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# Taste genomics and type 2 diabetes mellitus: a systematic qualitative meta-synthesis

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## Abstract

**Background:** Emerging evidence suggests that genetic variations in taste receptor genes may influence dietary behaviors, energy homeostasis, and metabolic risk, contributing to type 2 diabetes mellitus (T2DM) pathogenesis. The objective of this study is to evaluate the association between single nucleotide polymorphisms (SNPs) in taste receptor genes and T2DM.

**Methods:** This systematic review followed the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) 2020 guidelines and was registered with the International Prospective Register of Systematic Reviews (PROSPERO; CRD42022351880). A comprehensive literature search was conducted across PubMed, ScienceDirect, Cochrane Library, and Google Scholar through June 2025. Original studies examining SNPs in taste receptor genes among individuals with T2DM were included. Quality assessment was performed independently by using the Newcastle-Ottawa scale.

**Results:** Sixteen studies involving diverse populations met the inclusion criteria. Significant associations with T2DM were observed for SNPs in type 2 taste receptor gene family R member 3 (*TAS2R3*; rs11763979), *TAS2R4* (rs2233998), *TAS2R7*, *TAS2R9*, *TAS2R38*, *TAS2R50*, cluster determinant 36 (*CD36*; rs1761667, rs3211956, rs7755), carbonic anhydrase VI gene (*CA6*; rs2274327), transient receptor potential vanilloid-1 (*TRPV1*; rs161364, rs8065080), transient receptor potential cation channel subfamily M gene member 5 (*TRPM5*; rs4929982), and *TRPM8* (rs12472151). These polymorphisms may alter taste perception and gut hormone responses [e.g., glucagon-like peptide 1 (GLP-1)], affecting dietary intake, satiety, insulin secretion, and glucose regulation.

**Discussion:** The findings suggest that genetic variations in taste receptor genes may contribute to T2DM through behavioral and metabolic mechanisms. Incorporating gustatory phenotyping with genotypic profiling could enable personalized dietary strategies and inform novel therapeutic approaches targeting taste-mediated gut signaling. Further large-scale, multi-ethnic, and mechanistic studies are warranted to confirm these associations and elucidate their clinical implications.

## Keywords

Type 2 diabetes mellitus, taste receptors, taste gene polymorphism, genetic risk, dietary behavior

## Introduction

Over the past few decades, the global prevalence of type 2 diabetes mellitus (T2DM) has increased markedly, with projections estimating up to 7,862 cases per 100,000 individuals [1]. This upward trend may be attributed to a complex interplay of genetic and environmental risk factors, including obesity, insulin resistance, metabolic dysfunction, dietary habits, and epigenetic modifications [1]. In addition to these well-established risk factors, emerging evidence suggests that behavioral psychology and impaired satiety signaling also play a significant role in the pathogenesis of T2DM [2–4].

Recent studies have identified that single nucleotide polymorphisms (SNPs) in taste genes are significantly associated with elevated risk of metabolic syndrome, diabetes mellitus, obesity, carcinogenesis, Alzheimer's disease, Parkinson's disease, thyroid dysfunction, and substance use disorders [3–5]. Taste perception and signal transduction across the six taste modalities—sweet, salt, sour, bitter, umami, and fat taste—are mediated by various taste receptor genes such as type 1 taste receptor gene family R (*TAS1R*), *TAS2R*, sodium channel epithelial 1 (*SCNN1*), cluster determinant 36 (*CD36*), transient receptor potential cation channel subfamily M gene (*TRPM*), guanine nucleotide binding protein subunit alpha transducing 3 (*GNAT3*), carbonic anhydrase VI gene (*CA6*), *IZUMO* sperm-egg fusion 1 gene (*IZUMO1*), metabotropic glutamate receptor 1 gene (*GRM1*), and polycystic kidney disease (PKD)-like genes (e.g., *PKD1L3*, *PKD2L1*, and *PKD2L3*) [4–7]. Each taste modality is regulated by specific taste receptor genes. For instance: sweet taste is primarily mediated by *TAS1Rs*, *TRPMs*, and *GNAT3*; bitter taste by *TAS2Rs*, *TRPMs*, and *CA6*; salt taste by transient receptor potential vanilloid-1 (*TRPV1*; nerve endings) and *SCNN1s* (subunits alpha, beta, gamma, and delta); sour taste by *TAS1Rs* and PKD-like genes; umami taste by *TAS1Rs*, *TRPMs*, *GNAT3*, and *GRM1*; and fat taste by *CD36* and *IZUMO1* [5–7]. Taste perception begins with the interaction of food containing particular taste stimuli with oral and extra-oral taste receptors, triggering intracellular calcium release into the cytoplasm, depolarization of afferent nerve fibers and signal transduction via cranial nerves (facial nerve, glossopharyngeal nerve, sensory vagal afferents, trigeminal nerve and the trigeminal ganglion) to the central processing centers (nucleus of solitary tract, ventral posteromedial thalamic nucleus, the operculum, insular and the somatosensory cortex) [8]. These brain regions also influence gut hormone secretion [e.g., ghrelin, glucagon-like peptide 1 (GLP-1), glucose-dependent insulin-releasing peptide], thereby regulating satiety and energy homeostasis [8]. Genetic variations in taste receptors may impair this signaling cascade, leading to altered taste perception, eating behavior, impaired energy homeostasis, and increased susceptibility to T2DM [5]. Notably, *TAS1R* expression in the gut is upregulated in response to hyperglycemia in individuals with T2DM [9]. This suggests that modulating taste receptor pathways may have therapeutic potential, particularly through agents that mimic GLP-1 receptor agonists [2, 8].

Several studies have reported significant taste impairments in individuals with diabetes mellitus [10, 11]. Chamoun et al. [12] demonstrated associations between psychophysical measures of taste and 94 SNPs across 11 taste receptor genes, particularly those related to sweet, salty, umami, and fat taste perception. A recent review further highlighted the critical role of taste receptor function in the pathophysiology of T2DM and energy homeostasis [13].

Despite the growing body of evidence, no qualitative meta-synthesis has systematically evaluated the association between SNPs in taste receptor genes and T2DM. This study aims to evaluate the evidence on the association of taste gene polymorphisms and T2DM.

## Materials and methods

This qualitative meta-synthesis was registered with the International Prospective Register of Systematic Reviews (PROSPERO; Registration No. CRD42022351880) [14] and conducted in accordance with the Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) 2020 guidelines.

### Search strategy and selection criteria

A comprehensive literature search was performed across PubMed, ScienceDirect, Cochrane Library databases, and Google Scholar using the keywords "Diabetes mellitus" AND ("taste receptor gene" OR "taste gene" OR "taste gene polymorphisms" OR "taste gene mutations"). Manual screening of reference lists and citation tracking of relevant articles was also conducted to ensure thorough coverage of the literature up to June 2025. Inclusion criteria: Original research articles investigating SNPs in taste receptor genes among patients with T2DM were included. Exclusion criteria: Articles not relevant to the study, including review articles, case reports, editorials, consensus statements, clinical guidelines, conference abstracts, and book chapters, were excluded. Titles and abstracts were screened to identify potentially relevant records. Full-text screening of the identified records was assessed for inclusion by one reviewer and independently reassessed by the reviewer. The PICO framework for this study are as follows: (1) population: individuals diagnosed with T2DM, (2) intervention/exposure: presence of SNPs or variant genotypes in taste receptor genes, (3) comparison: comparison between individuals with variant versus wild-type genotypes, and (4) outcome: association between taste receptor polymorphisms and T2DM.

### Data extraction and quality assessment

Relevant study characteristics were systematically extracted, including first author, year of publication, study design, country of origin, sample size, age of participants, diagnostic criteria for T2DM, taste receptor genotyping, and other characteristic features, from studies that met the inclusion criteria. Methodological quality assessment of the included studies was carried out with the Newcastle-Ottawa scale (Table S1) [3, 4]. Data extraction was performed by one reviewer and cross-verified by another reviewer.

## Results

The PRISMA flowchart (Figure 1) illustrates the selection process. A total of 5,712 records were identified through database searches and manual screening. After title and abstract screening, followed by full-text review, sixteen studies met the eligibility criteria and were included in the qualitative meta-synthesis. Four studies were excluded due to the inclusion of populations other than T2DM (one study was conducted in individuals with prediabetes [15], and three studies were conducted in patients with gestational diabetes mellitus (GDM) [16–18]).

The characteristics of the included studies were summarized in Table 1 and Table S1. Diagnostic criteria for T2DM were based on the definitions used within each included study. Methodological quality assessment of the included studies by the Newcastle-Ottawa scale indicated that all sixteen studies were of good quality (Table S1).

The results revealed that SNPs in taste genes including *TAS2R3* gene (SNP rs11763979), *TAS2R4* gene (SNP rs2233998), *TAS2R7* gene (SNPs rs2588350 and rs619381), *TAS2R9* gene (SNP rs3741845), *TAS2R38*, *TAS2R50* gene (SNP rs6488334), *TRPV1* gene (SNPs rs161364 and rs8065080), *CD36* gene (SNPs rs1761667, rs3211956, rs7755, rs1049673, and rs1527479), and *CA6* gene (SNP rs2274327) were significantly associated with patients with T2DM. Additionally, *TRPM5* gene (SNP rs4929982) and *TRPM8* gene (SNP rs12472151) polymorphisms were significantly associated with metabolic syndrome, including T2DM.

## Discussion

The findings suggest that SNPs in several taste-related genes, including *TAS2R3*, *TAS2R4*, *TAS2R7*, *TAS2R9*, *TAS2R38*, *TAS2R50*, *TRPV1*, *CD36*, *CA6*, *TRPM5*, and *TRPM8*, may contribute to the etiology of T2DM and its associated metabolic complications. Although the precise mechanisms remain to be elucidated, current evidence supports the hypothesis that genetic variations in taste genes influence taste stimuli perception, individual food preferences, nutrient intake, and eating behavior, thereby increasing the risk of T2DM [22].

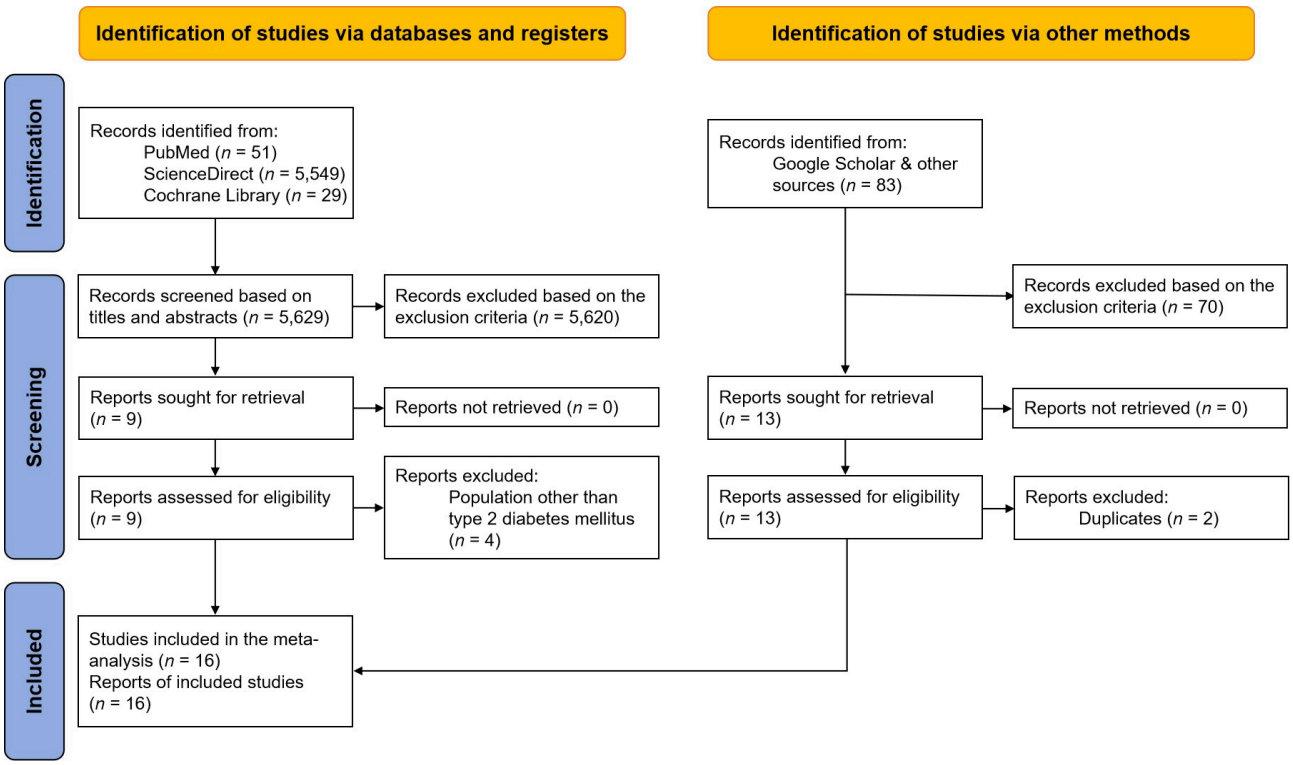


**Figure 1. Flow chart summarizing the selection process.** Adapted from [19], © 2021, The Author(s) (CC BY 4.0)

**Table 1. General characteristics of the studies included in the qualitative meta-synthesis**

| Study | Study design (country) | Population | Genotyping | Inference |
|---|---|---|---|---|
| Leprêtre et al. [20], 2004 | Cohort (France) | 454 | *CD36* gene (entire sequence) | No significant association was found between *CD36* gene and T2DM ($P > 0.1$) |
| Corpeleijn et al. [21], 2006 | Cohort (Netherlands) | 151 | *CD36* gene SNP rs1527479 and 478 C/T substitution | *CD36* gene SNP rs1527479 TT haplotype was significantly associated with T2DM ($P = 0.035$), fasting glucose concentration ($P < 0.05$) and insulin resistance ($P < 0.05$) |
| Dotson et al. [22], 2008 | Case-control (USA) | 503 | 70 SNPs in *TAS1Rs* and *TAS2Rs* gene subtypes | *TAS2R3* gene SNP rs11763979 ($P = 0.03$), *TAS2R7* gene SNPs rs2588350 ($P = 0.0007$) and rs619381 ($P = 0.009$), *TAS2R9* gene SNP rs3741845 ($P = 0.005$), and *TAS2R50* gene SNP rs6488334 ($P = 0.04$) were significantly associated with patients with T2DM |
| Banerjee et al. [23], 2010 | Case-control (India) | 400 | Two SNPs (rs1527483 and rs1761667) in the *CD36* gene | *CD36* SNP (G>A) rs1761667 GA was significantly associated with patients with T2DM ($P < 0.001$) |
| Wang et al. [24], 2012 | Case-control (China) | 113 | *CD36* gene SNPs (rs1527483 and rs1049673) | *CD36* SNP rs1049673 CG & GG haplotypes were significantly associated with impaired glucose tolerance ($P = 0.023$) and T2DM ($P = 0.011$) in patients with essential hypertension, respectively |
| Gautam et al. [25], 2015 | Case-control (India) | 100 | Six *CD36* SNPs (rs1984112, rs1761667, rs1527479, rs3211938, rs1527483, and rs3212018) | *CD36* SNPs rs1761667 (G>A) and rs3211938 (T>G) showed significant association with T2DM (GAATTC1, $P < 0.001$) |
| Tabur et al. [26], 2015 | Case-control (Turkey) | 308 | 25 *TRPM1–8* gene SNPs rs28441327, rs11070811, rs2241493, rs111649153, rs1618355, rs1328142, rs3760663, rs34364959, rs4929982, rs886277, rs34551253, rs3986599, rs3750425, rs62569677, rs55924090, rs1016062, rs2362294, rs2362295, rs10490018, rs2052029, rs6431648, rs10803666, rs12472151, rs2215173, and rs6740118 | *TRPM5* gene SNP rs4929982 A allele ($P = 0.0019$) and *TRPM8* gene SNP rs12472151 C allele ($P < 0.001$) polymorphisms might be related to the individual susceptibility to metabolic syndrome (including T2DM) |

**Table 1. General characteristics of the studies included in the qualitative meta-synthesis** (*continued*)

| Study | Study design (country) | Population | Genotyping | Inference |
|---|---|---|---|---|
| Park et al. [27], 2016 | Cohort (South Korea) | 8,842 | 7 SNPs in *TRPV1* gene such as SNPs rs161364, rs8065080, rs150908, rs222745, rs7217945, rs222741, and rs2737141 | *TRPV1* gene SNPs rs161364 C allele ($P$ = 0.0487) and rs8065080 C allele ($P$ = 0.0378) were significantly associated with the prevalence of T2DM in dominant genetic models |
| Zhang et al. [28], 2018 | Case-control (China) | 546 | Four *CD36* SNPs rs1194197, rs2151916, rs3211956, and rs7755 | Overweight/obesity individuals carrying SNP variant alleles of rs3211956 (GG+GT, $P$ = 0.024) and rs7755 (AA+AG, $P$ = 0.007) were associated with increased risk of T2DM compared to normal weight individuals carrying wild-type homozygous alleles |
| Fujii et al. [29], 2019 | Cross-sectional (Japan) | 495 | Two *CD36* gene SNPs (rs1761667 and rs1527483) | *CD36* gene SNP rs1761667 AA haplotype was associated with higher intake of total fat ($P$ = 0.01) and monounsaturated fatty acids ($P$ = 0.05) when compared to GG and GA haplotypes. In addition, the frequency of *CD36* gene SNP rs1761667 GG haplotype was higher in T2DM |
| Mrag et al. [30], 2020 | Cohort (Tunisia) | 300 | *CA6* gene SNP rs2274327 | The *CA6* gene SNP rs2274327 T allele in its dominant model (TT+CT vs. CC, 67.7% vs. 32.3%) was increasingly associated with T2DM. Similarly, taste impairment in T2DM was significantly associated with *CA6* gene SNP rs2274327 T allele in its dominant model (OR = 1.97 [95% CI = 1.21 to 3.23], $P$ = 0.006) |
| Hatmal et al. [31], 2021 | Case-control (Jordan) | 350 | *CD36* gene rs1761667 (G>A) and rs1527483 (C>T) were genotyped | No significant association was observed between *CD36* polymorphisms and patients with T2DM or dyslipidemia ($P$ > 0.1) |
| Touré et al. [32], 2022 | Cross-sectional (Senegal) | 100 | 2 tag SNPs in *CD36* (rs3211867 and rs1761667) | No significant difference was observed between controls and T2DM subjects ($P$ = 0.9) |
| Franzago et al. [33], 2023 | Cohort (Italy) | 23 | *CD36* gene SNPs rs1984112 (A>G) and rs1761667 (G>A), *BMAL1* gene SNP rs7950226 (G>A), and *CLOCK* gene SNPs rs1801260 (A>G), rs4864548 (A>G), and rs3736544 (G>A) | *CD36* gene SNP rs1761667 (G>A) A allele in its dominant form (AA+GG genotype) was significantly associated with patients with T2DM ($P$ = 0.001) |
| Lee and Shin [34], 2023 | Cohort (Korea) | 4,552 | *TAS2R4* SNP rs2233998 | *TAS2R4* SNP rs2233998 TT haplotype was significantly associated with the incidence of T2DM in women (HR [95% CI] = 1.48 [1.13–1.93], $P$ = 0.0182) |
| Husami et al. [35], 2025 | Case-control (India) | 680 | 2,658 gene variants | *TAS2R38* genetic variants were associated with an increased risk of T2DM ($P$ < 0.05) |

case: patients with T2DM as defined in the respective studies; control: normal healthy individuals as defined in the respective studies; *CD36*: cluster determinant 36; T2DM: type 2 diabetes mellitus; SNP: single nucleotide polymorphism; *TAS1R*: type 1 taste receptor gene family R; *TRPM1*: transient receptor potential cation channel subfamily M gene member 1; *TRPV1*: transient receptor potential vanilloid-1; *CA6*: carbonic anhydrase VI gene; OR: odds ratio; CI: confidence intervals; *BMAL1*: brain and muscle aryl hydrocarbon receptor nuclear translocator-like protein-1; *CLOCK*: circadian locomotor output cycles kaput; HR: hazard ratio

For instance, *TAS2R9* is expressed in gut entero-endocrine L-cells and mediates GLP-1 secretion in response to sweet taste stimuli [22]. Canivenc-Lavier et al. [13] recently described local glucose-dependent GLP-1 secretion by taste bud cells and proposed taste receptors as potential targets for T2DM treatment [3]. Similarly, reduced *CD36* expression due to genetic polymorphisms may influence fat taste sensitivity and intake behaviors, possibly exerting a protective effect due to reduced fat taste sensitivity [4, 33]. Variations in the *TRPM5* gene are associated with reduced GLP-1 levels, impaired insulin release, and altered glucose tolerance, highlighting the metabolic relevance of taste receptor polymorphisms [3, 22].

This is in line with previous observations that the taste-dependent manner of GLP-1 secretion, glucose-stimulated insulin secretion, and insulin sensitivity in patients with T2DM might be associated with the polymorphisms in taste receptor genes [3, 22, 26, 33]. These genetic variations may affect hunger signaling, gut motility, and taste-driven satiety hormone release [3, 4, 13]. For example, individuals carrying the *TAS2R38* proline-alanine-valine (PAV) allele [a 6-n-propylthiouracil (PROP) taster genotype] exhibit heightened sensitivity to bitter compounds, which may impact food choices and contribute to dietary avoidance of bitter but nutritionally beneficial foods [4, 36–38]. Such phenotypic taste differences can now be identified using validated clinical gustatory tests (e.g., taste strips, solutions), which could help screen individuals at risk of metabolic diseases, including obesity and diabetes [3, 4, 36]. Moreover, pharmacologic agents that modulate the taste receptor expression based on an individual's genotype may offer a novel therapeutic avenue [37]. Nevertheless, further large-scale molecular and clinical studies are warranted to validate these associations and uncover the underlying mechanisms driving gene-diet interactions in T2DM.

### Strengths and limitations

Overall, this study provides the first systematic synthesis of the association between taste receptor gene polymorphisms and T2DM. However, several limitations exist: (1) high methodological heterogeneity among included studies; (2) potential selection and publication biases; (3) lack of randomized controlled trials; and (4) insufficient data for meta-analysis. Allele frequency variations across ethnicities further limit generalizability. Due to insufficient data to conduct a statistical analysis for all the genes included in the present study, the meta-analysis was not feasible. To address the potential heterogeneity in genotyping methods, variability in study designs, population characteristics, and definitions of T2DM in the included studies, Table 1 and Table S1 summarize the comparison of the characteristics of the included studies, including study design, sample size, country or location of study, and main findings. The risk of bias was assessed using the Newcastle-Ottawa scale, and narrative synthesis and reporting followed the PRISMA 2020 guidelines. Further, citation searches and gray literature searches were performed and a predetermined inclusion and exclusion criteria based on the PROSPERO protocol was used to select the studies for inclusion.

### Future perspectives

Further molecular and clinical studies, particularly randomized controlled trials and large, multi-ethnic cohort studies, are warranted to validate the role of taste receptor gene polymorphisms in the pathophysiology of T2DM. Gustatory testing to assess taste phenotypes, when paired with genotypic profiling, may offer a cost-effective approach to identify at-risk individuals and guide personalized dietary interventions. Exploration of taste modulators and GLP-1 analogs targeting specific taste receptors may open new therapeutic avenues in obesity and T2DM management. Moreover, SNPs in taste genes could serve as genetic markers for early detection and risk stratification.

### Conclusion

In summary, this review provides evidence that SNPs in taste receptor genes are associated with T2DM. Altered taste perception and signal transduction may influence eating behavior, energy homeostasis, and glucose metabolism. Identifying taste phenotypes and targeting taste receptor gene expression may represent promising strategies for the prevention and treatment of T2DM. However, larger, well-designed studies are needed to confirm these associations and facilitate their clinical translation.

## Abbreviations

*CA6*: carbonic anhydrase VI gene

*CD36*: cluster determinant 36

GLP-1: glucagon-like peptide 1

*GNAT3*: guanine nucleotide binding protein subunit alpha transducing 3

*GRM1*: metabotropic glutamate receptor 1 gene

*IZUMO1*: *IZUMO* sperm-egg fusion 1 gene

PKD: polycystic kidney disease

PRISMA: Preferred Reporting Items for Systematic reviews and Meta-Analyses

PROSPERO: International Prospective Register of Systematic Reviews

*SCNN1*: sodium channel epithelial 1

SNPs: single nucleotide polymorphisms

T2DM: type 2 diabetes mellitus

*TAS1R*: type 1 taste receptor gene family R

*TRPM*: transient receptor potential cation channel subfamily M gene

*TRPV1*: transient receptor potential vanilloid-1

## Supplementary materials

The supplementary materials for this article are available at: https://www.explorationpub.com/uploads/Article/file/101439_sup_1.pdf.

## Declarations

### Author contributions

VS: Conceptualization, Visualization, Methodology, Investigation, Data curation, Writing—original draft, Writing—review & editing. SK: Writing—review & editing. VH: Writing—review & editing.

### Conflicts of interest

Part of the manuscript has been presented as a poster presentation in the International Diabetes Federation Congress 2025 and the preparation of this manuscript has not been influenced by the presentation or any other factors.

### Ethical approval

Not applicable.

### Consent to participate

Not applicable.

### Consent to publication

Not applicable.

### Availability of data and materials

The raw data supporting the conclusions of this manuscript will be made available by the authors, without undue reservation, to any qualified researcher.

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