the surface of the *papilla*. In contrast, the larger circumvallate *papillae* have approximately 100 taste buds located in the side walls.[8] The minute conical filiform *papillae* spread over the remaining tongue surface lack taste buds.

Taste buds are composed of ovoid bodies measuring 50–70 μm.[8] Each bud consists of basal cells as well as type 1, 2 and 3 cells. Type 1 and 2 cells (i.e. sustentacular cells) support type 3 cells (i.e. the main gustatory cells) and are connected with sensory nerve fibres.[8] Type 3 cells open up in the oral cavity via an opening called the taste pore which contain microvilli projecting from the taste cells. The necks of all three cell types are connected to each other and to the surrounding epithelial cells by tight junctions, so that only the microvilli are exposed to fluids in the oral cavity [Figure 1A].[9]

Each taste bud is innervated by 50 nerve fibres which connect up to five taste buds each.[8] The cells surrounding the taste buds give rise to basal cells which in turn differentiate into new taste receptor cells which replace the older ones every 10 days.[8] Taste buds degenerate and finally disappear when the sensory nerve supplying them is severed. However, if the nerve regenerates, the surrounding cells become organised into new taste buds; this is attributed to the influence of a chemical inductive from the redeveloping nerve fibre.[8]

Taste Pathway

The *chorda* tympani is a branch of the facial nerve which innervates the taste buds on the anterior two-thirds of the tongue, while the posterior third of the tongue and taste buds situated in other areas (i.e. the palate and *epiglottis*) are innervated via the glossopharyngeal and *vagus* nerves, respectively.[10] Taste sensation is carried to the gustatory area of the nucleus *tractus solitarius* in the *medulla oblongata* by slow-conducting myelinated nerve fibres. From the nucleus *tractus solitarius*, the neurons relay into the medial *lemniscus* of the *medulla oblongata*.[10]

The axons of these second-order neurons then transmit directly to the ventral posteromedial nucleus of the *thalamus* where they travel via thalamic radiation to the anterior part of the ipsilateral *insula* which facilitates conscious perception of taste and taste discrimination [Figure 1B].[10] However, in some individuals, all or some of these second-order neurons may cross over to the contralateral side and synapse at the *thalamus*, projecting to the contralateral cerebral *cortex*, while the remaining fibres continue to project to the ipsilateral cerebral *cortex*, thus resulting in bilateral representation.[11]

Taste Sensation

Previously, it was believed that different areas of the tongue were responsible for perceiving the five different categories of taste (sweet, salty, bitter, sour and *umami*).[12]

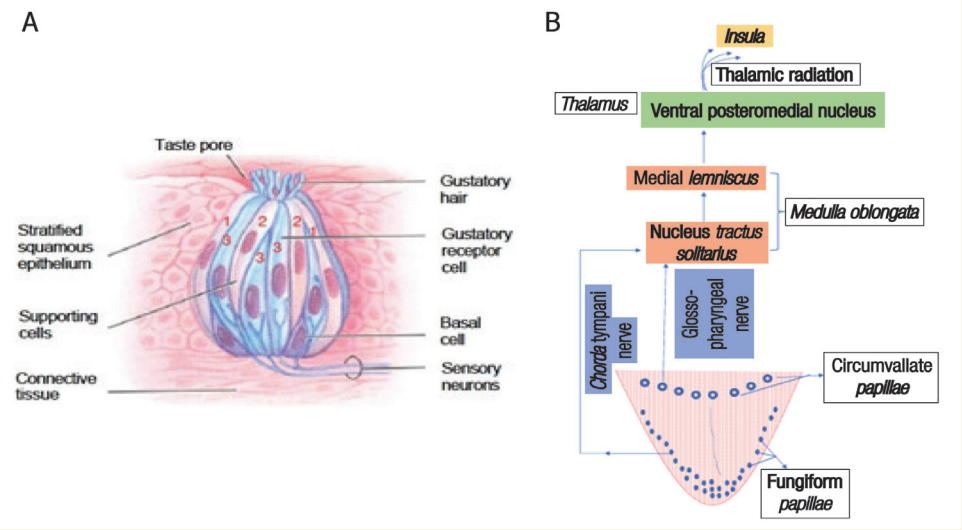

Figure 1: Annotated diagrams of (**A**) taste bud morphology and (**B**) the taste pathway.
Figure 1A was modified and reproduced with permission from Gowthamarajan et al.[9]

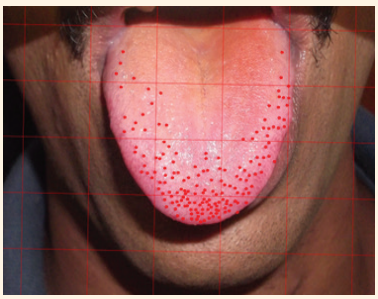

Figure 2: Image of fungiform *papillae* quantification using digital photography and computer software.
Reproduced with permission from Khan et al.[7]

However, it is now understood that the process of converting different taste *stimuli* into signals is not restricted to different zones of the tongue. Flavours are identified as a result of a backdrop of complex chemical mechanisms which involve ionic exchange through specific channels and secondary messenger activity.[8,13] This process is known as taste signal transduction.

SUPERTASTER PHENOMENON

Supertaster phenomenon refers to the existence of individuals who experience tastes with far greater intensity than normal, to the degree that they can perceive usually tasteless substances, such as phenylthiocarbamide (PTC) and propylthiouracil (PROP). This concept was first described by Fox in 1932 when it was noted that PTC was tasteless to some individuals, yet perceived as bitter by others.[14] While assessing the PTC taste response of over 2,500 subjects, Fox found that 65% of individuals recorded the taste as bitter, while 28% described it as tasteless and the remaining 6% described the taste in other ways.[14]

In recent years, research has revealed that this phenomenon is genetic in nature.[13,15] This has been attributed to varying genotypes of the *taste receptor 2* (*TAS2R*) *member 38* gene.[15–17] McMahon investigated supertaster phenomenon by assessing PROP taste status and *papillae* count among subjects who were able to appreciate unsweetened grapefruit juice; the results showed increased fungiform *papillae* density among supertasters, with non-supertasters having the lowest mean *papillae* densities.[17] However, more recent research has shown that no correlation exists between fungiform *papillae* density and differences in taste perception.[18,19]

Methods of Counting Fungiform *Papillae*

DIGITAL PHOTOGRAPHY

Nasri-Heir *et al.* utilised digital photography to assess fungiform *papillae* count by using a camera with a resolution of at least five megapixels to photograph the tongue alongside a millimetre slide rule.[6] Subsequently, using a computer, a grid was superimposed over the image and stretched to coincide with markings on the ruler in order to create 1 cm^2-sized boxes. The *papillae* within these boxes were then counted and averaged to arrive at a mean value.[6] This technique has also been supplemented with image analysis software [Figure 2].[7]

CONTACT ENDOSCOPY AND STAINING

Pavlos *et al.* evaluated fungiform *papillae* density using contact endoscopy and the application of a methylene blue stain.[20] First, a contact technique without staining was performed to aid imaging of the subepithelial capillaries; subsequently, the stain was applied to the epithelia and taste pores using a 1 cm^2-sized piece of filter paper placed around the tip of the tongue. The fungiform *papillae* were easily distinguishable as they were lightly stained in comparison to the darker filiform *papillae*.[20]

FOOD COLOURING DYE

Zhang *et al.* used food dye (brilliant blue FCF133) to stain the tip of the tongue.[21] The dye was transferred to the tongue using a 6-mm circular piece of filter paper. The stained area was then photographed using a digital camera and the photographs subjected to analysis using Adobe Photoshop® software (Adobe Inc., San Jose, California, USA).[21] An example of this technique is shown in Figure 3.[19]

DENVER *PAPILLAE* PROTOCOL

Nuessle *et al.* proposed a standardised method known as the Denver *papillae* protocol to define and prioritise the characteristics of fungiform *papillae*.[22] This method involved manually counting the number of *papillae* in a 10-mm circular section of the tongue stained with a blue-coloured dye. Using image analysis software and digital photography, the fungiform *papillae* were then characterised based on their shape, colour, size and height.[22] Individual fungiform *papilla* were rejected from the count if they were amorphous, stained blue in comparison to their surroundings, less than 0.5 mm in length or if they were lower in height compared to the surface of the tongue floor or the adjacent *papillae*.[22]

AUTOMATED DETECTION

Eldeghaidy *et al.* reported a method to automatically detect fungiform *papillae* on the *dorsum* of the tongue in collaboration with MATLAB® image analysis software (MathWorks, Natick, Massachusetts, USA).[18] The anterior 2 cm section of the *dorsum* of the tongue was automatically divided into eight regions by the software in which fungiform *papillae* were counted using various algorithms. However, one limitation of

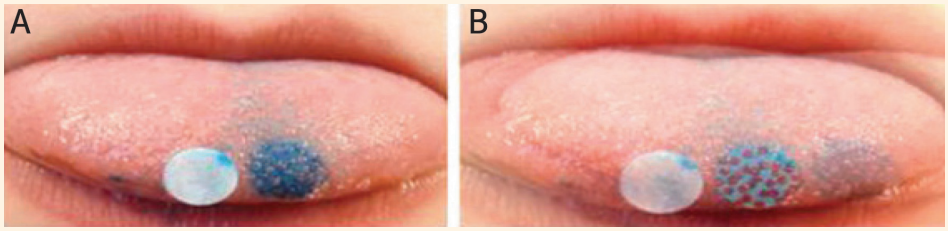

Figure 3: Photographs of tongue stained with brilliant blue FCF before (**A**) and after (**B**) fungiform *papillae* quantification.
Reproduced with permission from Jilani et al.[19]

this methodology is that the software may inaccurately assess the diameter of the *papillae* because the software considers all fungiform *papillae* to be exactly circular in shape.[18] Discrepancies have therefore been noted when the diameters of fungiform *papillae* are validated manually.[18,23]

Factors Affecting Fungiform *Papillae* Count

Nutrition and age are two major factors that affect the number of *papillae* present on the *dorsum* of the tongue.[24,25] Certain nutrients such as vitamin B12 and folate are important to maintain an optimum balance between cell regeneration and deterioration;[24] this is particularly important as the taste cells in the *papillae* have a high turnover rate.[8] In addition, vitamin A deficiency may result in keratinisation and the loss of integrity of such cells in the epithelium. Zinc deficiency can also have similar consequences.[24] Increasing age also slows down cellular regeneration, with 70% fewer taste buds recorded in individuals aged 70 years old compared to those aged 30 years.[24] This also explains why many older individuals often have decreased taste sensitivity.[25]

Due to their high metabolic activity, cells forming filiform and fungiform *papillae* are also sensitive to enzyme, circulation or nutrient disturbances which can lead to atrophy. During atrophy, filiform *papillae* are more vulnerable to such disturbances compared to fungiform *papillae*; moreover, following atrophy, fungiform *papillae* regenerate faster in comparison to filiform *papillae*.[24] Saito *et al*. reported that fungiform *papillae* atrophied among patients with normal pre-operative gustatory function after sectioning of the *chorda* tympani nerve; subsequently, the *papillae* recovered after regeneration or re-adaption of the nerve.[26] Spielman *et al*. biopsied fungiform *papillae* from the *dorsum* of the tongue and reported *papillae* regeneration at the same site after 40 days.[27]

Nasri-Heir *et al*. reported that the fungiform *papillae* count differed between patients with burning mouth syndrome and healthy controls (27.554 ± 2.122 *papillae* per cm^2 versus 31.575 ± 3.112 *papillae* per cm^2).[6] Zhang *et al*. reported an inverse correlation between the number of fungiform *papillae* and taste thresholds using sucrose among young male subjects.[21] Numerous other factors also affect the density of fungiform *papillae*, such as smoking.[20] Various other potential causes of lingual *papillae* loss are listed in Table 1.[28]

Methods of Evaluating Taste Sensation

The two primary methods of evaluating taste sensation are chemogustometry and electrogustometry.

Table 1: Factors potentially resulting in lingual *papillae* loss[28]

Nutritional deficiencies	Peripheral vascular diseases	Local factors	Therapeutic agents
• Iron-deficiency anaemia • Plummer-Vinson syndrome • Pernicious anaemia • Anaemia associated with parasitic infections (e.g. ascariasis and bilharziasis) • Tropical sprue or coeliac disease • Chronic alcoholism • Vitamin B deficiency (especially vitamin B2, B6, B12, folic acid and nicotinic acid)	• Diabetic angiopathy • Vasculitis in patients with SLE • Endarteritis obliterans • Syphilitic glossitis • Obliteration of the small blood vessels (e.g. in scleroderma or submucous fibrosis) • Localised vascular insufficiency in elderly patients	• Frictional irritation to the tip and lateral borders of the tongue • Atrophic *lichen planus* • Epidermolysis *bullosa* or ulceration which heals with scarring • Long-standing xerostomia	• Drugs that interfere with the growth and maturation of the epithelium (e.g. cyclosporine) • Drugs that induce candidosis (e.g. antibiotics and steroids) • Drugs that induce xerostomia (e.g. anticholinergic drugs and radiotherapy)

SLE = systemic lupus erythematosus.

CHEMOGUSTOMETRY

Chemogustometry involves the application of chemical solutions to the oral *mucosa*; subsequently, the degree to which any of the five types of taste presents itself is evaluated by equating the taste with that of a reference material.[29] Thus, taste detection thresholds are evaluated by asking the subject to taste a particular substance in various concentrations. This is done systematically by either increasing or decreasing the dilution of the substance. Water is used as a control during the process, with the subject ideally being able to discriminate between water and the diluted test solution.[29] In order to appreciate any differences in the taste intensity of the substance being tested, there should be at least a 30% difference in dilution between solutions.

Bitterness is detected by the action of the bitter substance on TAS2R receptors, a type of G protein-linked receptor.[16] Chemically, the threshold for bitterness is assessed using quinine hydrochloride at a dilution of 1 g in 2 L of water.[23] Saltiness is evaluated by assessing the action of the salty substance on epithelial sodium channel (ENaC) receptors using a diluted solution of sodium chloride. Taste transduction is thus induced by the influx of sodium ions in the ENaC receptors which facilitates the release of glutamate to depolarise neurons. Sourness, measured using diluted hydrochloric acid, also acts via the ENaC receptors which allow the inflow of protons and triggers neurons.[8,23]

Sweetness is assessed using a diluted sweet substance such as sucrose or artificial sweeteners like saccharin, which differ in taste due to their distinct chemical structures. Taste transduction occurs via another G protein receptor known as taste receptor 1. The final type of taste, *umami*, is savoury in nature. *Umami* taste is induced by the activation of metabotropic glutamate receptor 4 as a result of stimulants such as monosodium glutamate. Inosine monophosphate and guanosine monophosphate act as agonists during *umami* taste induction.[8,23]

Taste strips and disks

One method of chemogustometric evaluation involves presenting tastants to subjects in a clinical setting via soaked elongated strips or circular disks.[30,31] These taste strips or disks are usually made up of pullulan (α-1,4- and α-1,6-glucan) which is also combined with the polymer hydroxypropyl methylcellulose. Such strips or disks are dissolvable in the oral cavity and do not need to be retrieved afterwards.[30]

The three-drop method

Using this method, three drops of a chemical tasting solution are placed in the middle of the *dorsum* of the tongue, approximately 1.5 cm from the tip.[31] One drop contains the actual tasting solution and the other two are distilled water drops which act as a solvent. The testing process usually starts with the lowest concentration, with the solution increasing in concentration until the subject's tasting threshold is detected.[31]

Electroencephalography

Event-related potentials refer to the electrical responses evoked in the brain when an individual is presented with a *stimulus*. As such, after applying chemical gustatory *stimuli* to the tongue, electroencephalography can show cortical brain activity associated with the taste sensation, along with its topographical distribution.[32]

Advantages and disadvantages

Chemogustometry consists of using an array of chemical solutions in multiple concentrations to assess taste sensation. Some advantages of this method include the long shelf-life of the materials needed, the ease of administration, the rapidity of testing and the fact that this method allows for evaluation of each side of the tongue separately.[31] However, this method is qualitative in nature and more cumbersome in a clinical setting in comparison to electrogustometry.

ELECTROGUSTOMETRY

Electrogustometry quantifies taste and measures the threshold of taste sensation by passing a controlled current through the tongue using electrodes. As cathodal *stimuli* do not produce any significant recordable sensation, a weak anodal current is used.[33] The *stimulus* is a constant direct current of predefined amplitude and duration. The taste perceived during electrogustometry is described as sour-metallic or 'battery'-like and is attributed to the absorption of protons (or hydronium ions) released by the *stimulus*.[33]

In 1754, Sulzer first described the 'ferro-sulphate'-like metallic taste which occurs when two dissimilar metals come into contact with the tongue.[34] In 1955, Skouby invented the first electrogustometer based on taste thresholds determined by placing chemical solutions on the tongue.[35] Subsequently, the measurement of taste using an electric *stimulus* was reported by Krarup in 1958.[34] Over time, electrogustometers have evolved in terms of their design, electrode composition and size.[33,35] Electrogustometry is now a viable clinical tool to estimate taste function, though this method is yet to be commonly used.

When an electrode from an electrogustometer is placed on tongue, two types of sensations are induced—tingling and taste.[33] These two sensations are conducted via different nerves, with taste perceived as a visceral sensation by the *chorda* tympani nerve while the tingling is a mechanical sensation conducted by the lingual nerve. Therefore, electrogustometry aids in differentiating between the *chorda* tympani and lingual

nerves and is especially important in determining the integrity of the neural pathway.[6,33] However, since electrogustometric taste threshold measurements are subjective, uniformity must be maintained in the environment/set-up of the test and the way the subject is trained to respond to the sensations.

Advantages and disadvantages

Electrogustometry is a quick and quantitative tool to assess taste threshold, particularly among patients with taste disorders such as hemiageusia and ageusia.[32,36] Moreover, it allows for the evaluation of the most minute taste deficits, even in the absence of symptoms, and can be used to determine the topographical location of such deficits along the taste pathway and glossopharyngeal nerve. Furthermore, electrogustometry can also aid in determining patient prognosis.[36]

However, a major drawback of electrogustometry is that it is subjective and relies on feedback from the subject.[36] It also cannot be used to investigate or diagnose symptoms commonly associated with certain taste disorders, such as heterogeusia and spontaneous dysgeusia. Finally, this technique cannot be used for patients with artificial pacemakers as electrical *stimuli* from the electrodes may cause interference with electrical signals from the pacemaker.[36]

Clinical Applications

BURNING MOUTH SYNDROME

Braud *et al*. reported a significant association between electrogustometric values and pain intensity measured by visual analogue scale among patients with burning mouth syndrome, indicating a potent interaction between gustatory and nociceptive components among affected subjects.[37] Nasri-Heir *et al*. also noted significantly higher electrogustometric responses among patients with burning mouth syndrome in comparison to a normal control group.[6] The researchers concluded that burning mouth syndrome is a neurodegenerative phenomenon with decreased *chorda* tympani activity.[6]

CANCER

Ovesen *et al*. reported higher taste thresholds in subjects with small-cell lung, breast and ovarian cancer compared to controls with non-neoplastic disease.[38] Moreover, taste thresholds decreased among those patients who responded to chemotherapy, suggesting that malignant disease has an effect on taste sensation. These findings indicate that electrogustometry could be a useful diagnostic tool in neoplastic cancers.[38]

Epstein *et al*. found that patients with cancer developed taste disorders (i.e. dysgeusia) as a result of other factors apart from pathology, such as chemotherapy treatment.[39] This is because chemotherapy drugs are released into the saliva and adhere directly to the taste buds, causing altered taste perceptions and resulting in a metallic or chemical taste. As such, it is recommended that taste and olfactory electrogustometric evaluations be made mandatory for all patients undergoing cancer treatment.[39]

NEUROLOGICAL DISEASES

Dzaman *et al*. reported that 13 out of 35 subjects with nasal polyps had increased taste and olfactory thresholds as assessed by electrogustometry compared to controls.[40] Deeb *et al*. reported a deficit in electrogustometric thresholds among subjects with Parkinson's disease, indicative of disease severity.[41]

POSTOPERATIVE PATIENTS

Doty *et al*. assessed the impact of factors such as age and gender on taste perception among individuals undergoing *chorda* tympani nerve resection.[42,43] Taste assessment was done in different regions of the tongue using filter paper soaked in tastants such as sucrose, sodium chloride and caffeine; in addition, the patients were subjected to electrical *stimuli* via electrogustometry. The researchers reported a deterioration in taste sensitivity commencing in middle age and progressively reducing after 50 years of age.[42,43] This decline in taste sensitivity occurred for all *stimuli* at the anterior part of the tongue, with *chorda* tympani nerve resection resulting in taste deficits on the same side as well as the middle portion of the tongue.[42,43]

Boucher *et al*. investigated taste defects by electrogustometry in patients with severed afferent connections caused by dental treatment.[44] Higher electrogustometric thresholds were recorded in subjects with more than seven deafferented teeth compared to those with fewer deafferented teeth, with a significant direct correlation between electrogustometric thresholds and the number of deafferented teeth, regardless of age.[44] Similarly, Michael *et al*. found a greater prevalence of electrogustometric taste changes among patients following middle-ear surgery; this was ascribed to damage caused by the distention and, to a lesser degree, severance of the *chorda* tympani nerve.[45]

SMOKING

Depressed or altered taste sensation has been reported among chronic smokers.[46,47] Smoke from burning tobacco includes a variety of irritants, oncogenic particles such as tar and lead, as well as other poisonous substances such as carbon monoxide and nicotine. These not only topically affect taste receptors cells and impede the normal mechanism of taste conduction

but also affect the neurological transmission of taste sensations.[46]

The effects of tobacco on taste thresholds depend upon the individual's susceptibility, the quantity and frequency of use and the age of the individual when they started smoking. Using a modified form of electrogustometry, Khan *et al*. reported significantly lower fungiform *papillae* counts and greater electrical taste thresholds in smokers compared to non-smokers.[7] Moreover, in a follow-up study of smokers before and after quitting, Chéruel *et al*. found that smoking cessation lead to a recovery in taste sensitivity.[46] However, the time required to regain taste functionality depended on the susceptibility of the region of the tongue that had been affected. The researchers advocated for the use of electrogustometry as a method of motivating chronic smokers to stop smoking.[46]

Yekta *et al*. studied somatosensory function in the *mucosa* of the tongue.[47] Subjects were tested bilaterally in tongue regions innervated by the lingual nerves according to sensory heat, pain and mechanical detection thresholds. Increased heat thresholds were reported in smokers in comparison to non-smokers; this was attributed to damage caused by smoking to the myelinated Aδ- and C-fibres of the tongue.[47]

Conclusion

Fungiform *papillae* density offers valuable information regarding an individual's taste perception and taste sensation thresholds, with both chemical and electrical tools available for quantification purposes. However, as chemogustometry is a mostly qualitative method of determining taste sensitivity and requires a complex array of chemical solutions, it can be cumbersome in a clinical setting. In contrast, electrogustometry is a quick and quantitative tool and has a wide range of clinical applications, including for patients with taste disorders, burning mouth syndrome and neoplastic cancers as well as for smoking cessation purposes.

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