

Altered taste perception in Type 2 Diabetes Mellitus: A case-control study on taste thresholds and glycemic control

Julika De ^a, Hina Handa ^{a,*}, Gaurav Arya ^a, Sanskriti Agrawal ^a, Anirban Das ^a, Swagata Saha ^b

^a People's (Deemed to be University), People's Dental Academy, Department of Oral Medicine and Radiology, Bhanpur, Bhopal, 462037, Madhya Pradesh, India

^b Department of Pedodontics & Preventive Dentistry, Dr. D. Y. Patil Dental College & Hospital, Dr. D. Y. Patil Vidyapeeth (Deemed to be University), Pune, Maharashtra, India

ARTICLE INFO

Keywords:

Type 2 diabetes mellitus
Taste perception
Taste impairment
Gustatory function
HbA1c
Glycaemic control
Whole-mouth threshold test
Spatial taste test
Sweet taste
Sour taste
Bitter taste
Dietary counselling
Diabetic neuropathy

ABSTRACT

Background and objectives: Diabetes Mellitus (DM) is associated with altered taste perception, often leading to cravings for carbohydrate-rich foods. Although taste dysfunction has been linked to diabetic neuropathy, this association is debatable. Few studies have evaluated taste thresholds in individuals with diabetes, necessitating further investigations. This study aimed to assess gustatory function in patients with Type 2 Diabetes Mellitus (T2DM).

Materials and methods: A total of 138 participants were included in the study: 46 with controlled T2DM, 46 with uncontrolled T2DM, and 46 healthy controls. Taste perception was assessed using whole-mouth threshold and spatial tests. Data were analysed using the Kruskal-Wallis test, followed by Dunn-Bonferroni post hoc tests for pairwise comparisons. The significance level (p) was set at 0.05 for all statistical analyses.

Results: HbA1c levels were highest in the non-controlled diabetes group (11.22%), followed by the controlled DM group (7.43%) and the control group (5.08%) ($p < 0.001$). Whole-mouth threshold and spatial taste tests showed significant taste impairment in patients with diabetes, worsening with poor glycaemic control ($p < 0.001$), particularly for sweet, sour, and bitter tastes. Salt perception was unaffected ($p = 0.56$).

Conclusion: Individuals with diabetes exhibit taste dysfunction, particularly for sweet, sour, and bitter tastes. Blunted sweet perception may lead to increased sugar intake, worsening hyperglycaemia. These findings highlight the need for dietary counselling to mitigate the impact of taste impairment on glycaemic control.

1. Introduction

Gustation, or taste perception, is a critical chemosensory function essential for guiding dietary behaviour, ensuring optimal nutrient intake, and maintaining overall health. The human ability to detect and discriminate among five basic taste modalities—sweet, sour, salty, bitter, and umami—facilitates appropriate food choices and aids in the avoidance of potentially harmful substances. This sensory function is underpinned by a complex physiological system comprising three primary components: tastant molecules that stimulate sensory responses, specialised taste receptor cells located within taste buds, and an aqueous environment provided by saliva, which acts as a solvent and carrier, enhancing the interaction between tastants and their respective receptors. Saliva also modulates taste perception through its enzymatic

and biochemical composition, influencing both sensitivity and adaptation to flavours.¹

The initiation of taste sensation begins when tastants reach threshold concentrations and activate specific receptors on the gustatory cells. This interaction generates electrical signals that travel via afferent gustatory nerves to higher-order brain regions, where taste perception is processed. Disruption at any point in this cascade can lead to taste dysfunction, which may significantly affect food preferences, nutritional intake and metabolic health. A variety of factors—including aging, smoking, obesity, chronic alcohol intake, renal impairment, and exposure to cancer therapies—are known to adversely impact gustatory function.^{2,3}

Among systemic conditions, diabetes mellitus (DM), a chronic metabolic disorder characterised by sustained hyperglycaemia, has been

* Corresponding author.

E-mail addresses: dr.julika.de@gmail.com (J. De), hinahanda06@gmail.com (H. Handa), dr.gauravarya@gmail.com (G. Arya), sanskriti6agrawal@gmail.com (S. Agrawal), anirbandas52@gmail.com (A. Das), swagata.saha@dpu.edu.in (S. Saha).

<https://doi.org/10.1016/j.jobcr.2025.11.015>

Received 7 April 2025; Received in revised form 18 November 2025; Accepted 24 November 2025

Available online 30 November 2025

2212-4268/© 2025 The Authors. Published by Elsevier B.V. on behalf of Craniofacial Research Foundation. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

increasingly associated with alterations in taste perception. Both Type 1 and Type 2 DM (T1DM and T2DM) are associated with taste disturbances, which may manifest as decreased sensitivity or altered thresholds, particularly for sweet and salty tastes. These disturbances may contribute to maladaptive eating behaviours, such as increased carbohydrate cravings, which further impair glycaemic control.^{4,5} However, the pathophysiological mechanisms underlying taste dysfunction in diabetes are multifactorial and have not been fully elucidated yet. Importantly, taste perception may also be influenced by local oro-dental factors such as periodontal disease,⁶ salivary flow alterations,⁷ oral mucosal changes,⁸ dental deposits,⁹ and infections,¹⁰ all of which are prevalent in diabetic individuals.¹¹ This creates a diagnostic dilemma in determining whether altered taste perception arises primarily from systemic diabetic pathology or from local oral conditions. Highlighting this distinction is essential for understanding the true origin of taste disturbances and for developing targeted clinical interventions.

Proposed mechanisms include peripheral neuropathy affecting gustatory nerves, microvascular damage reducing blood supply to taste buds, direct injury to taste receptor cells, and central dysregulation of taste processing.¹² Emerging evidence also points to defective glucose sensing in gustatory cells, paralleling the dysfunction seen in pancreatic β -cells, as a contributor to altered taste responses in patients with diabetes.¹³ Moreover, pharmacological agents commonly prescribed to individuals with diabetes and the elderly (e.g. ACE inhibitors, anticonvulsants, and chemotherapeutic drugs) have also been implicated in the induction or exacerbation of taste disturbances.¹⁴

Additionally, some studies have reported a correlation between elevated blood glucose levels and blunted taste responses, suggesting a bidirectional relationship in which poor glycaemic control may both result from and contribute to impaired taste function. However, the literature remains inconsistent, with varying reports on the nature and extent of these alterations.^{15,16} This underscores the need for more focused studies to investigate the quantitative aspects of gustatory thresholds in diabetic populations.

Clinical evaluation of taste function employs both objective and subjective methods, including whole-mouth threshold testing (assessing the ability to detect different concentrations of each basic taste) and spatial or localised taste testing (applying tastants to specific areas of the tongue to identify regional deficits).^{17,18} These assessments offer valuable insights into the patterns and severity of gustatory dysfunction in patients with DM.

Despite the clinical significance of taste alterations in diabetes, limited research has systematically explored threshold-based taste changes in individuals with T2DM, particularly in comparing controlled and uncontrolled glycaemic states. The present study aimed to bridge this gap by evaluating gustatory function in individuals with controlled and uncontrolled Type 2 Diabetes Mellitus using both whole-mouth and spatial taste threshold assessments, in comparison with healthy controls. The findings aim to shed light on the nature and extent of taste impairments in diabetes, with implications for dietary counselling, behavioural modification, and improved glycaemic management strategies in affected individuals.

2. Materials and Methods

A total of 200 patients who presented to the Outpatient Department of Oral Medicine and Radiology at the People's Dental Academy, People's University, Bhopal, were screened for this study. The sample size was calculated for hypothesis testing between three means (assuming equal variances) using an effect size of 0.58, standard deviations of 25.4 and 29.4, a mean difference of 16, with a 5 % alpha error and 80 % power, yielding a required sample size of 46 per group,⁵ based on the formula provided by Lachenbruch et al. (1991).¹⁹ This cross-sectional observational study was conducted after obtaining approval from the Institutional Ethics Committee (Approval No. IEC/2023/100/02). Of these, 138 met the inclusion criteria and were assigned to the study

group, comprising 46 individuals with controlled type 2 diabetes mellitus and 46 with uncontrolled type 2 diabetes mellitus.

- Group 1: Controlled type 2 diabetes mellitus (n = 46)
- Group 2: Uncontrolled type 2 diabetes mellitus (n = 46)
- Group 3: Healthy controls (n = 46).

Eligibility for inclusion required a confirmed diagnosis of type 2 diabetes mellitus for a minimum duration of one year. Both male and female participants were included in this study. The exclusion criteria included individuals with mental or physical illnesses, habits such as tobacco use or alcohol consumption, systemic disorders other than type 2 diabetes mellitus, and use of medications other than oral hypoglycaemic agents. Additionally, patients with a history of cranial trauma, partial or complete glossectomy, pregnant or lactating women, or individuals with known food allergies were excluded from the study. Participants with any local oral conditions that could influence taste perception, such as active periodontal disease, oral mucosal lesions, recent oral surgical procedures, extensive metallic restorations or prostheses, or clinically evident oral infections, were also excluded to eliminate potential confounding factors. The control group comprised 46 healthy individuals matched for age and sex without any systemic disorders or medication use. The same exclusion criteria were applied to ensure homogeneity between the groups.²⁰

Taste solutions were prepared at various concentrations, including sucrose (sweet) at 0.19, 0.38, 0.75, 1.5, and 3 g/ml; sodium chloride (salty) at 0.06, 0.12, 0.25, 0.5, and 1 g/ml; citric acid (sour) at 0.15, 0.3, 0.6, 1.2, and 2.5 g/ml; and quinine hydrochloride (bitter) at 0.0012, 0.0025, 0.005, 0.01, and 0.02 g/ml. The preparation of these taste solutions was based on standard concentrations established in prior research to ensure consistency and comparability.²¹

Cotton swabs were used to apply the stimuli. Prior to participation, all participants were provided with a detailed explanation of the procedure and informed consent was obtained. A comprehensive case history was recorded, followed by a thorough oral examination before gustatory testing. Patients in the study group underwent laboratory assessment of glycosylated haemoglobin (HbA1c), based on which they were classified as follows: controlled diabetes mellitus (HbA1c 6–8 %), uncontrolled diabetes mellitus (HbA1c >8 %), and healthy controls (HbA1c <6 %).^{22,23}

To ensure accuracy in gustatory assessment, all participants were instructed to refrain from eating or drinking anything other than water for at least 1 h before testing. Gustatory function was evaluated using two standardised methods: the whole-mouth above-threshold taste test and the spatial taste test. Taste solutions were prepared at five concentration levels to assess the intensity of perception on a graded scale.

2.1. Whole-mouth, above-threshold taste test

The patient was asked to rinse their mouth thoroughly with distilled water, followed by presenting three rows of cups which were randomly arranged on a tray. One of the cups contained 5 ml of the sample solution, while the remaining two contained 5 ml of distilled water. The participants were given one cup of 5 ml solution at a time and were asked to sip, swish for 15 s, and spit out the sample. If the patient was unable to identify, a higher concentration was administered, and the same procedure was performed. Water was provided to remove the residual taste. The patient was then asked to correctly identify the quality of the taste (sweet, salty, sour, bitter, or tasteless), and the responses were recorded. If the participant failed to identify the correct solution, a solution with a higher concentration was presented. Once an accurate selection was made, the procedure was repeated at the same concentration until three consecutive correct responses were obtained, and it was recorded as the detection threshold and tabulated. Taste sensitivity was scored on a scale from 1 to 5, where a score of 1 indicated good taste sensitivity (detecting the lowest concentration) and a score of 5

indicated poor taste sensitivity (detecting only the highest concentration).²⁴

2.2. Spatial (localized) taste test

In this test, the ability of the participants to identify different tastes in various localised areas of the mouth was assessed. The six different locations were the right and left anterior and posterior lateral surfaces of the tongue and the two sides of the soft palate lateral to the midline. This test consisted of applying each taste stimulus at the highest concentration and distilled water (blank) in these areas for 5 s. The areas of application of the tastant and distilled water were at least 2 cm apart. Participants were asked to rate the perceived taste intensity on a 10-point scale from 0 to 9, where 0 denoted no perception and 9 denoted a very strong taste perception. To eliminate bias and residual effects, the mouth was rinsed with distilled water between each stimulus application.²⁴

Data from both whole-mouth and spatial taste tests provided a comprehensive evaluation of gustatory function across the study groups, allowing for an objective assessment of alterations in taste perception in individuals with type 2 diabetes mellitus. Statistical analyses were conducted using IBM SPSS Statistics (version 25; IBM Corp., Armonk, NY, USA) and Microsoft Excel (Microsoft Corp., Redmond, WA, USA). Descriptive statistics were used to summarise the data. The Kruskal-Wallis test was performed to compare differences among the three groups, followed by Dunn-Bonferroni post hoc tests for pairwise comparisons. The significance level (p) was set at 0.05 for all statistical analyses.

3. Results

3.1. Age distribution

The three groups had comparable age distributions. The mean age in the controlled diabetes group was 51.43 years (SD = 5.14), with an age range of 43–62 years. The non-controlled diabetes group had a slightly higher mean age of 52.2 years (SD = 5.53), ranging from 42 to 61 years. The control group had a mean age of 50.11 years (SD = 4.39), ranging from 40 to 58 years. (Table 1).

3.2. Gender distribution

The sex distribution across the three study groups showed a higher proportion of males in the diabetes groups than in the control group. In the controlled diabetes mellitus group, males accounted for 18.12 % (n = 25) and females accounted for 15.22 % (n = 21). The non-controlled diabetes mellitus group included 28 males (20.29 %) and 18 females (13.04 %). In the control group, 22 males (15.94 %) and 24 females (17.39 %) were included (Table 1).

3.3. HbA1c levels

The mean HbA1c levels varied significantly across the groups. The

controlled diabetes group had a mean HbA1c of 7.43 % (SD = 0.69), ranging from 6.1 % to 8.5 %. The non-controlled diabetes group exhibited significantly higher levels, with a mean of 11.22 % (SD = 1.65) and a range of 8.2 % to 14.2 %, reflecting poor glycaemic control. The control group maintained the lowest levels, with a mean of 5.08 % (SD = 0.63) and values ranging from 3.6 % to 5.9 %, which is consistent with normoglycaemia (Table 1).

3.4. Duration of diabetes

The duration of diabetes was marginally longer in the non-controlled diabetes group. The controlled diabetes group had a mean duration of 5.8 years (SD = 1.31), ranging from 3 to 8 years. In the noncontrolled group, the mean duration was 6.24 years (SD = 1.82), with a range of 3 to 10 years. As expected, the control group had no history of diabetes (Table 1).

3.5. Whole-mouth threshold testing scores

Whole-mouth threshold testing revealed significant differences in taste perception among individuals with controlled diabetes mellitus, non-controlled diabetes mellitus, and the control group, with poorer glycaemic control associated with higher taste thresholds.

For sweet perception, 60.87% of individuals with controlled diabetes scored 3, 10.87% scored 1 or 2, and 17.39% required higher concentrations (scores 4 or 5). In contrast, 58.70% of the noncontrolled diabetes group scored 5 and 23.91% scored 4, with no participants detecting sweetness at a score of 1. The control group exhibited the highest sensitivity, with 58.70% and 23.91% scoring 1 and 2, respectively.

Salt perception followed a similar trend. While all control participants detected salt at a score of 1, only 32.61% of the controlled diabetes group did, with 67.39% having a score of two. In the non-controlled diabetes group, 54.35% had a score of 2, and 21.74% had a score of 3.

Bitter perception was minimally affected in the controlled diabetes group (97.83% scored 1), but impairment was evident in the non-controlled diabetes group, where 73.91% scored 1, 17.39% scored 2, and 8.70 % scored 3. The control group detected bitterness at a score of 1.

Sour perception was the most impaired in the non-controlled diabetes group, with no sourness detected at a score of 1. Only 23.91% scored 2, while 56.52% required a score of 3, and 19.57% needed a score of 4. In contrast, 86.96% of the control group scored 1, while the controlled diabetes group showed moderate impairment, with 41.30% scoring 1 and 56.52% scoring 2.

The Kruskal-Wallis test revealed significant differences between the groups for all taste modalities (p < 0.001). Post hoc Dunn-Bonferroni analysis confirmed significantly higher threshold scores for sweet, salty, bitter, and sour tastes in the non-controlled diabetes group than in the controlled diabetes and control groups (p < 0.001). The controlled diabetes group also had significantly higher sour taste thresholds than the control group (p < 0.006), demonstrating a progressive decline in taste perception with worsening glycaemic control (Table 2 and Fig. 1).

Table 1
Descriptive statistics for age, HbA1c, duration, and gender distribution across study groups.

Variable	Group	N	%	Mean	SD	Min	Max	Male (n/%)	Female (n/%)
Age (years)	Controlled DM	46	33.33 %	51.43	5.14	43	62	25/18.12 %	21/15.22 %
	Non-controlled DM	46	33.33 %	52.20	5.53	42	61	28/20.29 %	18/13.04 %
	Control group	46	33.33 %	50.11	4.39	40	58	22/15.94 %	24/17.39 %
HbA1c (%)	Controlled DM	46	33.33 %	7.43	0.69	6.1	8.5	25/18.12 %	21/15.22 %
	Non-controlled DM	46	33.33 %	11.22	1.65	8.2	14.2	28/20.29 %	18/13.04 %
	Control group	46	33.33 %	5.08	0.63	3.6	5.9	22/15.94 %	24/17.39 %
Duration (years)	Controlled DM	46	33.33 %	5.80	1.31	3	8	25/18.12 %	21/15.22 %
	Non-controlled DM	46	33.33 %	6.24	1.82	3	10	28/20.29 %	18/13.04 %
	Control group	46	33.33 %	0	0	0	0	22/15.94 %	24/17.39 %

Table 2
Comparison of Whole-mouth threshold testing Scores of Sweet, Salt, Bitter, and Sour between Controlled diabetes mellitus, Non-Controlled diabetes mellitus and Control Group.

Taste Score	Group	N	Median	Mean Rank	Range (Min–Max)	P value
Sweet Score	Controlled diabetes mellitus	46	3	67.65 ^a	1–5	0.001 *
	Non-controlled diabetes mellitus	46	5	107.7 ^b	1–5	
	Control group	46	1	33.15 ^c	1–5	
Salt Score	Controlled diabetes mellitus	46	2	79.63	1–3	0.001 *
	Non-controlled diabetes mellitus	46	2	92.37	1–3	
	Control group	46	1	36.5	1–3	
Bitter Score	Controlled diabetes mellitus	46	1	64.46 ^a	1–3	0.001 *
	Non-controlled diabetes mellitus	46	1	81.04 ^b	1–3	
	Control group	46	1	63 ^c	1–3	
Sour Score	Controlled diabetes mellitus	46	2	60.7 ^a	1–4	0.001 *
	Non-controlled diabetes mellitus	46	3	111.15 ^b	1–4	
	Control group	46	1	36.65 ^c	1–4	

*Statistical significance set at 0.05; N: Number of samples; Controlled diabetes mellitus (a), Non-controlled diabetes mellitus (b), and Control group (c).

3.6. Spatial taste test scores

The spatial taste test demonstrated significant differences in taste perception across different tongue regions. The controlled diabetes group exhibited a mean sweet perception score of 39.61 (range: 24–48), indicating moderate impairment. The non-controlled diabetes group had a higher mean score of 49.3 (range: 42–54), reflecting significant deficits, while the control group showed a much lower mean score of 21.78 (range: 18–24), suggesting intact perception.

Salt perception was relatively stable across all groups. The controlled diabetes group had a mean score of 30.26, whereas the non-controlled diabetes and control groups exhibited comparable mean scores of 29.61 and 29.74, respectively, with overlapping ranges (24–36), suggesting that salt perception was largely unaffected by diabetes.

For bitter perception, the control group had a mean score of 19.43, which reflected normal function. The controlled diabetes group showed mild impairment, with a mean score of 23.87. However, the non-controlled diabetes group exhibited substantial variability, with a high mean score of 36.91 and a wide range (0–54), indicating inconsistent and often severe deficits in the bitter taste perception.

Sour perception was markedly impaired in the non-controlled diabetes group. The control group had the lowest mean score (6), which reflected normal perception. The controlled diabetes group had a mean score of 24, suggesting moderate impairment, whereas the noncontrolled diabetes group exhibited the highest mean score of 38.61 (range: 30–54), indicating profound dysfunction in sour taste perception.

The Kruskal-Wallis test confirmed significant differences in sweet, bitter, and sour taste perceptions among the groups ($p < 0.001$), leading to the rejection of the null hypothesis. Post hoc Dunn-Bonferroni tests revealed that all pairwise comparisons between the controlled diabetes, non-controlled diabetes, and control groups were statistically significant ($p < 0.05$), indicating distinct taste perception impairments in patients

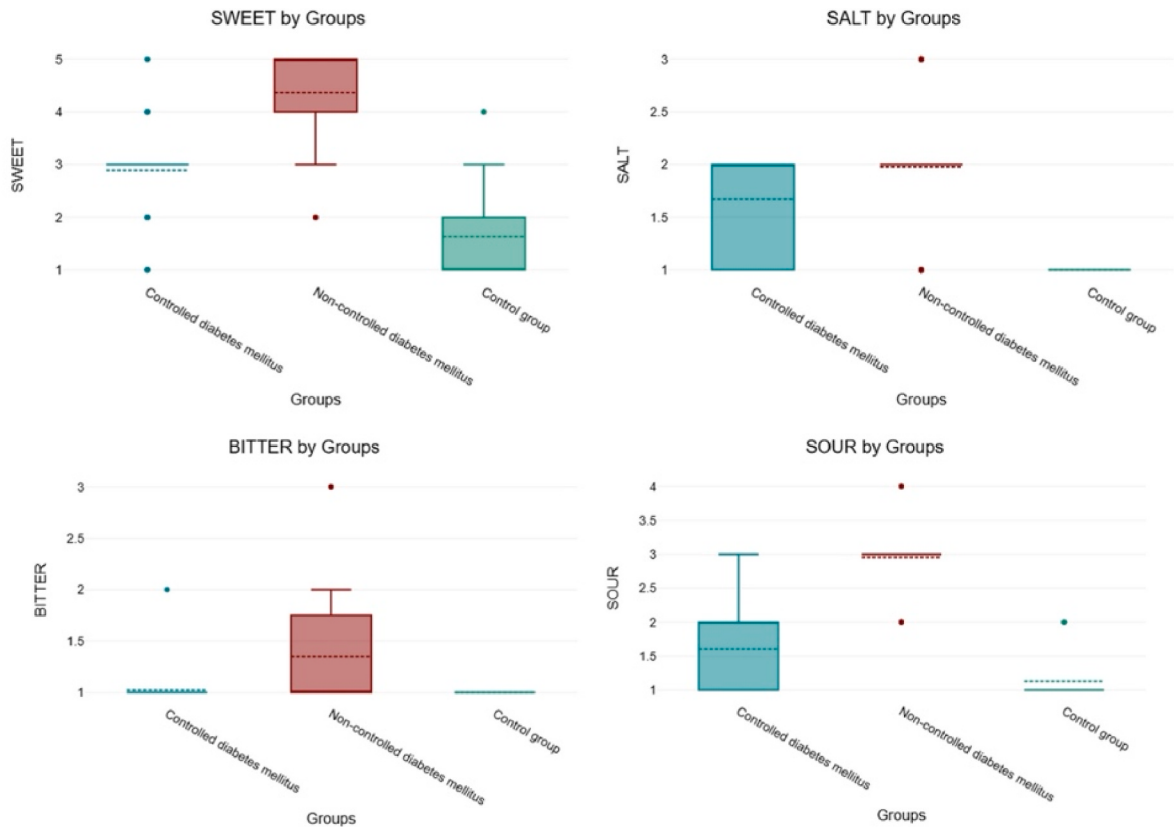


Fig. 1. Graphical comparison of whole-mouth threshold test scores for sweet, salty, bitter, and sour tastes among the controlled diabetes mellitus, noncontrolled diabetes mellitus, and control groups.

with diabetes. The non-controlled diabetes group demonstrated significantly higher spatial taste test scores for sweet, bitter, and sour perceptions than the controlled diabetes and control groups, whereas the controlled diabetes group exhibited significantly higher scores than the control group. In contrast, the Kruskal-Wallis test for salt perception showed no significant differences ($p = 0.56$), indicating that diabetes had no substantial impact on this modality (Table 3 and Fig. 2).

4. Discussion

Gustation is an essential chemical sense that plays a pivotal role in food selection, influencing dietary preferences based on an individual's nutritional needs and physiological requirements.²⁵ The taste perception mechanism is primarily dependent on the taste buds in the oral cavity, which detect various stimuli categorised into four fundamental tastes: sweet, salty, sour, and bitter.²⁶ Gustatory function is frequently compromised in individuals with diabetes mellitus; however, systematic screening for taste disorders remains neglected.²⁷ This oversight may stem from the common misconception that taste impairment is not as severe as other diabetic manifestations such as visual impairment.²⁴ Consequently, individuals with diabetes tend to ignore minor taste alterations until they experience a complete loss of taste perception, known as ageusia. However, studies have suggested that taste disorders can significantly affect food intake, leading to poor metabolic control and further exacerbating glycaemic dysregulation.²⁸

Several studies have reported that more than one-third of adult patients with diabetes experience hypogeusia, an attenuated taste perception that directly affects their ability to maintain a balanced diet.^{5,29,30} The aetiology of taste impairment in diabetes remains unclear; however, multiple hypotheses have been proposed. One possible explanation is that prolonged disease duration and long-term complications, such as peripheral neuropathy, contribute to taste dysfunction.³¹ Another perspective suggests an inherent defect in glucose taste

receptors, while an alternative hypothesis explores a broader impairment in glucose-sensing receptors, encompassing both taste receptors and pancreatic β -cells, in type 2 diabetes.³² Additionally, early onset taste disturbances within the first two years of diabetes diagnosis suggest the potential involvement of genetic or familial factors.⁴ Furthermore, persistent hyperglycaemia has been implicated as a nonspecific factor affecting gustatory function, reinforcing the multifactorial nature of taste impairment in diabetes.²⁹

The present study employed a physiological taste evaluation using solutions representing the four basic tastes (sweet, salty, sour, and bitter) to assess whole-mouth threshold taste perception and localised spatial taste sensitivity in diabetic and nondiabetic participants. The findings revealed a significant impairment in sweet taste perception, followed by sour, bitter, and salty tastes, with a highly significant p -value (<0.001) observed in the whole-mouth threshold taste test. Specifically, eight participants with uncontrolled diabetes exhibited complete ageusia to sweet taste, while 57 individuals (27 with controlled diabetes and 30 with uncontrolled diabetes) demonstrated hypogeusia. In the localised spatial taste test, two uncontrolled diabetic patients were unable to detect sweet taste, whereas 77 patients (38 with uncontrolled DM and 39 with controlled diabetic patients) exhibited hypogeusia with significant impairment in all six regions of the oral cavity. These findings highlight a generalised decline in sweet taste sensation among patients with diabetes, with more pronounced deterioration in uncontrolled diabetes. This aligns with the principle that hyperglycaemia-associated changes, rather than unrelated pathophysiological explanations, are the key contributors to the impairments observed in this cohort, thereby maintaining a results-driven discussion.

Bitter taste impairment was also observed, with three patients with uncontrolled diabetes exhibiting ageusia to bitter taste in the whole-mouth threshold test. A statistically significant difference ($p < 0.001$) was found between patients with uncontrolled diabetic patients and the control group, whereas a less significant difference ($p = 0.004$) was noted between patients with controlled diabetic patients and healthy controls. Additionally, six uncontrolled diabetic subjects failed to detect bitter taste across all six tested locations in the localised spatial taste test, confirming consistent impairment in bitter taste perception among diabetic subjects. These results contrast with those of certain studies, such as those by Gondivkar et al., who reported minimal alterations in bitter taste perception among patients with diabetes.³³

In contrast to other tastes, salt taste perception exhibited the least impairment. Although the whole-mouth threshold taste test showed significant differences between the diabetic and control groups ($p < 0.001$), the localised spatial taste test revealed no substantial differences in the mean intensity scores among the three groups. This suggests that salt taste perception remains relatively preserved in diabetes, which is consistent with some reports,²⁴ while contradicting others that have documented significant salt taste impairment.³⁴

Sour taste perception was notably impaired in patients with uncontrolled diabetes, with 23 individuals exhibiting hypogeusia. However, minimal changes were observed between patients with controlled diabetic patients and healthy controls ($p < 0.001$). These results indicate that uncontrolled diabetes is associated with a global decline in taste perception, supporting previous studies that have reported sour taste impairment in diabetic populations.^{24,34} The lack of significant differences between the controlled diabetic and control subjects suggests that glycaemic regulation may mitigate taste disturbances to some extent.

Taste perception can be influenced by numerous factors, including anatomical variations, deleterious habits, and microbial infections.³⁵ However, all the study participants presented with normal oral mucosal integrity, ruling out the possibility of infection as a confounding factor. Moreover, taste perception was bilaterally symmetrical across all 120 participants, consistent with the findings of previous studies. Saliva plays a critical role in taste function by dissolving taste stimuli and transporting them to taste buds. Reduced salivary flow, a common complaint among patients with diabetes, has been linked to gustatory

Table 3

Comparison of Spatial taste test of Sweet, Salt, Bitter, and Sour between Controlled diabetes mellitus, Non-Controlled diabetes mellitus and Control Group.

Taste Score	Group	N	Median	Mean Rank	Range (Min–Max)	P value
Sweet Score	Controlled diabetes mellitus	46	42	70.98 ^a	24–48	0.001*
	Non-controlled diabetes mellitus	46	48	113.96 ^b	42–54	
	Control group	46	22	23.57 ^c	18–24	
Salt Score	Controlled diabetes mellitus	46	30	73.28	24–36	0.56
	Non-controlled diabetes mellitus	46	30	66.92	24–36	
	Control group	46	30	68.29	24–36	
Bitter Score	Controlled diabetes mellitus	46	24	64.52 ^a	18–24	0.001*
	Non-controlled diabetes mellitus	46	30	112.87 ^b	0–54	
	Control group	46	18	31.11 ^c	18–30	
Sour Score	Controlled diabetes mellitus	46	24	69.5 ^a	24–24	0.001*
	Non-controlled diabetes mellitus	46	39	115.5 ^b	30–54	
	Control group	46	6	23.5 ^c	6–6	

*Statistical significance set at 0.05; N: Number of samples; Controlled diabetes mellitus (a), Non-controlled diabetes mellitus (b), and Control group (c).

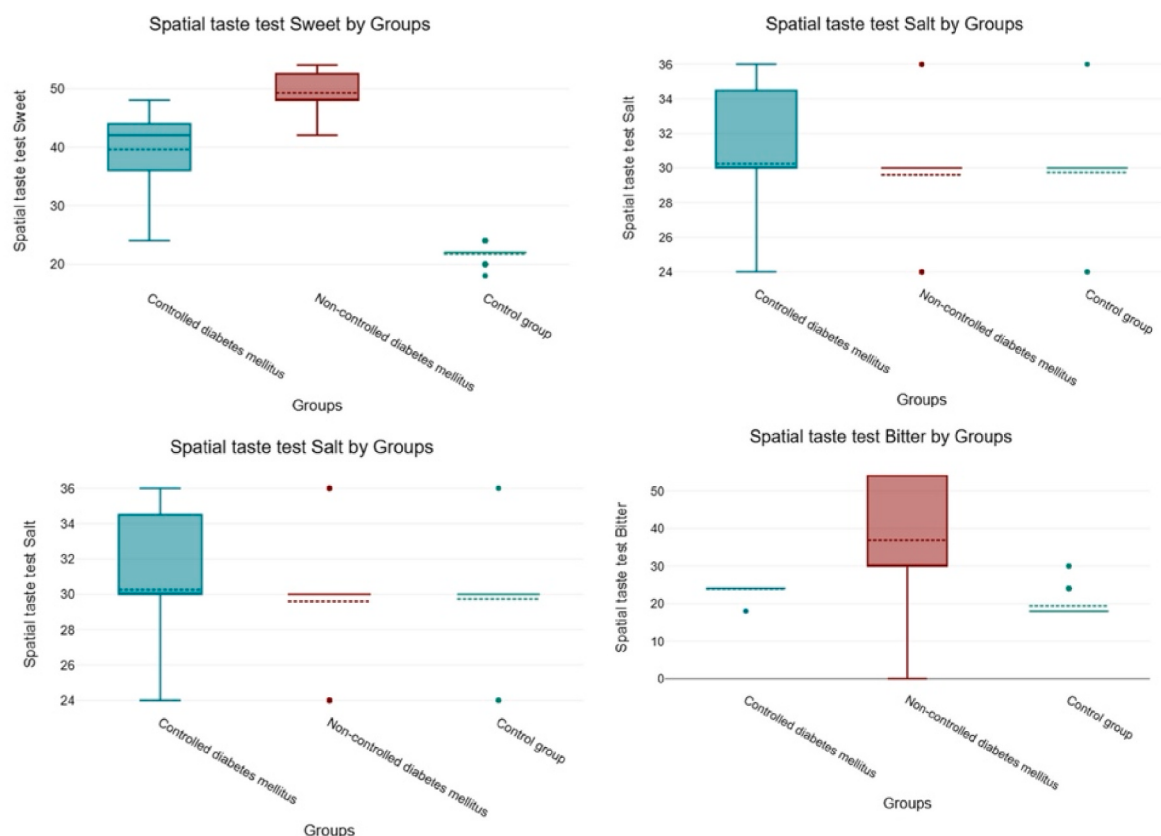


Fig. 2. Graphical comparison of spatial taste test scores for sweet, salty, bitter, and sour tastes among the controlled Diabetes Mellitus, noncontrolled Diabetes Mellitus, and control groups.

dysfunction due to impaired taste papillae maturation.³⁶ Sreebny et al. suggested that xerostomia in diabetic patients results from glycemic disturbances and direct metabolic effects on salivary glands. In the present study, subjective symptoms of oral dryness were recorded in four subjects with uncontrolled diabetes, reinforcing the hypothesis that reduced salivary function may contribute to taste impairment.³⁶

From a dental perspective, the evaluation of taste perception in individuals with diabetes has substantial clinical and preventive relevance. Taste plays a pivotal role in regulating dietary preferences, and gustatory alterations may indirectly affect oral health. In individuals with diabetes, diminished taste perception can lead to a preference for foods with higher sugar or salt content, thereby increasing the risk of dental caries, periodontal inflammation, and mucosal irritation.³⁷ Furthermore, reduced taste acuity may lower appetite or alter food selection patterns, compromising nutritional balance and ultimately influencing glycaemic control — a factor that directly affects periodontal and oral tissue health.³⁸ These dental implications directly contextualise the findings of the present study, fulfilling the need for a clearer oro-dental perspective and demonstrating how taste impairment intersects with oral disease risk and diabetic dietary behaviour.

In addition to systemic factors, several local oral conditions can modulate taste perception. Chronic periodontitis, gingival inflammation, and microbial dysbiosis can impair taste bud function through inflammatory mediators and alterations in the oral microenvironment.³⁹ Similarly, metallic restorations, prostheses, and dental appliances can contribute to dysgeusia via galvanic interactions, corrosion by-products, or surface oxidation, which interfere with taste receptor function.⁴⁰ Recent oral surgical procedures, mucosal trauma, or extractions may temporarily disturb the integrity of the lingual and chorda tympani nerves, resulting in transient taste dysfunction.⁴¹ Moreover, local salivary gland hypofunction, coating of the tongue with debris, and reduced epithelial turnover associated with diabetes can compound the

impairment of taste sensation.⁴²

From a clinical standpoint, awareness of these factors is crucial for dental practitioners, as altered taste perception may affect a patient's motivation to maintain oral hygiene, adherence to prescribed diets, and acceptance of oral health interventions. Since patients with diabetes frequently seek dental care for complications such as xerostomia, periodontitis, and candidiasis, incorporating taste assessment into routine dental evaluations could facilitate the early recognition of sensory disturbances and prompt interdisciplinary management. Therefore, the study of taste perception in diabetic individuals extends beyond systemic considerations and offers valuable insights into comprehensive oral healthcare, dietary counselling, and patient-centered preventive strategies within dental practice.⁴¹

A significant correlation was observed between glycosylated haemoglobin (HbA1c) levels and sweet taste impairment ($p < 0.001$) in both diabetic groups. Although other tastes did not show a direct correlation with HbA1c, the overall decline in taste threshold suggests a relationship between hyperglycaemia and altered gustatory function. Previous research has indicated that taste perception generally declines with age, particularly for salt and bitter tastes, whereas sweet and sour perceptions remain relatively unaffected.⁴³ Given that the mean age of the study participants was 51 years, age-related decline was unlikely to have influenced the observed taste impairments, further supporting the conclusion that diabetes was the primary factor affecting taste function.

The strengths of the present study include its systematic approach to taste assessment, exclusion of confounding factors such as peripheral neuropathy, medication-induced dysgeusia, and deleterious habits, and the use of both whole-mouth and localised spatial taste tests to ensure comprehensive evaluation. Additionally, this study provides valuable insights into the correlation between HbA1c levels and taste impairment, emphasising the importance of glycaemic control in preserving gustatory function. However, the limitations of this study include the

relatively small sample size and lack of assessment of other potential contributing factors, such as genetic predisposition, hormonal variations, and broader metabolic influences. Future studies should consider a larger sample size with more diverse demographic representations, along with advanced diagnostic tools such as electrogustatory testing and functional neuroimaging, to elucidate the underlying mechanisms of taste impairment in diabetes. Furthermore, investigating interventions to enhance taste perception in patients with diabetes, such as dietary modifications and taste receptor-targeted therapies, could provide novel strategies for improving nutritional intake and glycaemic management.

5. Conclusion

The present findings underscore the need for routine screening of gustatory function in patients with diabetes mellitus. Despite the high prevalence of taste impairment (95 % of the diabetic subjects in this study), only 2.5 % of the individuals reported subjective taste alterations. This highlights the need for objective testing to identify gustatory dysfunction at an early stage of disease. Given the significant impact of taste perception on dietary habits and metabolic regulation, oral health professionals and general physicians should incorporate taste assessments into standard diabetic care protocols. Future research should explore methods to enhance taste receptor sensitivity, refine dietary counselling approaches, and develop meal plans tailored to the altered taste perception of patients with diabetes, thereby promoting better glycaemic control and overall health outcomes.

Patient's/guardian's consent statement

Prior to enrollment in the study, written informed consent was obtained from all the participants. This process ensured that all participants and their guardians were fully informed about the study's procedures, potential risks, and benefits, and voluntarily agreed to take part in the research.

Ethical clearance statement

Ethical clearance for this study was obtained from the Institutional Ethical Committee of People's Dental Academy (Approval No: IEC/2023/100/02). Written informed consent was obtained from parents/guardians, and assent was obtained from the children before enrollment.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgement

We extend our heartfelt gratitude to the postgraduates, teaching and non-teaching faculty of the Department of Oral Medicine and Radiology, People's Dental Academy for their unwavering support and encouragement.

References

- Suzuki T. Cellular mechanisms in taste buds. *Bull Tokyo Dent Coll.* 2007;48(4): 151–161. <https://doi.org/10.2209/tdcpub.48.151>.
- Taruno A, Gordon MD. Molecular and cellular mechanisms of salt taste. *Annu Rev Physiol.* 2023;85(1):25–45. <https://doi.org/10.1146/annurev-physiol-031522-075853>.
- Melis M, Tomassini Barbarossa I, Sollai G. The implications of taste and olfaction in nutrition and health. *Nutrients.* 2023;15(15):3412. <https://doi.org/10.3390/nu15153412>.
- Catamo E, Robino A, Tinti D, et al. Altered taste function in young individuals with type 1 diabetes. *Front Nutr.* 2022;8. <https://doi.org/10.3389/fnut.2021.797920>.
- Pugnaloni S, Alia S, Mancini M, et al. A study on the relationship between type 2 diabetes and taste function in patients with good glycemic control. *Nutrients.* 2020; 12(4):1112. <https://doi.org/10.3390/nu12041112>.
- Tastan Eroglu Z, Kalender ME, Ucan Yarkac F, Babayigit O, Ozkan Sen D. Impact of non-surgical periodontal therapy on self-perceived halitosis, and the senses of smell and taste: a prospective clinical study. *BMC Oral Health.* 2025;25(1):321. <https://doi.org/10.1186/s12903-025-05702-2>.
- Mody MA, Ruparelia PB, Patel M, Ruparelia KP. Assessment of selective salivary properties and taste perception in subjects with oral submucous fibrosis – a case-control study. *J Oral Biol Craniofac Res.* 2025;15(6):1676–1680. <https://doi.org/10.1016/j.jobcr.2025.09.017>.
- Gokhroo A, Bhardwaj P, Chowdhary Z, Agarwal N, Soni A, Dubey T. Comparative evaluation of altered taste perception among oral submucous fibrosis patients. *Contemp Clin Dent.* 2021;12(4):426–432. <https://doi.org/10.4103/ccd.ccd.664.20>.
- Neyraud E, Morzel M. Biological films adhering to the oral soft tissues: structure, composition, and potential impact on taste perception. *J Texture Stud.* 2019;50(1): 19–26. <https://doi.org/10.1111/jtxs.12363>.
- Rud I, Almli VL, Berget I, Tzimiras D, Varela P. Taste perception and oral microbiota: recent advances and future perspectives. *Curr Opin Food Sci.* 2023;51, 101030. <https://doi.org/10.1016/j.cofs.2023.101030>.
- Mauri-Obradors E, Estrugo-Devesa A, Jane-Salas E, Vinas M, Lopez-Lopez J. Oral manifestations of diabetes mellitus. A systematic review. *Med Oral Patol Oral Cir Bucal.* 2017. <https://doi.org/10.4317/medoral.21655>. Published online, 0-0.
- Snyder DJ, Bartoshuk LM. Oral sensory nerve damage: causes and consequences. *Rev Endocr Metab Disord.* 2016;17(2):149–158. <https://doi.org/10.1007/s11154-016-9377-9>.
- Niki A, Niki H. IS diabetes mellitus a Disorder of the glucoreceptor? *Lancet.* 1975; 306(7936):658. [https://doi.org/10.1016/S0140-6736\(75\)90137-3](https://doi.org/10.1016/S0140-6736(75)90137-3).
- Mojet J. Taste perception with age: generic or specific losses in threshold sensitivity to the five basic tastes? *Chem Senses.* 2001;26(7):845–860. <https://doi.org/10.1093/chemse/26.7.845>.
- Tarekegn ET, Gobeze MY, Haile MB, Zerga AA. Glycemic control and associated factors among type 2 diabetes patients attending at Dessie comprehensive specialized hospital outpatient department. *Sci Rep.* 2025;15(1):9286. <https://doi.org/10.1038/s41598-025-93739-2>.
- Yahaya JJ, Doya IF, Morgan ED, Ngaiza AI, Bintabara D. Poor glycemic control and associated factors among patients with type 2 diabetes mellitus: a cross-sectional study. *Sci Rep.* 2023;13(1):9673. <https://doi.org/10.1038/s41598-023-36675-3>.
- Yamauchi Y, Endo S, Sakai F, Yoshimura I. Whole mouth gustatory test (part 1)–basic considerations and principal component analysis. *Nihon Jibiinkoka Gakkai Kaiho.* 1995;98(1):119–129.
- Spence C. The tongue map and the spatial modulation of taste perception. *Curr Res Food Sci.* 2022;5:598–610. <https://doi.org/10.1016/j.cofs.2022.02.004>.
- Lachenbruch PA, Lwanga SK, Lemeshow S. Sample size determination in health studies: a practical manual. *J Am Stat Assoc.* 1991;86(416):1149. <https://doi.org/10.2307/2290547>.
- Har-Zion G. Psychophysical testing of taste and flavour reactivity in young patients undergoing treatment with removable orthodontic appliances. *Eur J Orthod.* 2004; 26(1):73–78. <https://doi.org/10.1093/ejo/26.1.73>.
- Gudziol H, Hummel T. Normative values for the assessment of gustatory function using liquid tastants. *Acta Otolaryngol.* 2007;127(6):658–661. <https://doi.org/10.1080/00016480600951491>.
- Klimacka-Nawrot E, Suchecka W, Błońska-Fajfrowska B. Gustometry usefulness for the evaluation of taste sense efficiency. Part I. The range of taste substances concentrations and the result of gustometry examination. *Wiad Lek.* 2007;60(9-10): 409–414.
- Alberti KGMM, Zimmet PZ. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus. Provisional report of a WHO consultation. *Diabet Med.* 1998;15(7): 539–553. [https://doi.org/10.1002/\(SICI\)1096-9136\(199807\)15:7<539::AID-DIA668>3.0.CO;2-S](https://doi.org/10.1002/(SICI)1096-9136(199807)15:7<539::AID-DIA668>3.0.CO;2-S).
- Khera S, Saigal A. Assessment and evaluation of gustatory functions in patients with diabetes mellitus Type II: a study. *Indian J Endocrinol Metab.* 2018;22(2):204. <https://doi.org/10.4103/ijem.IJEM.555.17>.
- Hossain MdS, Wazed MA, Asha S, et al. Flavor and well-being: a comprehensive review of food choices, nutrition, and health interactions. *Food Sci Nutr.* 2025;13(5). <https://doi.org/10.1002/fsn3.70276>.
- Pritchard TC. Gustatory system. In: *Brain Mapping.* Elsevier; 2015:313–316. <https://doi.org/10.1016/B978-0-12-397025-1.00228-1>.
- Aframian DJ, Zedan A, Agbariah W, Rettman A, Almozno G. Quantitative assessment of gustatory function and its association with demographics, and systemic morbidity. *Biology (Basel).* 2024;13(1):50. <https://doi.org/10.3390/biology13010050>.
- Tsujimoto T, Imai K, Kanda S, Kakei M, Kajio H, Sugiyama T. Sweet taste disorder and vascular complications in patients with abnormal glucose tolerance. *Int J Cardiol.* 2016;221:637–641. <https://doi.org/10.1016/j.ijcard.2016.07.062>.

29. De Carli L, Gambino R, Lubrano C, et al. Impaired taste sensation in type 2 diabetic patients without chronic complications: a case-control study. *J Endocrinol Investig*. 2018;41(7):765–772. <https://doi.org/10.1007/s40618-017-0798-4>.
30. Catamo E, Tornese G, Concas MP, Gasparini P, Robino A. Differences in taste and smell perception between type 2 diabetes mellitus patients and healthy controls. *Nutr Metabol Cardiovasc Dis*. 2021;31(1):193–200. <https://doi.org/10.1016/j.numecd.2020.08.025>.
31. Risso D, Drayna D, Morini G. Alteration, reduction and taste loss: main causes and potential implications on dietary habits. *Nutrients*. 2020;12(11):3284. <https://doi.org/10.3390/nu12113284>.
32. Medina A, Nakagawa Y, Ma J, et al. Expression of the glucose-sensing receptor T1R3 in pancreatic islet: changes in the expression levels in various nutritional and metabolic states. *Endocr J*. 2014;61(8):797–805. <https://doi.org/10.1507/endocrj.EJ14-0221>.
33. Gondivkar SM, Indurkar A, Degwekar S, Bhowate R. Evaluation of gustatory function in patients with diabetes mellitus type 2. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2009;108(6):876–880. <https://doi.org/10.1016/j.tripleo.2009.08.015>.
34. Kushwaha JS, Gupta VK, Singh A, Giri R. Significant correlation between taste dysfunction and HbA1C level and blood sugar fasting level in type 2 diabetes mellitus patients in at a tertiary care center in north India. *Diabetes Epidemiol Manag*. 2022;8, 100092. <https://doi.org/10.1016/j.deman.2022.100092>.
35. Breslin PAS. An evolutionary perspective on food and human taste. *Curr Biol*. 2013; 23(9):R409–R418. <https://doi.org/10.1016/j.cub.2013.04.010>.
36. Matsuo R. Role of saliva in the maintenance of taste sensitivity. *Crit Rev Oral Biol Med*. 2000;11(2):216–229. <https://doi.org/10.1177/10454411000110020501>.
37. Angarita-Díaz M del P, Fong C, Bedoya-Correa CM, Cabrera-Arango CL. Does high sugar intake really alter the oral microbiota?: a systematic review. *Clin Exp Dent Res*. 2022;8(6):1376–1390. <https://doi.org/10.1002/cre2.640>.
38. Alves LSM, Munduri JM de S, Mattos MC de O, Stefani CM, Dame-Teixeira N. Changes in taste perception in elderly population and its potential impact on oral health: a systematic review with meta-analysis. *Front Oral Health*. 2024;5. <https://doi.org/10.3389/froh.2024.1517913>.
39. Dong H, Liu J, Zhu J, et al. Oral microbiota-host interaction mediated by taste receptors. *Front Cell Infect Microbiol*. 2022;12. <https://doi.org/10.3389/fcimb.2022.802504>.
40. Alnazzawi A. Effect of fixed metallic oral appliances on oral health. *J Int Soc Prev Community Dent*. 2018;8(2):93. https://doi.org/10.4103/jispcd.JISPCD_416_17.
41. Al-Maskari AY, Al-Maskari MY, Al-Sudairy S. Oral manifestations and complications of diabetes mellitus: a review. *Sultan Qaboos Univ Med J*. 2011;11(2):179–186.
42. Lafargue B, D'Andréa G, Fabre R, Alshukry A, Vandersteen C, Guevara N. Taste disorders after middle ear surgery: Chorda Tympani nerve injury and quality of life. *Otolaryngol Head Neck Surg*. 2024;171(6):1834–1841. <https://doi.org/10.1002/ohn.920>.
43. Alia S, Aquilanti L, Pugnali S, Di Paolo A, Rappelli G, Vignini A. The influence of age and oral health on taste perception in older adults: a case-control study. *Nutrients*. 2021;13(11):4166. <https://doi.org/10.3390/nu13114166>.