

How Real-time Blood Glucose Shapes Sweet Taste Sensitivity: A Comparative Study in Diabetic and Healthy Subjects



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Received: 13 December 2025; Accepted: 25 August 2026

[Visual Abstract](#)

ABSTRACT

Background: Diabetes mellitus is a prevalent medical condition with significant morbidity and mortality. Dietary and lifestyle changes are often prescribed to reverse insulin sensitivity in addition to pharmacological interventions. Significant alterations in dietary preferences are noted in patients with increased blood glucose levels and may adversely affect compliance with dietary recommendations.

Materials and methods: Seventy-three diabetic and nondiabetic subjects were recruited, and their random blood glucose levels were assessed. Their sweet taste thresholds were assessed immediately using concentration-graded glucose solutions. Differences in the sweet taste perception thresholds between the two groups were determined by Fisher's exact test. The association between the instantaneous blood glucose and the sweet taste thresholds was explored using Spearman's correlation.

Results: The diabetic group had a significantly higher threshold for sweet taste perception in comparison with the nondiabetic group ($p < 0.001$). A significant correlation was noted between the instantaneous blood glucose levels and the sweet taste threshold concentrations ($p = 0.840$, 95% confidence interval (CI): 0.756 to 0.897, $p < 0.001$). An independent association between body mass index (BMI) and the sweet taste thresholds could not be established ($p = 0.685$).

Conclusion: Significantly higher sweet taste thresholds in diabetic patients may attenuate their ability to perceive sweetness and may lead to a tendency toward higher consumption of glucose, deleteriously affecting their glycemic control.

Keywords: Blood glucose, Body mass index, Diabetes mellitus, Glycemic control, Taste perception.

Journal of The Association of Physicians of India (2026): 10.59556/japi.74.1745

INTRODUCTION

Diabetes mellitus (DM) is considered a worldwide epidemic currently affecting 589 million adults and projected to reach 853 million by 2050 as per International Diabetes Federation (IDF) research, contributing to about 3.4 million deaths annually.¹ Uncontrolled diabetes may potentially lead to significant complications such as coronary artery disease, neuropathy, infections, renal diseases, etc., and these adversely affect the quality of life of the patient, the family, and society.² Recent trends show a rapid rise in diabetic incidence among the younger populations in Asia. Central Asia has the fastest-growing age-standardized incidence rate (ASIR), and South Asia had the highest deaths (410,254) and disability-adjusted life years (DALYs) (16,851,968) in 2021.¹

In India, over 77 million are affected by type 2 diabetes mellitus (T2DM) and are expected to reach 134 million by 2045.³ Nearly 25 million people are prediabetic, with impaired glucose tolerance (IGT), and more than 50% are unaware of their diabetic status.⁴ T2DM can be mainly attributed to genetic predispositions as well as to the rapid socioeconomic transition that our

country is undergoing, which has resulted in changes in nutrition—increased calorie intake, decreased physical activity levels, and rising obesity rates.^{5,6} Dietary preference for specific compounds in the diet has been linked to multiple noncommunicable diseases. According to the 2020 Indian Council of Medical Research–National Institute of Nutrition (ICMR–NIN) report, urban and rural Indians consume about 73% and 78% of their energy through carbohydrates, respectively.⁷ A strong association was noted between the quality of carbohydrate, measured as glycemic load or glycemic index (GI), and risk for DM. This includes refined grains, processed meats, and sugary drinks.⁸

A less explored factor that could be attributed to the risk of developing DM is changes in taste sensitivity for certain dietary components. There appear to be alterations in dietary preferences in select noncommunicable diseases like dyslipidemia, diabetes, and obesity. Available literature has indicated that a decreased taste sensitivity to fat is linked to increased fat intake and a corresponding increase in body mass index (BMI).^{9–11} However, these studies have not clearly established the causal relationships between diet and the clinical disorder.

Similar studies linking sweet taste sensitivity and sugar intake have shown a range of results: from a negative association to no association to a positive association.¹² This can be attributed to the varied methods of estimation of food intake, demographics, sweet taste measurement methods, etc. Currently, there is limited evidence linking alterations in sweet taste threshold and instantaneous blood glucose levels, or whether there are differences between sweet threshold in diabetics and nondiabetics. A proven association between blood glucose levels and sweet taste threshold could contribute to understanding the changes in dietary preferences in diabetics and the subsequent glycemic control. Therefore, our primary objective was to explore the association between instantaneous blood glucose levels and sweet taste perception levels in diabetic and nondiabetic male subjects.

The relation between taste perception and body weight is essentially an area that needs to be explored. Olfactory and gustatory stimulations can induce excessive food consumption, which could lead to weight gain. Some studies have shown a positive association between body mass and sweet taste thresholds.^{13,14} Slower patterns of habituation to the palatability of sweet sensation were seen in obese women, which may lead to slower satiation rate and protracted eating episodes, which in turn could cause increased food intake.¹⁵ Therefore, our secondary objective was to explore the association between BMI and alterations in sweet taste perception thresholds.

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How to cite this article: Verghese S, Sneha PM, Raj JM, et al. How Real-time Blood Glucose Shapes Sweet Taste Sensitivity: A Comparative Study in Diabetic and Healthy Subjects. *J Assoc Physicians India* 2026;74(10):e8–e12.

MATERIALS AND METHODS

Subjects

The study was conducted for one year (between December 2023 and December 2024) at a tertiary care teaching hospital in South India. Since the study explored the taste thresholds in diabetics and non-diabetics, the study was designed as a cross – sectional observational study with blood glucose levels and sweet taste thresholds as the primary outcome measures.

Our study aimed at exploring the alterations in sweet taste thresholds in self-reported diabetic and non-diabetic subjects. The diabetic group included subjects with optimal as well as suboptimal glycemic control. The study was conducted on 73 eligible subjects between 45 and 65 years. All study procedures were reviewed and approved by the Institutional Ethics Committee. The subjects provided written informed consent on forms prior to participation. The eligibility of the subjects was assessed against the checklist before recruitment. Female subjects were not included in the study as hormonal changes during a menstrual cycle have been shown to affect taste perception.¹⁶ Subjects were screened for poor oral hygiene, oral ulcers, recent dental procedures, and use of medications that could affect taste perception. Once recruited, the participants were instructed not to have food, tea, or coffee; not to use mouth fresheners or any other substances that affect taste perception; and to abstain from smoking for 3 hours before participation. The participants were asked to wear light clothing and to avoid heavily scented products. Of the 98 participants initially screened, 11 subjects reported pain/ulceration in the mouth, 8 reported habitual consumption of tobacco products and betelnuts, and 6 reported concurrent medications that were known to affect taste perception.

For our secondary objective, we classified the subjects as non-obese if the BMI was <25.0 kg/m² and obese if the BMI was > 25.0 kg/m².¹⁷

Methodology

The participants were taken to a suitable, comfortable room with a sink available for expectoration. The following assessments were done:

- **Height:** Height was measured using a standardized scale to the nearest 0.1 cm.
- **Weight:** Weight was recorded using a standard weighing scale to the nearest 0.1 kg. The height and weight were used to calculate the body mass index (BMI).
- **Random blood glucose:** Instantaneous blood glucose was taken using a standard glucometer through capillary puncture under all aseptic precautions.

- **Sweet taste threshold:** The sweet taste testing method used was 'multiple forced-choice presentation' with 'staircase procedure' in an ascending series.¹⁸ Taste was assessed using serial dilutions of a sweet solution made by dissolving precise gravimetric measures of lab- and food-grade glucose in de-ionized water. Twelve glucose test solutions were prepared with concentrations from 0.73 mM to 405 mM, with each successive concentration being twice the previous one.¹⁹ During the testing, each concentration level had three solutions presented in a randomized order: two 5-mL deionized-water samples and one 5-mL test solution. For each level, participants were given one solution at a time, asked to swish it around the mouth and then expectorate it and report if they could perceive sweetness or not. If they reported correctly for the three solutions in a level, retesting at the same level was done after a gap of 5 minutes. If the subject failed to report correctly, either at the test or at the retest, then the next higher level was tested. The subjects were encouraged to refrain from guessing which one of the solutions is the sweet solution before the start of the taste threshold assessment. Between the presentation of each solution, the mouth was rinsed with deionized water to prevent residual carry-over effects. The lowest concentration level at which the subjects were able to correctly identify the sweet and the control solutions at the test and retest instances was taken as the sweet perception threshold.

Sample Size

According to the study by Wasalathanthri et al., the mean and standard deviation of taste threshold in normal and diabetic subjects were reported as 0.015 ± 0.01 mol/L and 0.025 ± 0.01 mol/L.²⁰ Expecting the same difference in taste threshold between normal and diabetic groups in our study, the estimated sample size was 17 subjects in each group with a 5% level of significance and 80% power.

Table 1: Baseline characteristics of the diabetic (self-reported) and non-diabetic population

Parameter	Diabetic (n = 37)	Nondiabetic (n = 36)	p-value
Age (years)	53.1 $\pm$ 5.4	51.4 $\pm$ 4.7	0.16
Height (cm)	165.4 $\pm$ 5.7	166.8 $\pm$ 5.5	0.29
Weight (kg)	69.8 $\pm$ 11.3	69.6 $\pm$ 9.6	0.91
BMI	25.5 $\pm$ 3.3	25.0 $\pm$ 3.0	0.53
Nonobese subjects (n)	19 (51.4%)	19 (52.8%)	0.99
Obese (n)	18 (48.6%)	17 (47.2%)	
Blood glucose (mg/dL)	226.0 (168.0, 298.0)	113.5 (101.0, 130.0)	<0.001

Anthropometric measures expressed as mean $\pm$ SD. Blood glucose expressed as median and interquartile range

Statistical Analyses

The statistical package used for all statistical analyses was Stata version 16.0 (StataCorp LLC, College Station, TX, USA). Diabetic status was defined as a binary variable (diabetic vs. nondiabetic). Continuous variables were summarized as mean $\pm$ standard deviation (SD) for approximately normally distributed data and as median (interquartile range, IQR) for non-normally distributed data, while categorical variables were expressed as frequencies and percentages. The Shapiro–Wilk test was used to assess normality.

Between-group comparisons for continuous variables were conducted using the independent samples *t*-test for normally distributed data and the Mann–Whitney *U* test for nonparametric variables. Categorical variables were compared using the Pearson's chi-squared test or Fisher's exact test when expected cell counts were less than five.

Associations between sweet taste threshold and blood glucose levels, and between sweet taste threshold and BMI, were assessed using Spearman's rank correlation coefficient (ρ). All the statistical tests performed were two-tailed, and a *p*-value < 0.05 was considered statistically significant.

RESULTS

Participant Characteristics

A total of 73 participants were included in the analysis, comprising 37 individuals with diabetes and 36 nondiabetic controls. The self-reported diabetic and nondiabetic groups were comparable for age, height, and weight. The distribution of obese and non-obese subjects was comparable between the groups (Table 1).

Blood Glucose and Sweet Taste Threshold Measurements

As expected, blood glucose levels were significantly higher in the self-reported diabetic group compared to nondiabetic controls [median (IQR): 226.0 (168.0, 298.0) vs. 113.5 (101.0, 130.0) mg/dL, *p* < 0.001].

Analysis of taste threshold levels and diabetic status showed a statistically

significant association (Fisher's exact test, $p < 0.001$). The distribution patterns showed marked differences between diabetic and nondiabetic participants (Table 2 and Fig. 1):

- Nondiabetic participants demonstrated lower taste thresholds, with 69.45% having thresholds ≤ 23.08 mM, including 30.56% at 7.3 mM and 33.33% at 12.98 mM.
- Diabetic participants showed elevated taste thresholds, with 64.86% having thresholds ≥ 73 mM, including notable concentrations at 228.94 mM (27.03%) and higher levels.
- No diabetic participants had taste thresholds below 12.98 mM, while no nondiabetic participants required thresholds above 41.05 mM.

Table 2 : Distribution of the sweet taste thresholds among diabetic (self-reported) and nondiabetic subjects

Sweet threshold level (mM/L)	Diabetic	Nondiabetic	p-value
Level 1 (0.73)	0 (0.0%)	0 (0.0%)	$p < 0.001$
Level 2 (1.30)	0 (0.0%)	0 (0.0%)	
Level 3 (2.31)	0 (0.0%)	0 (0.0%)	
Level 4 (4.11)	0 (0.0%)	2 (5.6%)	
Level 5 (7.30)	0 (0.0%)	11 (30.6%)	
Level 6 (12.98)	4 (10.8%)	12 (33.3%)	
Level 7 (23.08)	5 (13.5%)	9 (25.0%)	
Level 8 (41.05)	4 (10.8%)	2 (5.6%)	
Level 9 (73.00)	5 (13.5%)	0 (0.0%)	
Level 10 (129.21)	4 (10.8%)	0 (0.0%)	
Level 11 (228.94)	10 (27.0%)	0 (0.0%)	
Level 12 (405.35)	5 (13.5%)	0 (0.0%)	
Total	37 (100.0%)	36 (100.0%)	

Analysis was done with Fisher's exact test.

Table 3: Baseline characteristics of the non-obese (BMI < 25) and obese (BMI > 25) subjects

Factor	BMI > 25 (Obese) (n = 35)	BMI < 25 (nonobese) (n = 38)	p-value
Age (years)	52.0 $\pm$ 5.4	52.9 $\pm$ 4.7	0.599
Height (cm)	166.0 $\pm$ 5.1	166.0 $\pm$ 6.1	0.949
Weight (kg)	77.1 $\pm$ 8.6	62.9 $\pm$ 6.4	<0.001
BMI	27.9 $\pm$ 2.1	22.8 $\pm$ 1.4	<0.001
Diabetes status (n)	18 (51.4%)	19 (50.0%)	0.99
Diabetic	17 (48.5%)	19 (50.0%)	
Nondiabetic			
Blood glucose (mg/dL)	146.0 (121.0, 215.0)	135 (110.0, 238.0)	0.404

Anthropometric measures were expressed as mean $\pm$ SD. Blood glucose was expressed as median and interquartile range. Diabetic status was self-reported.

Body Mass Index and Sweet Taste Threshold

The two groups (obese group and the non-obese group) were comparable for age, blood glucose levels, and diabetes status (Table 3). No statistically significant differences in sweet taste threshold levels were observed between the two study groups (Fisher's exact test, $p = 0.685$). Both categories showed similar distributions across taste threshold levels, with a relatively even spread across the lower to middle threshold ranges (7.3–23.08 mM representing 52.15% and 55.26% for the two groups, respectively) (Table 4 and Fig. 2).

Correlation Analysis

Spearman's rank correlation analysis revealed a strong positive association between taste threshold (mM/L) and blood glucose levels ($\rho = 0.840$, 95% confidence interval (CI): 0.756 to 0.897, $p < 0.001$). This demonstrates that higher blood glucose levels are significantly associated with elevated sweet taste threshold levels (Fig. 3).

In contrast, no significant correlation was found between taste threshold levels and BMI ($\rho = 0.121$, 95% CI: -0.112 to 0.341, $p = 0.309$), suggesting that body weight status does not significantly influence sweet taste perception in this population.

Discussion

Our study showed that there was a strong and significant association between the instantaneous blood glucose concentrations and the threshold for the perception of sweet taste. Since this is a cross-sectional study, we are unable to derive insights into the causal framework of this relationship. One proposed concept is the adaptation of sweet taste receptors to the increased blood glucose levels, similar to the acute adaptation

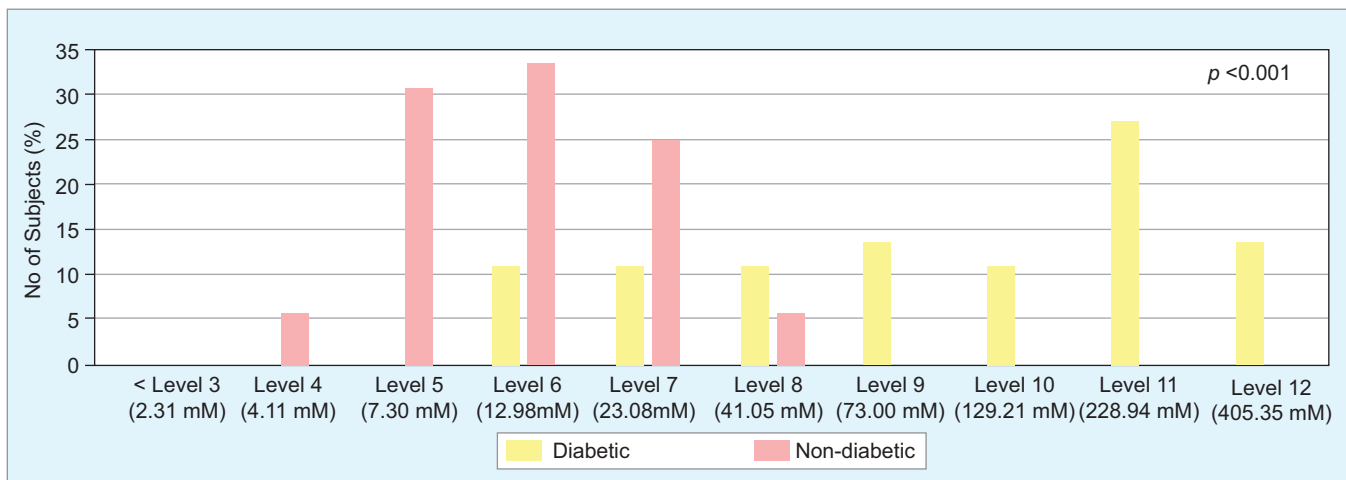


Fig. 1: Distribution of the sweet taste thresholds among self-reported diabetic and non-diabetic subjects

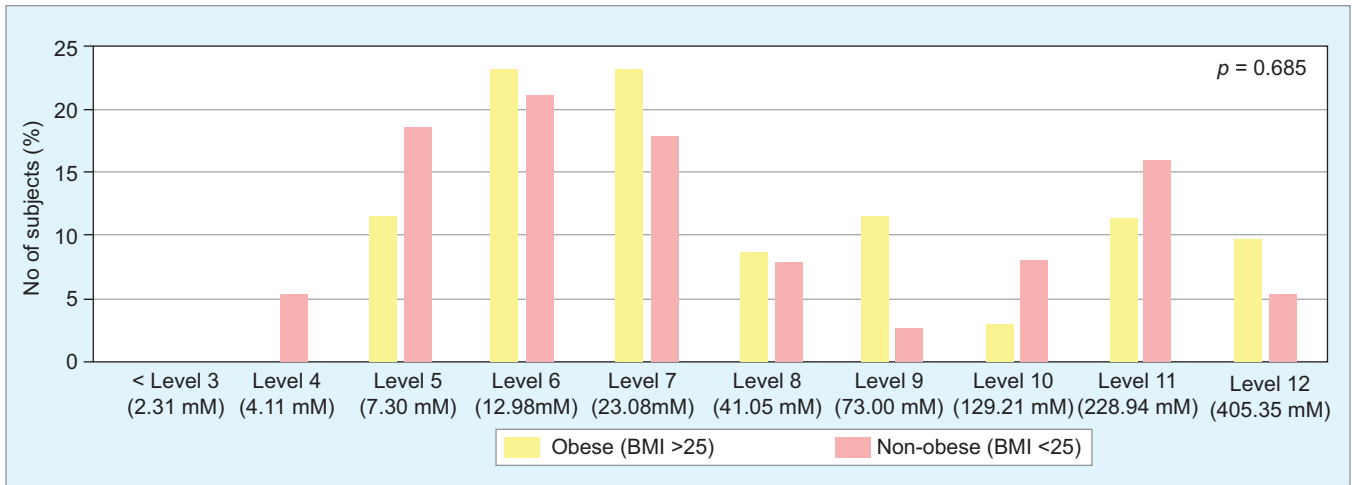


Fig. 2: Distribution of the sweet taste thresholds among nonobese and obese groups. Analysis was done with Fisher's exact test

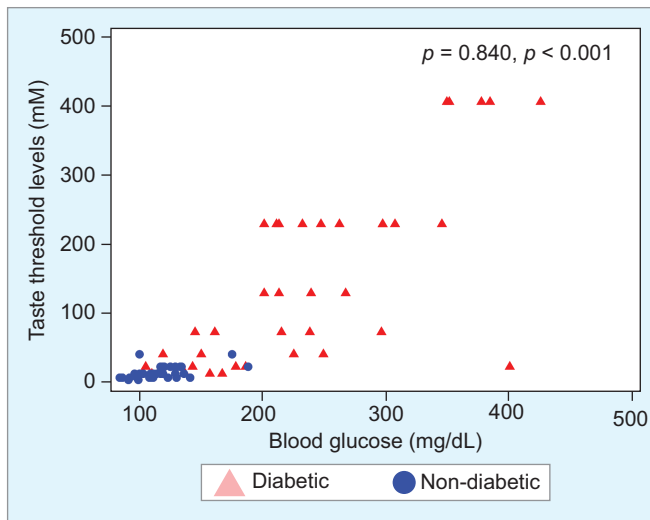


Fig. 3: Correlation between instantaneous blood glucose levels and sweet taste threshold levels

Table 4: Distribution of the sweet taste thresholds among non-obese and obese subjects.

Sweet threshold level (mM)	Obese (BMI > 25)	Nonobese (BMI < 25)	p-value
Level 1 (0.73)	0 (0.0%)	0 (0.0%)	p = 0.685
Level 2 (1.30)	0 (0.0%)	0 (0.0%)	
Level 3 (2.31)	0 (0.0%)	0 (0.0%)	
Level 4 (4.11)	0 (0.0%)	2 (5.3%)	
Level 5 (7.30)	4 (11.4%)	7 (18.4%)	
Level 6 (12.98)	8 (22.9%)	8 (21.1%)	
Level 7 (23.08)	8 (22.9%)	6 (15.8%)	
Level 8 (41.05)	3 (8.6%)	3 (7.9%)	
Level 9 (73.00)	4 (11.4%)	1 (2.6%)	
Level 10 (129.21)	1 (2.9%)	3 (7.9%)	
Level 11 (228.94)	4 (11.4%)	6 (15.8%)	
Level 12 (405.35)	3 (8.6%)	2 (5.3%)	
Total	35 (100.0%)	38 (100.0%)	

Analysis was done with Fisher's exact test.

of the sweet receptors to the consumption of sugary drink/food. Some corroboration to this concept could be the tracking of sweet taste threshold (in mM) to the blood glucose

levels (in mg/dL). If salivary glucose levels could have been estimated in these patients, this framework could have received more credence. However, estimation of salivary

glucose is not a routinely available or a clinically relevant test, and this constrains our hypothesis. The association between glycemic control and the threshold for taste perception was also explored in a study by Catamo et al., although instantaneous blood glucose levels were not similarly assessed.²¹

Our results also showed a significant attenuation of sweet taste perception in diagnosed chronic diabetic patients, which is congruent with existing literature. There are several hypotheses that explain the relation of the smell and taste impairments with diabetes. One hypothesis is hyposalivation, involving qualitative and quantitative reductions in salivary production and reduced salivary flow, possibly induced by polyuria and replacement of functioning tissue in the major salivary glands by adipose tissue.^{22,23} The decreased salivary flow rate has significantly favored the growth of microorganisms such as *Candida* and *Lactobacillus* spp.²³ The other factor is deficiency of zinc as well as gustin, a zinc-binding protein which is linked to taste bud abnormalities.²⁴ In diabetes, there is decreased absorption of zinc from the gastrointestinal system and increased urinary excretion.²² There is frequent reporting of a strong association between diabetic polyneuropathy and taste impairment.²⁵ Gondivkar et al. showed that type 2 diabetes mellitus (T2DM) patients have impaired taste sensation for sweet and salt tastes, while it was not different for the other tastes—sour, bitter, and umami.²⁶ There are studies that looked at the salivary hormones, especially the leptin levels, in T2DM. Leptin levels were positively correlated with glycated hemoglobin (HbA1c) levels and were found to suppress sweet sensation. Increased glucose metabolism is also linked to altered taste sensation. The intracellular glucose in hyperglycemic state elevates the

concentration of advanced glycosylation end products (AGEs). The elevated diacylglycerol activates protein kinase C (PKC) which can modify the transcription rates of genes for extracellular matrix proteins in neurons and possibly lead to neuropathy.²⁷ A recent study showed that T2DM subjects, even with good glycemic control, with no evidence of neuropathy, retinopathy, or nephropathy, also showed a decreased sensitivity to sweet taste.²⁸

A systematic review by Peinado et al. looked at 19 studies that explored the association linking obesity and taste sensation. An increase in the threshold of taste perception in obese subjects was reported in 13 of the studies, with some conflicting reports on the intensity of the taste perceived.²⁹ The review also mentioned the difficulty in standardizing the taste threshold assessments and the non-uniformity of the subject criteria followed in the various studies. The decreased taste sensitivity to glucose could increase the amount of sugar uptake to produce a desired effect. This taste alteration could negatively affect the subjects' compliance to follow a controlled diet leading to suboptimal glycemic control and an increased tendency to the development of obesity. Taste recognition thresholds were higher in uncomplicated T2DM, and central obesity was significantly associated with this impairment. Diet-induced obesity in rodents showed increased secretion of glucagon-like peptide-1 (GLP-1) in lingual taste buds, possibly explaining their observed preference for sweet taste.³⁰

CONCLUSION

We explored the intricate relationship between the blood glucose and the perception of sweet taste. An increase in blood glucose was consistently associated with a higher threshold for perception of sweet taste. Since this was a cross-sectional study, we are unable to derive the causal relationship between the two studied parameters. But, given that the sweet taste is attenuated in patients with high blood glucose, it would seem plausible that there is a tendency towards increased glucose consumption in an *ad libitum* situation. This, in turn, would exacerbate insulin resistance and further compound the pathophysiology. On

the other hand, good glycemic control could decrease glucose intake, due to disinhibited perception of sweet taste. In this context, awareness of the relationship between instantaneous blood glucose and sweet taste perception may result in better compliance with a glucose-restricted diet.

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