ORIGINAL ARTICLE

Association Between TAS1R2 Gene Polymorphism (rs12033832) and Sweet Taste Perception Amongst Malay Obese and Non-obese Subjects

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ABSTRACT

Introduction: A growing evidence supported that variation of sweet taste perception, mediated by TAS1Rs gene variants could lead to excess sweetened food and beverages intake and also obesity. However, obesity development may also alter individuals' taste sensitivity and perception. Thus, it is best to further investigate whether or not the individuals' sweet taste sensitivity and acceptance are associated with variation in TAS1R2 gene and Body Mass Index (BMI) status. **Methods:** This comparison cross sectional study comprised of 88 obese and 92 non-obese subjects aged 20-45. All the subjects were genotyped for TAS1R2 gene variant at rs12033832 using polymerase chain reaction – restriction fragment length polymorphism (PCR-RFLP). Suprathreshold sensitivity for sweet taste was assessed using general Labeled Magnitude Scales. Intensity rating and hedonic test were carried out on 2 food samples (tea drink and rose flavoured agar) to examine subject's intensity rating and liking at different sugar contents. **Results:** Our results showed that rs12033832 of TAS1R2 gene is associated with sweet taste perception among obese and non-obese subjects. No interaction effect between BMI status and TAS1R2 gene variant (rs12022832) was found on sweet taste measures. Overall, non-obese subjects with AA genotype on rs12033832 had the highest sweet taste sensitivity and dislike high sugar content products the most. The effect was reverse among the obese subjects with GG homozygous. **Conclusion:** These findings suggest that TAS1R2 gene variation plays an important role in sweet taste perception among individuals and may have nutritional implications and obesity.

Keywords: Obesity, Taste perception, Sweetness, TAS1R2 gene, rs12033832

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INTRODUCTION

Obesity has become a major health issues around the globe. With the increasing number of overweight and obese people each year, there is an urgent need to develop effective and efficient measures to combat this epidemic disease. The linkage between dietary sugar intake and obesity has long been debated (1-4) . Some studies had reported on a positive relationship between weight gain and sugar intake (5), sugar - sweetened beverages consumption (6,7), sweet food preference (1,4,8) and sweet taste sensitivity (9,10). All these findings indicate a strong association between sweet food consumption and obesity. Understanding the etiology of sweet preference among individuals, whether someone have a 'sweet tooth' or not might be able to explain this phenomenon (11,12). By this way, a crucial indicator to assess individuals who have a potency in sweet preference can be produced and their application in obesity management can be tested (13).

Genetic variation could affect individuals' sweet taste perception. Finding from twin study showed that genetic variation could contributed up to 33% in sweet taste threshold and 53% for the frequency of sweet foods consumption among individuals (14). The variation of human sweet taste perception is caused by variation in sweet taste receptors genes. TAS1R2 and TAS1R3 genes were identified as major taste receptor in detecting sweet taste (15). Both genes are co-expressing in binding sweet molecules/ ligand. However, the TAS1R2 is specific to sweet taste perception while TAS1R3 is involved in the detection of umami taste when it dimerizes with TAS1R1 (16).

A few studies have examined the effect of genetic variation in TAS1R2 gene on sweet taste perception. Fushan et al.

(17) found that all the 34 TAS1R2 gene variants were not associated to sweet taste sensitivity. However, their study did not account for body mass index (BMI), which may affect sweet taste perception (18,19). Meanwhile, study by Dias et al., (2015) revealed that the variation at rs12033832 of TAS1R2 gene affects their subjects' sweet taste sensitivity and the relationship was modified by obesity status. In addition, some studies found that TAS1R2 gene variants particularly at rs12033832 were linked to sugar and dietary intake which could lead to the usage this gene variant as potential marker for taste perception, food intake and obesity (20). However, most of previous have been conducted among Caucasian healthy subject in measuring the effect of rs12033832 on sensory responses (18, 21) and food intake (22, 23). Thus, studies that focus on BMI status and its relationship to taste perception among other population are required to better understand the role of genetic variation on sweet taste among individuals. Based on this consideration, the present study was carried out to investigate whether the TAS1R2 gene variant (rs12033832) is associated with sweet taste perception between obese and non-obese Malays subjects using various taste responses.

MATERIALS AND METHODS

Study Design

Comparative cross-sectional design was employed in this exploratory study, whereas purposive sampling method was used in recruiting subjects. This study involved two main laboratory techniques: sensory test and genotyping analysis. All subjects attended two sensory sessions, which took them approximately 30 to 40 min to complete. Prior to each sensory tasting session, the subjects' anthropometries measurement and DNA samples (buccal cell) were obtained.

Subjects Recruitment and Criteria

The subjects were recruited via word of mouth, advertisement, internet postings and flyers that were distributed around the Universiti Putra Malaysia (UPM) campus in Serdang, Selangor. Power calculation was performed before commencing this study to determine the subject sample size, using G*power 3 software. 65 subjects are required for each group (obese and non obese) at 80% statistical power with a medium effect size and type I error of 0.05. The potential subjects were screened using a questionnaire once they agreed to participate in this study. The screening questions requested information regarding their background, health status and other items (e.g depression status, eating restriction, and food allergy), which reflect the inclusion and exclusion criteria of the subjects for this study, as depicted in Table I. All the screening processes were administered via email. Subjects who meet the inclusion and exclusion criteria were contacted through phone to be informed of the study's objectives and protocols. The study protocols were approved by the Ethic Committee for Research Involving Human Subjects, Research Management Centre, Universiti Putra Malaysia [Ref. No. RMC/1.4.18.1 (JKEUPM)/ F2].

Table I: Subject's inclusion and exclusion criteria for this study

Inclusion Criteria	Exclusion Criteria
Individuals with BMI of 18-25 or ≥ 30 kgm^{-2}	Had a score of ≥ 13 on Restrained Scale[a]
Individual with age between 20 and 45 years	Had a score ≥ 50 on Zung Self-rating Depression Scale[b]
Malays	Pregnant or Lactating
	Had self reported food allergy and chronic diseases such as diabetes, cardiovascular disease

[a] based on score of Restrained Scale adapted from van Strien et al. (24)
[b] based on score of Self-Rating Depression Scale adapted from Zung (25)

Anthropometric Measurement

Weight and height were measured from all subjects, where they had to wear light clothing and remove their shoes during the measurement. Their BMI was calculated using the following formula:-

$$BMI = \frac{\text{Weight in kilograms (kg)}}{\text{Height in meter square } (m^2)}$$

Body weight was measured with a weighing scale (Omron HN-288, Kyoto, Japan) and height was measured with a mechanical measuring tape (Seca 206, Hamburg, Germany). Subjects with BMI above 30kgm-2 were categorized as obese whereas those between 18.5 and 25kgm-2 were categorized as having normal weight (26).

Sensory Test

Subjects attended two different sensory sessions in this study. They were presented blank sucrose solution in first session and two food products; namely tea drink and rose flavored pudding during the second session. All the samples (blank and food products) varied in 5 different sucrose concentrations. Different levels of sucrose in blank solution were prepared based on Drewnowski et al. (27) with slight modification whereby preliminary test was conducted among 40 subjects. They were asked to rate sweetness intensity of ten concentrations of sucrose solution (ranged from 10 g/L to 350g/L) using 9-point intensity scale. Five concentrations (17, 35, 72, 145 and 285 g/L) which cover a range of the mean sweetness rating between 3 and 7 were chosen as samples for sensory test (28). Meanwhile, the selection of five different sucrose concentration of both of food products was subjected to second preliminary test using the same subjects. During this preliminary session, ten different sucrose concentrations ranged from 15 g/L to 200g/L in tea drink and 50g/L to 400 g/L in rose-flavored pudding was rated for sweetness and liking using the similar scale and procedures. Five concentrations which cover a range of palatable intensity with the mean rating between 3 and 7 were chosen as samples for sensory test (28). The chosen amount of sugar added in tea drink was

25, 40, 63, 100 and 158 g/L, for rose flavoured pudding was 120, 150, 190, 240 and 300g/L.

Tea drink was prepared by adding 2 sachet of teabag (Lipton, Malaysia) into 1L hot water. It was stirred for 1 min and the tea bag was removed before respective amount of sugar were added to the mixtures. Meanwhile, rose flavored pudding was prepared by dissolving 10g agar-agar (SCS Food Manufacturing Sdn. Bhd., Malaysia) in 1 L boiling water (100°C). Then, respective amount of sugar was added followed by 7-10 drops of rose flavored syrup and stirred for 1 min. All the mixture were filtered before it was poured into individual cup (25ml) and allowed to cool at room temperature. The rose flavored pudding was kept in a chiller (5 - 80°C) before being served to the subjects. Tea was prepared at least 1 h prior to sensory testing while the rose flavored pudding was prepared at least 24 h before testing.

In the sensory session, all the samples were coded with 3-digit numbers, arranged in randomized order and served at room temperature. Subjects were instructed to taste the samples from left to right, to rinse their mouth before and between tasting and allow 30s rest before tasting the next sample. They rated the sweetness intensity by making a vertical line on the general labelled magnitude scale (gLMS); a scale with 15 cm horizontal line that have a ranges of sensation strength descriptors from 'no sensation' as the lowest (0) to 'the strongest imaginable sensation of any kind' as the highest (15cm). On the other hand, hedonic gLMS was used with the highest and lowest scales described as the 'strongest imaginable liking' (+7.5cm) and the 'strongest imaginable disliking' (-7.5cm), with neutral zero at the center. Subjects' rating on gLMS and hedonic gLMS were measured as distance from left (gLMS) or from centre (hedonic gLMS) by using ruler and recorded in centimeter (cm) as data. All the tasting sessions was conducted under red light condition to mask any visual cues (e.g color).

Buccal Cell Collection and Genotyping

Subjects' buccal cells were collected using a cytobrush (Medical Packaging Corporation, USA) in 2 sampling occasions; prior to the sensory sessions (session 1 and 2). Cytobrushes were kept in the original packaging and sealed in the paper envelope before being transferred to the laboratory for DNA extraction. The standard commercial kit, innuPREP DNA mini kit (Analytik Jena, Germany) was used to extract the DNA based on the manufacturer's protocols. Polymorphism of rs12033832 was genotyped using polymerase chain reaction – restriction fragment length polymorphism (PCR-RFLP) method. PCR was performed in 25µl total reaction mixtures containing 1ul (10 pmol/µL)of the primers (each forward and reverse), 12.5ul ready to use 2x premix (Bioline Reagents Limited, UK), 1ul of DNA templates (100 ng/µL) and 8.5ul of sterile distilled water. The sequences of primers was designed by Primer3 software (National Health Institute, USA) and the forward and reverse sequences used were 5'-CAGGAGGTTGAGCACAGTGA-3' and 5'-TCCTGTAACCCCAACTACCG-3' The DNA amplification procedures was done using a Mastercycler Gradient PCR machine (Eppendorf, Germany) with the following conditions; initial denaturation at 95°C for 2 min, 35 cycles of denaturation at 95 °C for 30 s, annealing at 62 °C for 30 s, extension at 72 °C for 20 s and final extension at 72 °C for 5 min.

The successfully amplified PCR products were sent to First BASE Laboratories Sdn. Bhd. for sequencing procedures. The sequencing results were subjected to the Basic Local Alignment Search Tool (BLAST) to validate the sequence of the amplified products with the published loci in NCBI database for the respective polymorphism with aided of MEGA 5.2 software (Molecular Evolutionary Genetics Analysis version 5; http://www.megasoftware.net). Once verified, the 249bp PCR product was digested by 1U of BtsCI restriction enzyme (New England Biolabs, England) for genotyping of TAS1R2 rs12033832. Digested products was electrophoresed on 2% agarose gel with 1ul novel juice as staining reagent and visualized under UV light. The size of band was 249bp for GG genotype; 249bp, 179bp and 70bp for GA and 179bp and 70bp for AA genotype (Figure 1).

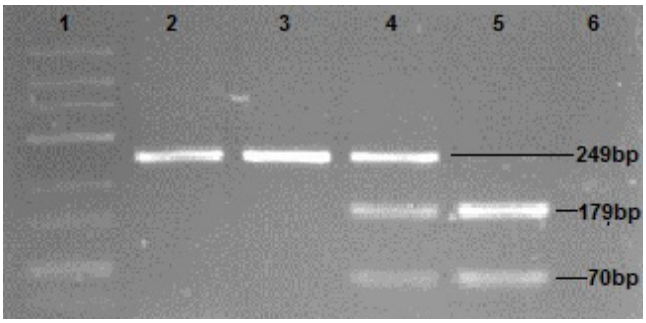

Figure 1: The enzymatic digestion of TAS1R2 gene at rs12033832 on 3% agarose gel. Figure shows the restricted profile for 50bp DNA ladder marker (lane 1), positive control (lane 2), homozygous AA (lane 3), heterozygous AG (lane 4), homozygous GG (lane 5) and negative control (lane 6). Positive control consisted of confirmed amplified products (with sequencing and BLAST-NCBI) while negative control contained reagents and PCR-grade water.

Statistical Analysis

Statistical analyses were performed by using Statistical Package for Social Sciences (SPSS) version 21.0 (SPSS Inc., Chicago, USA). The sensory measurements data were screened for outliers based on Hansen et al. (29) whereas participants were removed if they: (1) rated lowest concentration as stronger than moderate, or: (2) if there was a large discrepancy (>80 mm) between ratings for the first and second presentations. Normality test was used to verify any missing values and data outliers. Two-way repeated measures analysis of variance (ANOVA) was used to test the difference and the interaction in the sweetness and liking rating among the genotype groups. Rating data for each stimulus

concentration were treated as within-subject variables whereas BMI status and genotype groups as grouping variables. Age and gender was used as covariate in this statistical test. When appropriate, post hoc comparisons was conducted with the Scheffe test to determine any differences in the genotype groups among obese and non-obese subjects.

RESULTS

Subject's Characteristic

A total of 202 subjects were recruited at the initial stage of this study. However, only 180 subjects were included in the final statistical analysis. They consisted of 88 obese subjects (30 males, 50 females) and 92 non-obese subjects (24 males, 68 females). Four subjects were excluded due to DNA extraction failure, 7 were excluded for being outliers in sensory measurements, one dropped out and 9 were omitted due to other reasons. The total number of subjects being excluded were 22. The mean age of the 180 subjects was 25.78 ± 5.65, with majority of them in the age range of 20 to 35 years old. The BMI range was between 19 and 45.5kg/m^2, with the mean BMI of 27.56 ± 6.74 kg/m^2. Table II shows the basic characteristics of the obese and non-obese subjects involved in this study.

Table II: Basic characteristic of subjects

	Overall (n=180)	Non- Obese (n=92)	Obese (n=88)	p-value
Age (year) [a]	25.78 (5.65)	25.86 (5.28)	27.59 (6.24)	ns
Weight (kg) [a]	71.05 (5.16)	55.38 (8.49)	87.43 (12.21)	<0.001
Height (cm) [a]	160.26 (9.13)	159.41 (9.77)	161.16 (8.36)	ns
BMI (kg/m^2) [a]	27.56 (6.74)	21.74 (2.20)	33.65 (3.88)	<0.001
Gender [b]				
Male	54 (0.3)	30 (0.17)	24 (0.13)	ns
Female	126 (0.7)	58 (0.32)	68 (0.38)	

[a] Mean (SE) ; means differences analyzed by t-test
[b] n (%) ; variables association analyzed by Fisher exact test
ns:not siginificant

Suprathreshold Rating, Hedonic Response and BMI status

The means sweetness intensity rating of obese and non-obese subjects are shown in Figures 2 A and B. There was no significant difference between both BMI groups' ($p > 0.05$; main effect) on sweetness rating for sucrose solutions and food products at all the different sugar concentrations studied. Our result also showed that BMI status was not associated with suprathreshold rating of aqueous sucrose solutions and sweetness intensity of prepared sweet food products (P>0. 05; interaction effect). A similar observation was found on the hedonic responses on the food products (Figure 2C). A subsequent analysis using Mann-whitney test at all concentrations in sucrose solution revealed that there is significant difference in sweetness rating between obese and non-obese subjects at lower sucrose concentration (Figure 2A). On contrary, the sweetness rating was significantly

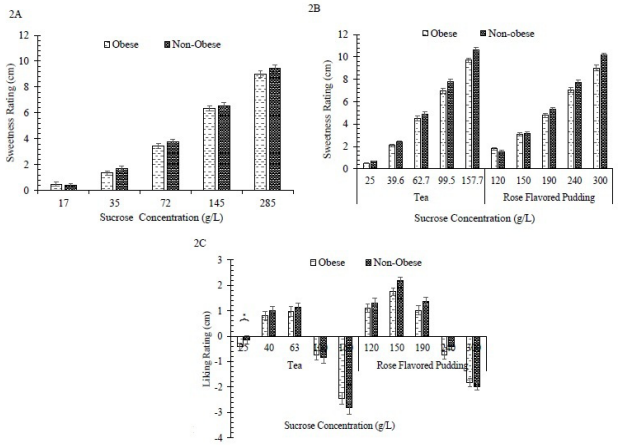

Figure 2: Mean (± SEM) sweet suprathreshold in aqueous sucrose solution between obese and non-obese (A). Mean (± SEM) sweet intensity rating of tea drinks and rose flavored pudding at different sucrose concentrations between obese and non-obese (B). Mean (± SEM) sweet liking rating of tea drinks and rose flavored pudding at different sucrose concentrations between obese and non-obese (C). * indicates significance at $p < 0.05$. Significance was compared using Mann-Whitney test.

detected at higher sugar concentration in rose-flavored pudding (Figure 2B). However, no significant difference was observed on hedonic response between obese and non-obese subjects (Figure 2C).

Sweet Suprathreshold Rating and TAS1R2 Gene Variants

TAS1R2 gene variation at rs12033832 showed a significant association with sweetness suprathreshold rating in both BMI groups ($p<0.05$). The analysis by pairwise comparison revealed that individuals with G allele for rs12033832 had lower suprathreshold sensitivity rating than the respective minor allele homozygote AA ($p < 0.05$) but no differences was found among lean individuals group (Figure 3A and 3B).

Effect of TAS1R2 Gene Variant on Sweetness Rating and Hedonic Response of Sweet Food Model

There was a significant interaction effect of TAS1R2 gene variant (rs12033832) and sweetness rating for both

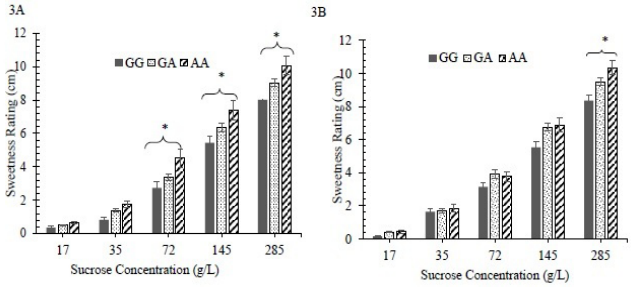

Figure 3: Sweetness rating of sucrose solution at 5 different suprathreshold concentration Obese (A) and non-obese (B). * indicates significance at $p < 0.05$. Significance was compared by pairwise test following the 2 way ANOVA repeated measure.

food products among obese and non-obese subjects ($p < 0.05$; interaction effect). Further analysis by pairwise comparison showed that individuals with G allele for rs12033832 had lower sweetness rating than the respective minor allele homozygote AA ($p < 0.05$; main effect) in both BMI groups (Figure 4A and 4B). However, the association between taste genetic variation and liking rating were different in both food products. No association was found between any of TAS1R2 gene variant (rs12033832) towards liking rating of tea ($p > 0.05$) but the reverse effect was observed for liking rating of rose flavored pudding ($p < 0.05$). Albeit, regardless of the sucrose level in rose flavored pudding, subjects with G alleles showed higher degree of acceptance compared to homozygous AA subjects in the obese and non-obese subjects (main effect; $p < 0.05$) (Figure 4C).

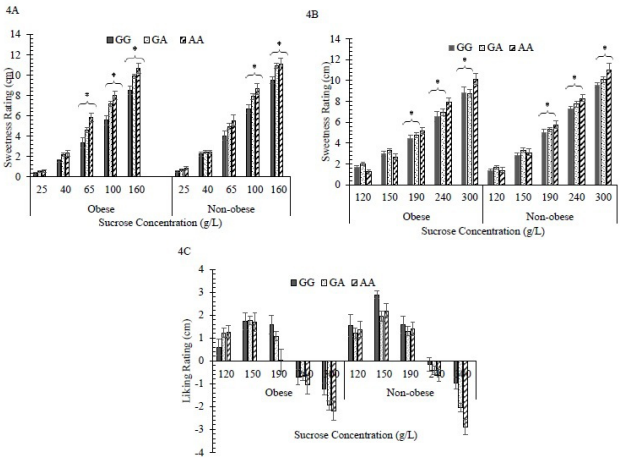

Figure 4: Sweet intensity and hedonic rating among subjects stratified by BMI status X rs12033832 of TAS1R2 gene at 5 different concentration of food products. Mean (± SEM) sweet intensity of tea drinks at 5 different sucrose concentrations (A); sweet intensity of rose flavored pudding at 5 different concentration (B); Mean (± SEM) liking rating of rose flavored pudding at different sucrose concentrations (C). * indicates significance at $p < 0.05$. Significance was compared by pairwise test following of the 2 way ANOVA repeated measure.

DISCUSSION

The present study is the first study comparing the influence of sweet taste gene variant based on BMI status (obese vs. non-obese) using different food stimulus. Results from this study showed that rs12033832 of TAS1R2 gene was associated with sweet taste perception in both obese and non-obese subjects.

Meanwhile, this present study did not find any significant difference in any taste measurements between obese and non-obese subjects. This is in line with previous studies, which indicated that the BMI status did not influence individuals' sweet taste perception (4, 30, 31). On the contrary several other studies demonstrated that obese subjects had lower sweet taste sensitivity (19, 32). Hardikar et al. (33) showed that obese subjects tend to perceive sweetness more intense than lean subjects. The differences observed in these findings could be due to several factors such as parameters measured (i.e. taste threshold vs. suprathreshold) (34), scaling procedures (35, 36) and differences in body weight status or classification (37). Most of the previous studies using taste threshold method or concentration at threshold value in determining their subjects' taste sensitivity which could not represent real taste experience (34, 35). Furthermore, some of the studies using different type of scale such as 9-point scale which not have absolute measurement as compared to gLMS been used in current study (35, 36).

Interestingly, our results revealed that there was no significant interaction between BMI status and rs12033832 of TAS1R2 gene. Indeed, the normal weight subjects seemed to give higher sweet intensity rating and lower liking towards sweetness, however no significant difference was obtained. It is important to note that sweetness intensity rating were significantly difference between obese and lean subjects at low sucrose concentration in suprathreshold rating, and an opposite trend in this relationship was observed in food product (tea and rose flavored agar-agar). This could be due to the taste-flavor interaction in the food product as compared to the suprathreshold samples which involved aqueous solution (38, 39) . Another plausible explanation could be that in food product such as tea and rose flavored pudding, the sweet taste could have been suppressed by the presence of tannin and rose flavor, respectively. As a result, sweet taste was diminished or decreased at lower concentration as the sucrose concentration was increased in the food products. In comparison, sweet taste in the aqueous solution was retained even at low concentration and it increased as the sucrose concentration was increased. Therefore, at low sucrose concentration, no significant difference sweetness rating of food product between obese and lean subject was detected because the sweet taste was either diminished or decreased and caused the sweetness rating for both groups subject to be almost similar. Study by Calvino et al. (40) showed that the suppression of sweetness by coffee flavor was highest at the lowest concentration of sucrose but the suppression was reversed as the concentration of sucrose was increased. In addition, the usage of aqueous solution in sensory measurement (e.g. taste threshold) does not fully represent the taste perception of the real food product since most food exist in matrix form (34,41) . Despite the inconsistent findings in sensory measurement results, our data consistently showed that BMI status are not associated with both suprathreshold rating and sweet intensity rating in food products.

The addition of sucrose resulted in a different hedonic response pattern for both obese and lean subjects. Obese subjects showed a lower liking rating at lower concentrations but higher liking rating at high concentration compared to lean subjects. Similar finding was observed by Pasquet et al. (37), Pepino et al. (31) and

Low et al. (42) where there was no difference in terms of hedonic response to various sucrose concentrations. Not only that, a study by Frijters and Rasmussen-Conrad (43) that included psychophysical and psychohedonic measurements indicated that the obese subjects have similar liking to sucrose solution sweetness at suprathreshold concentration. It had been reported that certain individuals are not only different in pleasantness rating but the pleasantness changes with concentration (44,45). Some subjects in this study showed quadratic trend with an optimal point, whereas others showed steady increases in pleasantness as sucrose concentration increases. Thus, the greater variations in pleasantness rating and intensity rating among the subjects can lead to similar sweetness perception between the groups with different BMI status (43, 46). However, several studies have reported that obese individuals tend to have lower sweetness sensitivity and higher preference for sweet food (23,47). This implies that differences between the obese and lean subjects still exist.

The extent to which genetic variation influences inter-individuals variability in sweet taste perception remains unknown. Our study showed that rs12033832 of TAS1R2 gene was associated with the sweet suprathreshold in both obese and lean subjects. However, the sweetness rating pattern between genotypes and BMI groups in this study was different from that reported among the Caucasian population by Dias et al. (18). They observed that the AA subjects of rs12033832 had lower sweetness rating than the G allele subjects in the obese group, while the AA subjects in the normal weight group had the opposite rating pattern. The differences may due to the small sample size in their study whereby only 2 out of 95 subjects were classified as obese with AA genotypes. In contrast, our study had showed that the G allele subjects had lower sweet suprathreshold rating in both BMI groups. In fact, obese subjects had lower sweetness rating than the lean individuals for genotype groups. Thus, it can be hypothesized that the TAS1R2 gene expression decreases in the G allele subjects in both obese and non-obese groups, which in turn lowers their sensitivity toward sweet taste (48).

There are limited studies on genetic variation and sweet taste perception in food samples. In fact, most studies used aqueous solutions to measure the subjects' taste sensitivity or hedonic rating response in relation to genetic variations in taste (17, 18). In this study, sweet food was used as the sample for measuring the subjects' sweet taste responses to reflect real life experience, as people do not usually consume blank solutions in daily life (49). Our results showed that the TAS1R2 gene variant (rs12033832) had an interaction effect on sweetness rating in obese and non-obese individuals. Furthermore, the finding is consistent with the suprathreshold rating results, in which the TAS1R2 gene variations at rs12033832 and PROP taster status are associated with sweet taste perception in both groups. This study suggests that regardless of the BMI status, any individuals with AA genotypes of TAS1R2 gene at rs12033832 have higher chances of giving higher sweetness rating in food products as they probably sensitive towards sweet taste.

When the association between the variation of rs12033832 and liking rating of food products was analyzed, the significant main effect only appeared on rose flavored pudding but not on the tea drinks. This could be due to the taste-taste interaction differences between the samples. As mentioned earlier, the suppression by other compound (e.g tannin) in tea might give a negative and narrower score among the panelists compared to enhancement of rose flavor in rose-flavored pudding (50). Despite that, a consistent pattern on hedonic response was observed in both obese and non-obese groups towards the tea drinks whereby individuals with G allele tend to give higher rating compared to AA homozygote individuals. To the best of our knowledge, this study is the first attempt that was carried out to investigate the influence of TAS1R2 genes on sweet taste acceptance in food stimulus. Most of the previous studies focus on the association among sweet taste sensitivity, dietary intake and preference. Albeit, Han et al. (47) hypothesized that individuals with low sweet taste sensitivity have higher preference and consumption of sweet food. Hence, findings from this study somewhat support the findings of Han et al. (47), where individuals with AA genotypes of rs12033832 had higher sweet taste sensitivity, lower sweet taste acceptance and lower sweet food consumption.

The present findings need to consider some limitations. Firstly, the gene variants in this study were selected based on the previous studies that showed association towards sweet taste perception. As such, additional studies that look at the effect of a greater number of variants in that particular genes on sweet taste are required as some gene variants might affect certain taste phenotype (polygenic) or even showed a pleiotropy effect. Secondly, the findings throughout this study cannot be generalized to other simple sugar or sweeteners. Different type of sweeteners might have different taste and ligand binding site in taste receptor. Additionally, food samples included in this present study were simple foods whereby sweetness is the dominant taste. Most of the food in our daily life exist in matrix form or multiple taste. Thus, the influence of genetic variation might be different compared to simple sweet food.

CONCLUSION

This present study demonstrated that obese and non-obese did not differ in their sweet taste perception. However, the sweet taste variability still existed within the groups. This variability could be attributed by TAS1R2 gene variant (rs12033832) among the individuals. This study has shown that the rs12033832 variant of TAS1R2 gene was associated with the sweet taste sensitivity and

perception in both obese and lean subjects. Regardless of BMI status, A allele of rs1203832 individuals showed higher sweet taste sensitivity but lower sweet taste perception at higher level of sucrose concentration. However, caution should be taken in generalizing the genetic variation effect on individual's sweet preference especially in relation to complex food product. The findings from this study could contribute to the existing knowledge on the variability of human taste perception particularly on the influence of taste genetic variation on sweet taste sensitivity and acceptance among obese and lean individuals.

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