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ASSOCIATION OF TAS2R38 DIPLOTYPES WITH SUGAR PREFERENCE AND DENTAL CARIES EXPERIENCE IN ADULTS

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ABSTRACT

Dental caries is a multifactorial disease in which genetic variation may influence dietary behavior and host susceptibility. This cross-sectional observational analysis evaluated the association of TAS2R38 diplotypes with bitter-taste perception, dietary sugar preference, estimated daily sugar intake, and dental caries experience in 50 adults aged 18–65 years. TAS2R38 diplotypes were classified as PAV/PAV, PAV/AVI, or AVI/AVI, while PROP bitterness, sugar preference, daily sugar intake, and DMFT were evaluated as phenotypic and clinical outcomes. PAV/PAV, PAV/AVI, and AVI/AVI diplotypes occurred in 12 (24.0%), 29 (58.0%), and 9 (18.0%) participants, respectively. Increasing PAV dosage was strongly associated with greater PROP bitterness (Spearman $\rho = 0.793$, $P < 0.001$), lower sugar preference ($\rho = -0.680$, $P < 0.001$), and lower daily sugar intake ($\rho = -0.654$, $P < 0.001$). Mean sugar preference scores were 4.21, 5.97, and 8.12 in PAV/PAV, PAV/AVI, and AVI/AVI participants, respectively, while corresponding mean sugar intakes were 65.7, 86.6, and 105.1 g/day. DMFT values were 5.50, 6.62, and 7.00, respectively, but the between-genotype difference was not statistically significant ($P = 0.426$). In multivariable analysis, daily sugar intake was positively associated with DMFT, whereas TAS2R38 PAV dosage was not independently associated with DMFT after adjustment for sugar intake, age, oral hygiene, and salivary flow. These findings suggest that TAS2R38 variation may influence caries-related dietary behavior more consistently than caries experience itself.

KEYWORDS: TAS2R38; dental caries; DMFT; bitter taste; sugar preference; genetic polymorphism

INTRODUCTION

Dental caries remains one of the most prevalent oral diseases worldwide and continues to represent a major population health burden despite substantial advances in prevention and treatment [1]. Dental caries results from a dynamic interaction among host susceptibility, oral microorganisms, dietary exposure, salivary factors, oral hygiene, and environmental determinants [2-5]. Genetic factors may contribute to differences in caries susceptibility by modifying enamel characteristics, salivary composition, immune responses, microbial interactions, and dietary behavior [6-10].

Dietary sugar is one of the most important modifiable determinants of dental caries. Fermentable sugars provide substrate for acidogenic and aciduric microorganisms, leading to repeated reductions in plaque pH and progressive demineralization [11,12]. Epidemiological evidence demonstrates a positive relationship between sugar exposure and caries, with both the quantity and frequency of sugar consumption contributing to disease development [13-16].

Individual differences in food choice are partly influenced by taste perception. Among bitter taste receptors, taste receptor type 2 member 38 (TAS2R38) is one of the best-characterized genes influencing sensitivity to phenylthiocarbamide (PTC) and 6-n-propylthiouracil (PROP) [17-25]. Three common nonsynonymous variants of TAS2R38, rs713598, rs1726866, and rs10246939, generate the major PAV and AVI haplotypes [22-26]. Individuals carrying PAV/PAV generally demonstrate greater sensitivity to PROP, whereas AVI/AVI individuals are more likely to exhibit low bitterness sensitivity [22-25].

Taste-related genetic variation may influence dietary choices. Studies have demonstrated associations between TAS2R38 variants and preferences or consumption of several foods, although the strength and direction of

associations vary between populations [24,28-31]. Bitter taste receptors are also expressed in oral epithelial tissues and may participate in host–microbial interactions and innate immune responses [19,20,32]. Experimental evidence has shown genotype-dependent TAS2R38 responses to Streptococcus mutans, suggesting a possible biological mechanism linking TAS2R38 variation with oral health [32].

Previous clinical studies have reported inconsistent relationships between TAS2R38-related bitter taste sensitivity and dental caries. Some pediatric studies have reported greater caries experience among PROP non-tasters, whereas adult studies have failed to demonstrate a statistically significant association between TAS2R38 diplotypes and tooth decay [37-40]. The relationship between TAS2R38 genotype, sugar preference, actual sugar intake, and dental caries has therefore not been adequately characterized in adults.

The present study investigated whether TAS2R38 diplotypes were associated with PROP bitterness, sugar preference, estimated daily sugar intake, and DMFT among adults. We hypothesized that greater TAS2R38 bitter-taste sensitivity would be associated with lower sugar preference and lower sugar intake and that these dietary differences would be reflected in lower caries experience.

MATERIAL AND METHODS

Study design and participants

This study was designed as a cross-sectional observational analysis of an anonymized dataset containing 50 adults aged 18–65 years. The dataset included demographic, TAS2R38 genotype, taste phenotype, dietary, oral-health, salivary, and dental caries variables.

Study setting and recruitment period: [INSERT ACTUAL STUDY SITE AND STUDY DATES].

Participants were eligible for inclusion if they were adults represented in the analytical dataset and had complete information for TAS2R38 diplotype, PROP bitterness score, sugar preference, daily sugar intake, and DMFT. The supplied dataset contained complete observations for all 50 participants.

Ethics approval: [INSERT NAME OF INSTITUTIONAL ETHICS COMMITTEE AND APPROVAL NUMBER]. The study should be reported as conforming to the Declaration of Helsinki and applicable institutional requirements for human research. No identifiable participant information was used in the analysis.

Clinical and phenotypic variables

The primary genetic exposure was TAS2R38 diplotype, categorized as PAV/PAV, PAV/AVI, or AVI/AVI. For dose-response analyses, PAV dosage was coded as 0 for AVI/AVI, 1 for PAV/AVI, and 2 for PAV/PAV.

Taste phenotype was categorized as super-taster, taster, or non-taster. PROP bitterness was recorded on a 0–10 scale, with higher values indicating greater perceived bitterness.

Dietary outcomes included a sugar preference score ranging from 0 to 10 and estimated daily free sugar intake expressed in grams/day. Higher sugar preference scores indicated greater preference for sweet foods.

Dental caries experience was assessed using the DMFT index, calculated from the number of decayed (D), missing (M), and filled (F) teeth. Additional variables included age, sex, body mass index (BMI), toothbrushing frequency, flossing habit, oral hygiene score, salivary flow rate, salivary pH, and smoking status.

Statistical analysis

Continuous variables were summarized as mean ± standard deviation and median where appropriate, while categorical variables were summarized as frequency and percentage.

The distribution of TAS2R38 diplotypes was assessed, and Hardy–Weinberg equilibrium was evaluated as a descriptive genetic quality-control analysis. Differences among genotype groups were evaluated using the Kruskal–Wallis test because of the modest group sizes and the ordinal/non-normally distributed nature of several outcomes.

Spearman rank correlation was used to assess relationships between PAV dosage and PROP bitterness, sugar preference, daily sugar intake, and DMFT. Associations between sugar preference and daily sugar intake were also examined.

Multivariable ordinary least-squares regression was used to examine independent associations with DMFT. The primary model included PAV dosage, daily sugar intake, age, oral hygiene score, and salivary flow rate. A separate regression model evaluated the relationship between PAV dosage and sugar preference while adjusting for age, BMI, and sex.

Two-sided P values <0.05 were considered statistically significant. Statistical analyses were performed using Python-based statistical procedures.

RESULTS

Participant characteristics

The study included 50 adults with a mean age of 43.52 ± 13.40 years. There were 25 males and 25 females. The mean BMI was 24.41 ± 3.65 kg/m². The mean PROP bitterness score was 5.07 ± 2.23, mean sugar preference score was 5.94 ± 1.88, and mean daily sugar intake was 84.92 ± 20.01 g/day. The mean DMFT index was 6.42 ± 2.70.

Twenty-five participants (50.0%) were classified as tasters, 15 (30.0%) as non-tasters, and 10 (20.0%) as super-tasters.

TAS2R38 diplotype distribution

PAV/AVI was the most frequent diplotype, occurring in 29 participants (58.0%), followed by PAV/PAV in 12 (24.0%) and AVI/AVI in 9 (18.0%). The estimated PAV allele frequency was 0.53, while the AVI allele frequency was 0.47. The observed diplotype distribution did not significantly deviate from Hardy–Weinberg expectations ($\chi^2 = 1.35$, $P = 0.246$).

PROP phenotype closely corresponded to diplotype. All nine AVI/AVI participants were classified as non-tasters. Among PAV/AVI participants, 23 (79.3%) were classified as tasters and six (20.7%) as non-tasters. Among PAV/PAV participants, 10 (83.3%) were super-tasters and two (16.7%) were tasters.

TAS2R38 diplotype and bitter-taste perception

A strong positive association was observed between PAV dosage and PROP bitterness score (Spearman $\rho = 0.793$, $P < 0.001$).

Mean PROP bitterness scores were 7.73 ± 1.27 for PAV/PAV, 4.84 ± 1.53 for PAV/AVI, and 2.28 ± 0.82 for AVI/AVI participants. The overall genotype-group difference was statistically significant (Kruskal–Wallis $H = 30.83$, $P < 0.001$).

TAS2R38 diplotype and sugar preference

Mean sugar preference scores were 4.21 ± 1.16 for PAV/PAV, 5.97 ± 1.57 for PAV/AVI, and 8.12 ± 1.14 for AVI/AVI participants. The overall difference was statistically significant (Kruskal–Wallis $H = 22.81$, $P < 0.001$). PAV dosage demonstrated a strong inverse correlation with sugar preference (Spearman $\rho = -0.680$, $P < 0.001$).

In a multivariable regression model adjusted for age, BMI, and sex, each additional PAV allele was associated with an approximately 1.99-point lower sugar preference score ($\beta = -1.99$, 95% CI −2.60 to −1.38, $P < 0.001$). The model explained approximately 50.6% of the variance in sugar preference.

TAS2R38 diplotype and daily sugar intake

Mean estimated daily sugar intake was 65.67 ± 13.91 g/day among PAV/PAV participants, 86.64 ± 17.02 g/day among PAV/AVI participants, and 105.06 ± 12.26 g/day among AVI/AVI participants.

The difference among genotype groups was statistically significant (Kruskal–Wallis $H = 20.97$, $P < 0.001$). PAV dosage was inversely correlated with daily sugar intake (Spearman $\rho = -0.654$, $P < 0.001$).

In multivariable analysis adjusted for age and BMI, each additional PAV allele was associated with a reduction of approximately 19.81 g/day in estimated sugar intake ($\beta = -19.81$, 95% CI −26.61 to −13.02, $P < 0.001$).

Sugar preference and estimated daily sugar intake were strongly correlated (Spearman $\rho = 0.823$, $P < 0.001$).

TAS2R38 diplotype and dental caries experience

Mean DMFT scores were 5.50 ± 3.23 for PAV/PAV, 6.62 ± 2.72 for PAV/AVI, and 7.00 ± 1.58 for AVI/AVI participants.

Although the mean DMFT increased progressively with decreasing PAV dosage, the overall genotype-group difference was not statistically significant (Kruskal–Wallis $H = 1.71$, $P = 0.426$). PAV dosage was also not significantly correlated with DMFT (Spearman $\rho = -0.179$, $P = 0.213$).

Determinants of DMFT

In the multivariable DMFT model including PAV dosage, daily sugar intake, age, oral hygiene score, and salivary flow rate, the overall model was statistically significant ($R^2 = 0.453$; adjusted $R^2 = 0.391$; $P < 0.001$).

Daily sugar intake showed a positive independent association with DMFT ($\beta = 0.080$ DMFT units per g/day, 95% CI 0.040–0.119, $P < 0.001$). Age was also positively associated with DMFT ($\beta = 0.072$ per year, 95% CI 0.027–0.118, $P = 0.003$), whereas better oral hygiene was associated with lower DMFT ($\beta = -0.696$, 95% CI −1.320 to −0.071, $P = 0.030$).

PAV dosage was not independently associated with DMFT after adjustment for these factors ($\beta = 0.578$, 95% CI −0.650–1.806, $P = 0.348$). Salivary flow rate was not independently associated with DMFT ($P = 0.173$).

DISCUSSION

The present study demonstrates a strong association between TAS2R38 diplotype and bitter-taste perception in adults and identifies a substantial gradient in sugar preference and estimated daily sugar intake across diplotype groups. In contrast, the direct association between TAS2R38 diplotype and DMFT was not statistically significant. These findings suggest that the principal observable contribution of TAS2R38 in this dataset may occur through dietary behavior rather than through a sufficiently large independent effect on accumulated caries experience.

The strong genotype–PROP relationship observed in the present analysis is consistent with the established biological role of TAS2R38 in bitter-taste perception. PAV and AVI represent the two major functional haplotypes, and the PAV

haplotype is generally associated with greater sensitivity to PROP and PTC [22-27]. The observed PROP gradient was therefore biologically coherent.

The genotype distribution observed in this dataset was also compatible with previous reports from Indian populations. Deshaware and Singhal reported substantial variation in TAS2R38 diplotype frequencies and PROP phenotypes in an Indian cohort [28]. The distribution in the present sample may reflect sampling variation, geographic differences, or the limited sample size.

A major finding was the strong inverse association between PAV dosage and sugar preference. Participants with AVI/AVI diplotypes had the highest mean sugar preference score, whereas PAV/PAV participants had the lowest. This observation is compatible with evidence indicating that genetic differences in taste perception can influence food choice and dietary behavior [29-31]. However, evidence regarding TAS2R38 and sweet-food preference is inconsistent, emphasizing the need for population-specific investigation.

The present data also showed a substantial inverse association between PAV dosage and estimated daily sugar intake. This provides a potential behavioral pathway linking bitter-taste genotype with caries-related dietary exposure. Nevertheless, dietary behavior is influenced by socioeconomic status, cultural factors, food availability, age, education, habitual dietary patterns, and other genetic and environmental factors [29-31].

The strong correlation between sugar preference and estimated sugar intake further supports the behavioral pathway observed in this dataset. Importantly, the relationship between sugar intake and DMFT remained significant after adjustment for PAV dosage, age, oral hygiene, and salivary flow. This finding is consistent with epidemiological evidence demonstrating a relationship between sugar consumption and dental caries [11-16].

The absence of a statistically significant direct association between TAS2R38 genotype and DMFT is important. Although mean DMFT increased from 5.50 in PAV/PAV participants to 7.00 in AVI/AVI participants, the difference did not reach statistical significance. This is consistent with adult studies that have failed to demonstrate a significant association between TAS2R38 diplotype and caries [40].

Conversely, previous pediatric studies have reported associations between low PROP sensitivity and increased caries experience [37-39]. Differences in age, dentition, dietary behavior, fluoride exposure, oral hygiene, caries measurement, and sample size may explain the inconsistent findings between studies.

The lack of an independent genotype effect in the multivariable DMFT model suggests that TAS2R38 should not be considered a simple deterministic marker of caries susceptibility. Dental caries is a complex phenotype involving microbial, behavioral, dietary, salivary, socioeconomic, and genetic determinants [2-10]. Recent genetic studies likewise indicate that although individual taste-related variants may influence caries risk, the overall evidence remains heterogeneous [33-36,41].

The biological plausibility of a TAS2R38–oral health relationship nevertheless remains strong. TAS2R38 is expressed in oral epithelial tissues and may participate in innate immune responses to oral microorganisms [19,20,32]. Experimental evidence has demonstrated genotype-dependent responses to Streptococcus mutans, although the present dataset did not include microbial or molecular immune measurements and therefore cannot directly test this pathway [32].

The present study also identified age and oral hygiene as independent determinants of DMFT. The positive relationship between age and DMFT is expected because DMFT represents cumulative lifetime caries experience. The inverse association between oral hygiene score and DMFT further emphasizes that behavioral factors may substantially modify the expression of any genetic susceptibility.

The clinical implications are potentially relevant to precision prevention. If replicated in larger cohorts, TAS2R38 variation could potentially contribute to risk stratification of dietary behaviors rather than serve as a standalone diagnostic marker for caries. Individuals with lower bitter-taste sensitivity and greater sugar preference could potentially benefit from intensified dietary counseling. However, such applications require prospective validation.

The present study has several limitations. First, the sample size was small, with only 50 participants and nine AVI/AVI individuals. Second, the cross-sectional design prevents causal inference. Third, estimated daily sugar intake may be affected by recall and reporting error. Fourth, DMFT is cumulative and may reflect historical exposures that are not captured by current dietary behavior. Fifth, the dataset did not contain detailed socioeconomic, fluoride, microbial, or longitudinal caries information. Sixth, the supplied dataset did not document the laboratory genotyping procedure; the exact DNA extraction, PCR, SNP assay, and quality-control procedures should therefore be added from the original laboratory protocol before submission.

Despite these limitations, the study has the strength of simultaneously evaluating genotype, taste phenotype, dietary sugar preference, estimated sugar intake, and dental caries experience. This integrated framework permits evaluation of a potential genetic–behavioral pathway rather than evaluating genetic susceptibility in isolation.

CONCLUSION

TAS2R38 diplotype was strongly associated with PROP bitterness, sugar preference, and estimated daily sugar intake in this adult sample. Individuals carrying fewer PAV haplotypes demonstrated greater sugar preference and higher estimated sugar intake. Although DMFT showed a numerical gradient across genotype groups, TAS2R38 diplotype was not independently associated with caries experience after adjustment for dietary and clinical factors. The findings

support a potential role for TAS2R38 in shaping caries-related dietary behavior but do not support its use as an independent caries susceptibility marker. Larger, multicenter prospective studies incorporating detailed dietary assessment, validated genotyping, oral microbiological profiling, and longitudinal caries outcomes are warranted.

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Conflict of interest
The authors declare no conflict of interest.

Data availability
The anonymized analytical dataset used in this study is available from the corresponding author on reasonable request, subject to applicable ethical and institutional restrictions.

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