ASHDIN
publishing

Research Article

Alcohol Consumption in a Non-Clinical Sample: The Role of Sweet-Liking, PROP Bitterness and Sex

Jamie-Lee Robb[1] and Gary J. Pickering[1-5] *

[1]*Department of Psychology, Brock University, St. Catharines, ON L2S 3A1, Canada*

[2]*Department of Biological Sciences, Brock University, St. Catharines, ON L2S 3A1, Canada*

[3]*Cool Climate Oenology and Viticulture Institute, Brock University, St. Catharines, ON L2S 3A1, Canada*

[4]*Charles Sturt University, Wagga Wagga, NSW 2678, Australia*

[5]*University of the Sunshine Coast, Sippy Downs, Queensland 4556, Australia*

Address correspondence to Gary J. Pickering, gpickering@brocku.ca

Abstract

Some previous studies have suggested an association between sweet-liking and alcohol use in male alcohol-dependent individuals. However, if sweet-liking is to have value as an indicator of potentially hazardous drinking behaviour, the relationship needs to be established in non-dependent individuals, and determined for women and younger individuals, who may be at increased risk of alcohol use disorders. This study comprised of a non-clinical sample of 223 male and female university students. Responsiveness to 3 sucrose-impregnated taste discs (9 g/l, LSD; 90 g/l, MSD; 900 g/l, HSD) and a 50 mM 6-n-propyl-2-thiouracil (PROP)-impregnated disc were collected and used to classify participants as sweet-likers (HSD/LSD $\geq$ 1.5), sweet-dislikers (HSD/LSD < 1.5), PROP non-tasters (gLMS intensity score $\leq$ 12 mm), PROP medium-tasters (13-55 mm), or PROP supertasters ($\geq$ 56 mm). Data on familial history of alcoholism, alcohol intake, and hazardous drinking (Alcohol Use Disorders Identification Test, AUDIT) were also collected. Two-way Analysis of Variance showed a significant main effect for sweet-liking on alcohol consumption in males (F (1)=4.10, p=0.04), with monthly intake (natural log transformed) of sweet-liking males higher than sweet-disliking males. Neither alcohol consumption (t (191)=1.97, p=0.23), sweet-liking (ratio of HSD liking over LSD; t (191)=1.97, p=0.41), or PROP responsiveness (t(191)=1.97, p=0.56) varied with AUDIT classification or family history of alcoholism (p> 0.05). Overall, our results partially support the hypothesis that ethanol and sucrose influence the opioid reward system in the brain in a similar way to reinforce use.

Keywords: alcohol, sweetness, sweet-liking, taste perception, individual differences, PROP

3. Introduction

A common staple in the diet for many societies, the consumption of alcohol may have a more intricate influence on human health than the effects of smoking [1]. When consumed in moderate amounts, alcohol may provide benefits to overall health by reducing risk of cardiovascular disease and overall mortality [1-4]. However, high consumption over time is associated with increased risk of developing an alcohol use disorder (AUD), and may lead to a number of other health issues [1, 5]. Although men tend to consume more alcohol than women, women may be at higher risk of harm to health as they often engage in abstinence-binging patterns of use, and are generally more susceptible to the effects of alcohol [1, 3, 5], and other drugs of abuse [6]. Similarly, younger individuals are vulnerable to AUDs and health complications as their social environment often supports hazardous drinking behaviour. Interestingly, relatively few studies have reported on alcohol consumption in these groups [1, 3, 7-8].

Although factors such as socioeconomic status, stress, and pre- and post- natal exposure to alcohol can influence consumption patterns [1, 3, 9-10], individual variation in orosensory responsiveness can also influence alcohol use behaviours, including risk of dependence (reviewed in Thibodeau & Pickering [11]). For instance, it is likely that individual differences in responsiveness to the orosensory properties elicited by ethanol partially explain consumption [9, 12]. Greater sensitivity to the sweetness of ethanol may increase likelihood of consumption, while enhanced sensitivity to its bitterness and irritant properties might deter intake [4, 13].

3.1. Sweetness perception

Over evolutionary time, humans have developed an innate preference for sweet substances [14]. Further, sweet foods and beverages are able to activate natural reward

systems in the brain and reinforce future consumption [15-16]. Although hedonic response to sweetness has been reported as a genetically determined trait [14, 17-19], one study of over 300 twin dyads did not find any evidence to corroborate a genetic basis to sweet preference [69]. Previous studies suggest that the consumption of sweet foods is influenced by multiple factors, including homeostasis in the body, food adventurousness [21], and sweet-liking, and have noted that individual variation in the perceived intensity of sweet foods and stimuli also influence liking and subsequent consumption [8, 16, 17, 22]. In the literature, 'sweet-liking' typically refers to a positive correlation between reported liking and the concentration of sweet-eliciting stimuli, while 'sweet-dislikers' are often defined as individuals who prefer lower concentrations of sweet-eliciting stimuli [19]. Liking is often measured in lieu of reported consumption to provide a more accurate measure of dietary intake, as it reduces the effects of cognitive limitations and underreporting [23].

3.2. Sweetness perception and alcohol use

A relationship between consumption of alcohol and sweet-liking is generally supported in the literature [12, 24-26]. Ethanol has been shown to stimulate chemoreceptors for sweetness in both humans [8] and non-human primates [27]. Ethanol and sucrose activate two of the primary neurotransmitter systems in the brain, the mesolimbic dopaminergic and opioidergic pathways, both of which originally evolved to respond to naturally rewarding stimuli (e.g., sweet foods) in the environment, and promote survival [4, 12, 15, 22, 28-29]. After consuming ethanol or sucrose opioid receptors in the ventral tegmentum area and the ventral striatum of the brain are activated by sweet-responding fibres, increasing extracellular levels of dopamine in the nucleus accumbens and reinforcing future behaviours through desensitization, thereby regulating the hedonic response for such stimuli [12, 15, 19, 25, 28-34].

Using predominantly male clinical samples, several studies have found that more than half of alcoholic participants demonstrate a preference for the strongest sweet solution when presented with multiple concentrations [20, 25, 26], and other studies have reported that these individuals are more likely to be sweet-likers than dislikers [31]. Further, research with animal models has demonstrated that a preference for sucrose solutions is a strong predictor of alcohol consumption in rodents [18, 25]. However, this assumption may be too simplistic for humans, as sweet-preference can also be influenced by factors such as poor diet, oral or olfactory damage, and heavy drinking [8, 22]. Further, if sweet-liking is to be useful as a phenotypic marker for risk of alcoholism, the association should be evident in all individuals at risk for AUDs and established in a non-clinical sample [35].

3.2.1. Sweet-liking and family history of alcoholism.
As heritability of alcoholism is estimated at 50%, family history of alcoholism is the best single predictor of AUDs [19, 24]. Previous research has demonstrated that individuals with a family history of alcoholism (FH+) may be genetically predisposed to abnormalities in the brain opioid system, whereby the release of β-endorphins is enhanced when activated by alcohol or sucrose ingestion, thereby reinforcing the likelihood of future consumption [24, 26].

Research has demonstrated that FH+ individuals are more likely to be sweet-likers than individuals without familial alcoholism (FH-), independent of their own alcohol use [19, 24-26]. For example, Kampov-Polevoy and colleagues [25] found that 61% of participants with a family history of alcoholism (FH+) preferred the highest concentration of five sweet solutions compared to only 19% of FH- participants. In contrast, Tremblay et al. [35] did not find an association between the intensity ratings of or preference for five different sucrose concentrations and FH+ in a sample of over 90 individuals with alcohol dependence, consistent with three other reports that did not find a relationship between sweet-liking and familial alcoholism [20, 34]. Overall, these divergent findings suggest that the relationship between these variables is still poorly understood, and likely more complex than originally reported.

3.3. PROP responsiveness and alcohol use

PROP or 6-*n*-propyl-2-thiouracil, is a synthetic bitterant used as a proxy for general taste sensitivity. Approximately 30% of Caucasian North Americans and Western Europeans are unable to taste PROP and are commonly referred to as PROP non-tasters (pNT), whereas the other 70% of the population are classified either as PROP medium-tasters (pMT) if they perceive the compound as moderately bitter, or PROP supertasters (pST) if they perceive it as intensely bitter [4, 37-40]. PROP bitterness associates in many studies with general responsiveness to sweetness, sourness, bitterness, astringency, irritation [4, 37, 40-42] and possibly retronasal olfaction [43], all of which are elicited by alcoholic beverages.

Interestingly, PROP tasters may be at lower risk of AUDs due to increased responsiveness to the aversive orosensory properties of ethanol and alcoholic beverages [8-9, 40-42, 44-46]. For example, those who rate PROP as being very bitter also report red wine [45-46], scotch and some beers [8, 47] as more irritating or bitter. Several [8, 38, 41, 47-48], although not all [9, 41, 49-50] studies have reported higher alcohol consumption in individuals who experience less PROP bitterness, corroborating the hypothesis that PROP tasting may be protective against risk of AUDs [38, 44].

3.3.1. PROP responsiveness and alcoholism.
A link between PROP responsiveness and familial alcoholism has also been noted in the literature, where those with a FH+ are more likely to be pNTs than FH- individuals [4, 44, 49, 51]. However, a study using a sample of

younger adults did not find an association between PROP responsiveness and familial alcoholism [52], consistent with the reports of Kranzler et al. [52] and [53]. Sex may be an important mediator of any association, with Tepper [44], also using college participants, reporting that pST males had less risk of AUDs and familial alcoholism, while pST females report higher risk of AUDs and familial alcoholism [44].

Thus, the literature is conflicted with respect to both the relationship between sweet responsiveness and alcohol use, as well as PROP responsiveness and risk of AUDs, with significant methodological differences between studies likely a contributing factor. As the majority of prior research has focused on male clinical samples, and very few have investigated the sweetness-alcohol relationship in non-clinical, younger females, the generalizability of much of the previous findings is limited. Additionally, we are not aware of any research that has investigated the role of PROP bitterness and sweet responsiveness on alcohol use/familial history of alcoholism in the same sample.

3.4. Study rationale

The present study aimed to contribute to an improved understanding of the factors that influence alcohol intake and use disorder in a non-clinical sample of male and female university students. Based on previous research, we hypothesized that sweet-liking and alcohol use would be positively correlated, and that the PROP taster phenotype may mediate this relationship. We also hypothesized that individuals with FH+ would be more likely to be sweet-likers than dislikers compared with FH- individuals. From these analyses, we hope to determine whether sweet-liking may be used as a phenotypic marker for risk of alcoholism in

a non-clinical sample, and what other factors may influence alcohol use and misuse in younger individuals.

4. Material and Methods

4.1. Participants

Two hundred and twenty-three students and other individuals affiliated with Brock University were recruited using posters, an online recruitment tool (SONA), and personal invitation. Participants who did not meet the age restrictions of 19-50 years ($n = 5$), did not drink alcohol ($n = 4$), submitted blank data ($n = 13$), reported drinking more than 250 alcoholic beverages per month ($n = 7$), or reported prior diagnosis of alcohol dependence ($n = 1$) were removed from the data set, leaving 193 respondents (Table 1). All recruitment tools and measures received ethics clearance from the Brock University Bioscience Research Ethics Board (file number 11-122-PICKERING).

4.2. Materials

Each participant received an envelope containing four individually wrapped and randomly coded taste discs, an instruction sheet indicating the participant's unique 3-digit identification code, and guidelines. The guidelines directed participants to complete the study in a quiet location without any distractions, to avoid consuming anything besides water for at least half an hour before beginning and while completing the survey, and to contact the researcher when completed so they could be compensated for their time. Six versions of the online survey were created in SelectSurvey.NET v4.049.003 (ClassApps.com, Overland Park, KS 66223), and housed on a secure Brock University-administered server. Participants were directed to the main login page and had

Table 1: Characteristics of Participants ($n = 193$). Data presented as the mean ± standard deviation (S.D).

Characteristic		Overall	Males	Females
Age (years)		20.86 ± 3.29	21.5 ± 4.30	20.6 ± 2.74
Ethnicity	Caucasian	80.8 %	78.9 %	81.6 %
	Other	19.2 %	21.1 %	18.4 %
Average monthly alcohol consumption[1] **		3.37 ± 1.15	3.05 ± 1.23	3.51 ± 1.08
Average drinks per occasion		2.6 ± 1.5	2.11 ± 1.31	2.32 ± 1.57
AUDIT Classification[2]	Hazardous	63.7 %	64.9 %	63.2 %
	Non-hazardous	36.2 %	35.1 %	36.8 %
Average HSD/LSD liking ratio[3]		1.9 ± 1.27	1.69 ± 1.16	1.96 ± 1.32
Sweet-liking classification[4] **	Sweet-likers	51.3 %	33.3 %	57.8 %
	Sweet-dislikers	48.7 %	66.6 %	42.2 %
Family History of Alcoholism[5]	FH +	14.5 %	17.5 %	13.2 %
	FH -	85.5 %	82.5 %	86.8 %
PROP responsiveness[6]		46.5 ± 25.0	40.49 ± 24.36	49.1 ± 24.92
PROP Taster Status	Non-taster	13.5 %	15.8 %	12.5 %
	Medium-taster	57.0 %	61.4 %	55.1 %
	Supertaster	29.5 %	22.8 %	32.4 %

[1]Data (standard drinks per month) natural log transformed.[2] Measured using the Alcohol Use Disorders Inventory Test [3] Liking was measured on a 9-point hedonic scale separately for highest sweetness disc (HSD) and lowest sweetness disc (LSD), anchor terms ranged from *dislike extremely (1)* to *like extremely (9),* with a neutral option, *neither like nor dislike (5)* in the middle. [4]Determined by the ratio of liking for the HSD:LSD, ≥ 1.5= sweet-liker, < 1.5 = sweet-disliker. [5]Measured using an adapted version of the Shortened Michigan Alcohol Screening Test (SMAST). [6]mm, measured on a generalized labeled magnitude scale (gLMS). ** = $p < 0.05$.

to enter one of six alphanumeric codes to access their randomly assigned survey. Guidelines to complete the study were reiterated on the screen, and a consent form was presented before participants could enter their unique ID code and begin the survey (pages 1-3). Pages 4-17 of the survey consisted of demographic questions, inventory-type measures, questionnaires, and three sweet-taste tests. All measures and sweet-taste tests were randomized so that every possible combination of tests was used. Finally, participants were instructed to self-administer the PROP disc and record the perceived intensity on a generalized labeled magnitude scale (gLMS), before being debriefed and thanked for their time.

4.3. Measures

4.3.1. Current alcohol behaviours.

4.3.1.1 Frequency of alcohol consumption. Similar to the procedure used by Pickering and Cullen [54], participants were asked to report how many occasions per month they consumed each of six categories of alcoholic beverages (red wine, white wine, beer, spirits, mixed drinks and other). In addition, they were asked how many standard drinks (i.e., 12 oz. bottle of beer, 6 oz. glass of wine, 1.5 oz. of spirit) they consumed per occasion when they were drinking for each of the six categories. The product of these two measures was calculated for each alcoholic beverage category and summed for each participant to create a composite score of the total intake of alcoholic beverages per month. As a Shapiro-Wilk's test revealed this composite variable was not normally distributed, the skewed data was transformed using the natural logarithm to normalize the data, and this transformed variable was used for all further analyses.

4.3.1.2 Alcohol Use Disorders Identification Test (AUDIT). The third measure of alcohol behaviour was a 10-item self-report inventory originally created by the World Health Organization. The AUDIT [55] has been validated for use with different populations and age groups, including clinical samples, adults and college students, and is a reliable measure of hazardous (developing), as well as harmful (already established) drinking behaviours [8, 55-56]. Scores can range from 0-40. Following the approach of Lanier *et al.* [8], we used a cut-off score of ≥ 8 to classify participants as high versus low risk of hazardous drinking.

4.3.2 Family history of alcoholism. A modified version of the 13-item Short Michigan Alcoholism Screening Test (SMAST) [57] was used to assess family history of alcoholism. Items were answered using yes/no responses

and each yes response was coded as 1 with the exception of three items (1, 4, 5) that were reverse-scored before analyses. Scores ranged from 0-13, and a cut-off score of ≥ 4 was used per Crews and Sher [57] to indicate high likelihood of family history of alcoholism.

4.3.3 Taste tests. After the procedure of Zhao, Kirkmeyer, and Tepper [58], taste tests consisted of 3.2 cm circular filter paper discs (Whatman International Ltd., England) that had been impregnated with tastant solutions then dried. The use of filter discs to assess taste responsiveness has been used previously in several sensory studies [e.g., 14, 50, 59], including studies where participants self-administer the stimuli [e.g., 60-61]. Three concentrations of food-grade sucrose (BioShop Canada Inc. ON, Canada) were selected after bench-testing to represent a wide range of sweetness intensities: low sweetness (9 g/l; LSD), medium sweetness (90 g/l; MSD), and high sweetness (900 g/l; HSD). A 50 mM concentration of 6-n-propyl-2-thiouracil (PROP; MP Biomedicals. OH, USA) was also prepared to assess PROP taster status after the method of Zhao and colleagues [58]. To minimise carry-over effects, the survey was designed so that there was a five-minute break between the (self) administration of each taste. Additionally, participants were instructed to rinse their mouth with tap water after each disc.

4.3.4 Sweetness hedonics and intensity. Expanding on the procedure of Cruickshanks *et al.* [62], each participant completed three sucrose-impregnated taste tests. For each sweet-taste test, participants were instructed to place the disc on the tip of their tongue for 30 seconds, or until the disc was wet, then remove it from the mouth, and rate the sweetness intensity on a 15 cm Generalized Visual Analog Scale (gVAS) [59, 63] (Figure 1). The gVAS was anchored on the left with "no sensation" and on the far right with "strongest sensation imaginable". Sweet-liking was subsequently assessed by asking participants to rate their liking of each sweet-disc on a separate 9-point hedonic scale [17, 64], prefaced with "Rate your liking of this sweetness". Sweet-likers were defined as those individuals whose HSD/LSD liking score was ≥ 1.5, and sweet-dislikers as a HSD/LSD liking ratio of < 1.5.

4.3.5 PROP. Participants were instructed to place a filter paper disc impregnated with 6-n-propyl-2-thiouracil (PROP) on the tip of their tongue for approximately 30 seconds [58]. The disc was then removed from the mouth and intensity was reported on a 100 mm generalized Labeled Magnitude Scale (gLMS), which is valid for across-group comparisons [62, 65]. To ensure that the scale would display correctly on the range of common Internet

Figure 1: Example of the generalized visual analog scale (gVAS; adapted for online response) used for the sweet taste tests.

browsers, each point on the scale translated to a four-point increment, as adapted from the procedure of Lanier *et al.* [8]. Participants received written instructions, which stressed they could click anywhere on the scale to record their intensity rating, and an example image was provided. Anchor terms indicated that the top of the scale represented the "strongest imaginable sensation of any kind" (100 mm) and the bottom of the scale represented "no sensation" (0 mm) experienced. Intermittent anchor terms included "very strong" (52 mm), "strong" (36 mm), "moderate" (16 mm), and "weak" (8 mm). PROP Taster Status phenotypes were classified as follows: PROP Non-taster (pNT, ≤ 12 mm), PROP Medium-taster (pMT, 13-55 mm), PROP Supertaster (pST, ≥ 56 mm; Figure 2). These cut-offs reflect natural breaks in the distribution of PROP intensity scores and are similar to those used by Pickering et al. [60].

4.4. Data analysis

All analyses were carried out using XLSTAT 2015 (*Addinsoft*. New York, NY). Outliers for frequency of alcohol consumption were identified using Grubb's test, and individuals who did not meet inclusion criteria (refer Section 2.1) were removed from the data set. An alpha level of 0.05 was used for all analyses. Correlation matrices were computed for all continuous variables, and Student's *t*-tests were used to analyze differences in sweet-liking, current and hazardous drinking behaviours, and family history of alcoholism. Analyses of Covariance (ANCOVAs) were also used to examine the effects and interactions of sweet-liking (sweet-likers vs. sweet-dislikers), PROP taster status and responsiveness, and sex (male vs. female) on monthly alcohol consumption, as well as family history of alcoholism. All data are presented as means ± standard deviation (S.D). Cohen's *d* statistic is reported for significant results to indicate the effect size.

5. Results

5.1. Validation check – sweetness

Student's t-tests confirmed that sweet-likers (M = 6.81 ± 1.16) liked the highest concentration disc (900g/l) significantly more than individuals classified as sweet-dislikers (M = 5.87 ± 1.40; t (180) = 1.97, p = < 0.0001), while sweet-dislikers preferred the lowest concentration disc (9g/l; M = 5.05 ± 0.89; t (156) = 1.98, p < 0.001) and medium concentration disc (90g/l; M = 5.16 ± 1.28; t (174) = 1.97, p = 0.05) compared to sweet-likers (M =4.76 ± 1.46; M = 3.09 ± 1.24, respectively). Although there were no significant differences in the perceived intensity of the medium concentration disc (t (181) = 1.97, p = 0.27), sweet-likers rated intensity higher for the lowest (M = 4.92 ± 3.92) and highest (M = 9.35 ± 2.66; t (181) = 1.97, p < 0.001) concentration disc than sweet-dislikers (M = 3.63 ± 2.99; M = 7.76 ± 3.11, respectively). Interestingly, sweet-likers and dislikers rated PROP intensity differently (t (175) = 1.97, p = 0.01, d = 0.44) with sweet-likers (M = 51.66 ± 26.41) perceiving PROP more intensely than sweet-dislikers (M = 41.52 ± 23.64).

5.2. Alcohol use, sweetness perception, and PROP

Aside from a weak inverse correlation between age and monthly alcohol consumption (r = -.15, p = 0.03), no significant correlations were found between PROP responsiveness, liking or intensity of each of the sweet discs, sweet-liking classification (HSD:LSD ratio) and alcohol consumption. However, a t-test comparing the monthly alcohol consumption of sweet-likers (M = 3.52 ± 1.06) and sweet-dislikers (M = 3.23 ± 1.22) approached significance (t (191) = 1.97, p = 0.08, d = 0.23). Neither alcohol consumption (t (191) = 1.97, p = 0.23), sweet-liking (based on HSD:LSD ratio; t(191)=1.97, p = 0.41), nor PROP intensity (t(191)=1.97, p= 0.56) varied with AUDIT classification. However, when individual sweet-discs were analyzed, intensity, but not liking for the MSD was found to be significantly different (t (191) = 1.97, p = 0.01) between individuals at high risk of AUDs (M = 5.98 ± 3.32) and individuals at low risk of AUDs (M = 4.83 ± 2.07). No difference was found between liking or intensity of the LSD or HSD (p > 0.05).
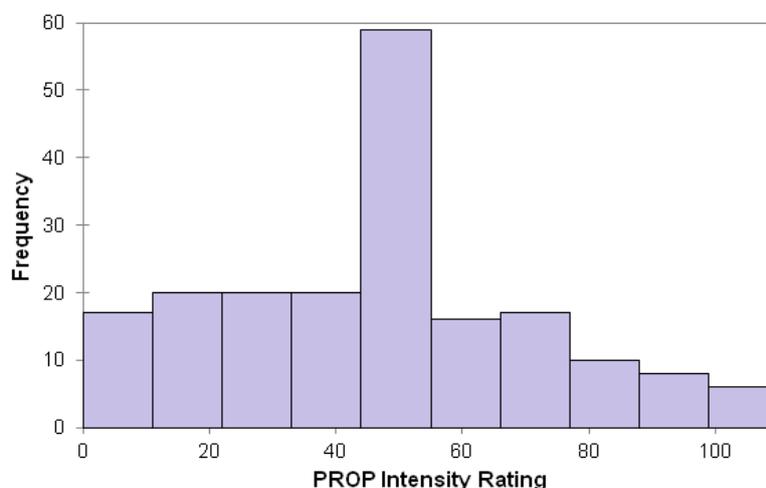

Figure 2: Frequency distribution of 6-*n*-propylthiouracil (PROP) scores as measured on a generalized labeled magnitude scale (gLMS). PROP taster status cut-offs are indicated using arrows.

5.2.1. Sex effects: When male and female data were separated, a positive correlation was found between the sweet-liking ratio (HSD:LSD) and PROP responsiveness for both males (r = 0.16, p = 0.22) and females (r = 0.21, p = 0.01). Further, a significant difference was found between males and females in their monthly consumption of alcohol (t (191) = 1.97, p = 0.01, d = 0.4), with male participants (M = 3.05 ± 1.24) consuming fewer drinks than female participants (M = 3.51 ± 1.08). With respect to the sweet discs, females reported greater intensity for the highest concentration (M = 9.13 ± 2.85, t (104) = 1.98, p < 0.001), medium concentration (M = 5.89 ± 3.40; t (130) = 1.98, p = 0.02), and lowest concentration (M = 4.98 ± 3.83; t (175) = 1.97, p < 0.001) than did males (HSD: M = 7.12 ± 2.89, MSD: M = 4.82 ± 2.71, LSD: M = 2.77 ± 2.02). However, male participants reported greater liking of the LSD (M = 4.48 ± 1.29; t (120) = 1.98, p =0.01) and MSD (M = 5.21 ± 1.04; t (148) = 1.98, p = 0.06), but not the HSD (M = 6.34 ± 1.24, t (120) = 1.98, p =0.87), while female participants reported greater liking of the higher concentration disc (HSD: M = 6.31 ± 1.43, MSD: M = 4.86 ± 1.50, LSD: M = 3.95 ± 1.49). Additionally, females (M = 49.1 ± 24.92) rated PROP bitterness higher (t (114) = 1.98, p = 0.01, d = 0.34) than males (M = 40.49 ± 24.36) (Figure 3).

A two-way ANOVA was conducted to determine whether sweet-liking and PROP taster status would interact to influence the monthly alcohol consumption (natural log transformed) of females. There were no significant main effects, nor interactions (F (2) = 0.91, p > 0.41; Figure 4A). However, a two-way ANOVA showed a significant main effect of sweet-liking on alcohol consumption for males (F (1) = 3.94, p = 0.05; Figure 4B), with sweet-liking males consuming more alcohol per month (M = 3.56 ± 1.06) than sweet-disliking males (M = 2.86 ± 1.22). PROP taster status did not have a main effect on alcohol consumption in males (F (2) = 0.11, p = 0.89)

5.3. Family history of alcoholism

Student's t-tests did not reveal any significant differences for sweet-liking (HSD:LSD ratio; t(191) = 1.97, p = 0.74), intensity, or liking for each of the sweet discs (HSD: p = 0.57 and 0.58; MSD: p = 0.06 and p = 0.29; LSD: p = 0.12 and p = 0.77, respectively), or monthly alcohol consumption (t (180) = 1.97, p = 0.57) between individuals with and without a family history of alcoholism. Further, no significant differences in PROP intensity were found between FH+ and FH- groups (t (180) = 1.97, p = 0.58; Table 2). Further, a t-test for independent samples examining family history as a
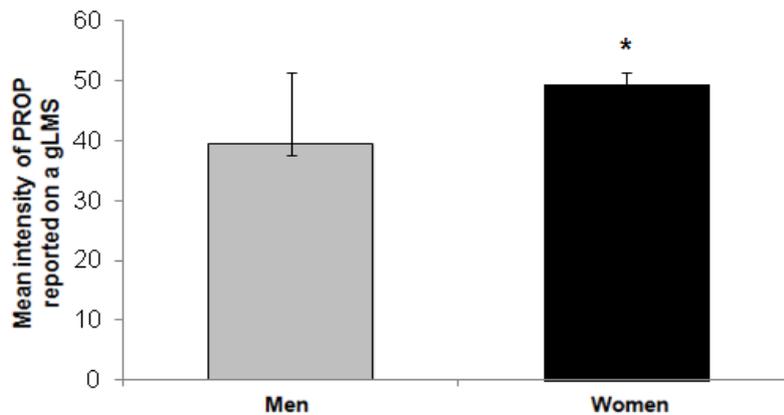

Figure 3. Sex differences in sensation intensity elicited by 6-n-propyl-2-thiouracil (PROP), as reported on a generalized labeled magnitude scale (gLMS). Data presented are the mean ± standard deviation (S.D). *p < 0.05.
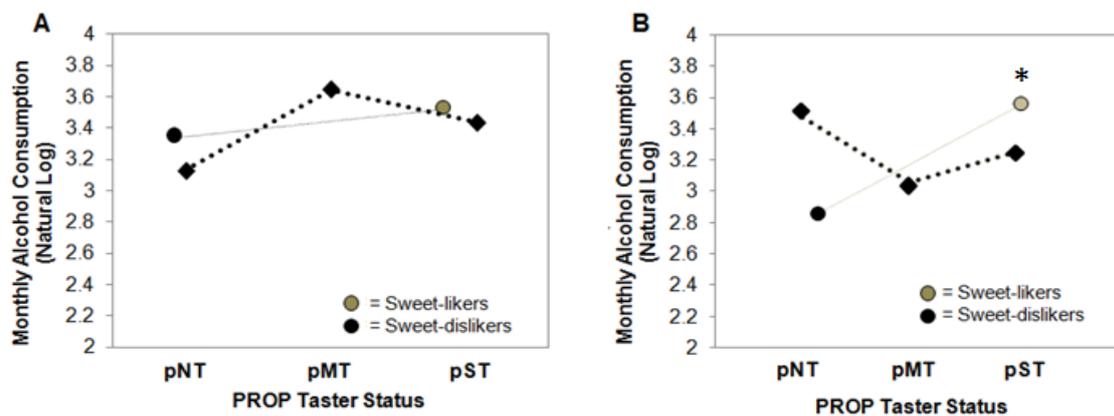

Figure 4. Monthly alcohol consumption (natural log transformed) of A) female (n = 136), and B) male (n = 57) participants, by PROP taster status and sweet-liking. Note: pNT = PROP non-tasters, pMT = PROP medium-tasters, pST = PROP supertasters. *p < 0.05.

Table 2: PROP responsiveness, sweet-liking and alcohol use in individuals with and without a family history of alcoholism (n = 182-193).

	Family History of Alcoholism	N	M	S.D
PROP intensity rating (mm on gLMS)	FH-	165	46.92	25.83
	FH+	28	44.07	22.89
Sweet-liking (HSD:LSD)	FH-	165	1.89	1.30
	FH+	28	1.81	1.13
Monthly alcohol consumption (average number of standard drinks consumed)	FH-	165	3.39	1.18
	FH+	28	3.26	0.96
Average number of alcoholic drinks consumed per occasion	FH-	155	2.25	1.50
	FH+	27	2.31	1.59

HSD = high sweetness disc, *LSD* = low sweetness disc, *PROP* = 6-n-propyl-2-thiouracil, *gLMS* = generalized labeled magnitude scale, *FH +* = Family history of alcoholism, *FH-* = No family history of alcoholism. Monthly alcohol consumption data was transformed using the natural logarithm to improve normality.

continuous variable did not reveal a significant difference between sweet-likers and sweet-dislikers (HSD:LSD ratio; t (191) = 1.97, p = 0.84).

6. Discussion

Based on some prior studies supporting a relationship between alcohol use and sweet-liking in humans [12, 24-25], we hypothesized that sweet-liking and alcohol use would be positively related, and that this relationship may be influenced by PROP responsiveness. Although results approached significance and in the direction expected for the sweet-liking:consumption hypothesis, the effect size appears relatively small overall. Importantly, this relationship appears to hold true for male but not female respondents, in agreement with the findings of Kampov-Polevoy *et al.* [25] and Lemon *et al.* [12] who recruited male alcohol-dependent participants. The increased alcohol consumption in our male sweet-liking cohort did not however extend to an increased likelihood of hazardous drinking, although the analysis was likely underpowered by the small sample number in individuals cells. In part, this sex difference might be influenced by hormones [6], or genetic variability in PROP responsiveness and ethnicity [66]. For example, it appears as though individuals who experience the orosensory qualities of alcohol more intensely demonstrate a trend towards decreased consumption [11, 50].

It has previously been reported that pSTs are more likely to consume less alcohol [37], and be classified as sweet-dislikers [17, 39] than pMTs or pNTs. Further, it has been suggested that the pST phenotype may be protective against risk of alcoholism, particularly if a low preference for sweetness is demonstrated [8-9, 38, 42, 44]. That is, pSTs are generally deterred from consuming large amounts of alcohol due to their heightened responsiveness to its aversive orosensations [4], however if hedonic preference for sweetness is present, consumption may be increased. Our data failed to confirm this hypothesis, possibly due to sample size and the age range of the participants.

In the present sample, female participants reported greater liking of the highly concentrated sweet discs, and rated PROP more intensely than male participants, as found in

past research [44]. However, it is still unclear how the PROP taster phenotype might influence risk of hazardous drinking behaviours, such as abstaining and then binging, as often seen in females and younger individuals [3]. Since sweet-liking varies with concentration, we measured intensity of sweet stimuli independently from PROP responsiveness to determine whether it associates with alcohol use [17]. We did not find a significant relationship between the two variables, although there appears to be a positive trend between sweet-liking and super tasting for both males and female participants. While the balance of literature shows that orosensory responsiveness to both ethanol and alcoholic beverages as estimated by the PROP bitterness proxy likely does associate with intake and risk of alcohol use disorders (reviewed in Thibodeau & Pickering [11]), it clearly is not a sufficient predictor.

Past research has demonstrated that individuals with a family history of alcoholism may not only be more likely to consume high amounts of alcohol and sweets due to heightened activation in their brain opioid system [24, 26, 36], but they may also be less responsive to PROP bitterness than individuals without familial alcoholism [4, 44, 52]. We did not find a relationship between familial history of alcoholism and sweet-liking, PROP taster status or alcohol use, however we acknowledge that the limited number of FH+ participants (28) increased the likelihood of Type II error. Further, it is possible that familial alcoholism may influence sweetness perception and preference through non-biological mechanisms. For example, individuals raised in families with alcohol dependence may experience greater poverty, putting them at risk of malnutrition, and may consume a higher proportion of typically cheaper and more readily available high-glycemic foods, and thus exhibit potentially different exposure and preference profiles for sweetness than FH- individuals.

6.1. Study Limitations and Future Directions

Although the present study was able to recruit a moderately sized non-clinical sample, the survey was completed at a time and place of the participant's convenience, rather than in the laboratory under more controlled conditions.

It is therefore possible that participants may have misinterpreted instructions for some measures. The use of taste discs rather than whole mouth procedures for delivering the sweet stimuli requires further validation with respect to the intensities elicited, which in turn are used for sweet-liking classification. Cell sizes for some analyses once parsed apart, including those used to examine the sweet-liking x PROP interaction, were too small to make robust conclusions regarding effects. Finally, our sample may be too young to accurately represent the non-clinical population as a whole, since some of these individuals may not have established drinking behaviours independent of other influences, such as social pressure and novelty of drinking.

Factors that may have contributed to a null result with respect to genetic risk of alcoholism on current consumption or sweet-liking and that could be considered in future research include novelty seeking [8, 24], acute or chronic stress experienced by the participants [10], and, with respect to the role of PROP, family history of depression [67]. For example, some reports have found that brain opioid system dysfunction (as measured by risk of familial alcoholism), novelty seeking and sex are able to predict alcohol consumption in clinical samples when combined, although they are unable to independently predict alcohol use [19, 24]. We encourage further research on the influence of orosensory responsiveness on alcohol consumption and use disorder across the lifespan, and on the neural mechanisms that underlie responsiveness to rewarding stimuli. For instance, although the limited literature generally suggests that older individuals may be at decreased risk for hazardous alcohol use [1], their alcohol behavior has not been well studied; it is possible that the age-related degeneration of gustation and olfaction may decrease responsiveness to the aversive qualities of alcohol, altering intake [68].

This study contributes to the existing literature by recruiting a non-clinical sample to test some of the hypotheses that have been supported in previous work with clinical samples and animal models. Additionally, we used both females and males, which facilitated the finding that the sexes differ in their consumption of dietary ethanol and in the contribution from sweet-liking.

Understanding the role of orosensory responsiveness and hedonics, family history, and the opioid reward system on alcohol use and misuse is important at the level of the individual, and also for society as a whole to ensure that our citizens are developing and aging in a healthy manner.

7. Acknowledgments

References

1. M. Gronbaek, *The positive and negative health effects of alcohol- and the public health implications*, J Intern Med, 265 (2009), 407-420.

2. B. Boden-Allbala, and R. L. Sacco, *Lifestyle factors and stroke risk: Exercise, alcohol, diet, obesity, smoking, drug use, and stress*, Curr Atheroscler Rep, 2 (2000), 160-166.

3. M. P. Ferreira, and D. Willoughby, *Alcohol consumption: The good, the bad, and the in different*, Appl Physiol Nutr Metab, 33 (2008), 12-20.

4. R. D. Mattes, and D. DiMeglio, *Ethanol perception and ingestion*, Physiol Behav, 72 (2001), 217-229.

5. J. B. Saunders, and N. K. Lee, *Hazardous alcohol use: its delineation as a subthreshold disorder, and approaches to its diagnosis and management*, Compr Psychiat, 41 (2000), 95-103.

6. M. E. Carroll, W. J. Lynch, M. E. Roth, A. D. Morgan, and K. P. Cosgrove, *Sex and estrogen influence drug abuse*, Trends Pharmacol Sci 25 (2004), 273-279.

7. D. Catanzaro, E. C. Chesbro, and A. J. Velkey, *Relationship between food preferences and PROP taster status of college students*, Appetite 68 (2013), 124-131.

8. S. A. Lanier, J. E. Hayes, and V. B. Duffy, *Sweet and bitter tastes of alcoholic beverages mediate alcohol intake in of-age undergraduates*, Physiol Behav, 83 (2005), 821-831.

9. A. A. Bachmanov, S. W. Kiefer, J. C. Molina, M. G. Tordoff, V. B. Duffy, L. M. et al, *Chemosensory factors influencing alcohol perception, preferences, and consumption*, Alcohol Clin Exp Res, 27 (2003), 220-231.

10. N. D. Volkow, and R. A.Wise, *How can drug addiction help us understand obesity?* Nature, 8 (2005), 555-560.

11. M. Thibodeau, and G. J. Pickering, *The Role of Taste in Alcohol Preference, Consumption and Risk Behavior*, Critical Reviews in Food Science and Nutrition, (2017).

12. C. H. Lemon, S. M. Brasser, and D. V. Smith, *Alcohol activates a sucrose-responsive gustatory neural pathway*, J Neurophysiol 92 (2004), 536-544.

13. A. A. Nolden and J. E. Hayes, *Perceptual qualities of ethanol depend on concentration, and variation in these percepts associates with drinking frequency*, Chemosens Percept, 8 (2015), 149-157.

14. K. Keskitalo, A. Knaapila, and M. Kallela, *Sweet taste preferences are partly genetically determined: Identification of a trait locus on chromosome 16*, Am J Clin Nutr 86 (2007), 55-63.

15. A. E. Kelley, and K. C. Berridge, *The neuroscience of natural rewards: Relevance to addictive drugs*, J Neurosci, 22 (2002), 3306-3311.

16. A. S. Levine, C. M. Kotz, and B. A. Gosnell, *Sugars: Hedonic aspects, neuroregulation, and energy balance*, Am J Clin Nutr 78 (2003), 834S-842S.

17. M. R. Yeomans, B. J. Tepper, J. Rietzschel, and J. Prescott, Human hedonic responses to sweetness: Role of taste genetics and anatomy, Physiol Behav, 91 (2007), 264-273.

18. Y. A. Blednov, D. Walker, M. Martinez, M. Levine, S. Damak, and R. F. Margolskee, *Perception of sweet taste is important for voluntary alcohol consumption in mice*, Genes Brain Beha, 7 (2008), 1-13.

19. L. A. Lange, A. B. Kampov-Polevoy, and J. C. Garbutt, *Sweet liking and high novelty seeking: Independent phenotypes associated with alcohol-related problems*, Alcohol Alcohol, 45 (2010), 431-436.

20. H. R. Kranzler, K. A. Sandstrom, and J. Van Kirk, *Sweet taste preference as a risk factor for alcohol dependence*, Am J Psychiatry 158 (2001), 813-815.

21. N. V. Ullrich, R. Touger-Decker, J. O'Sullivan-Mallet, and B. Tepper, *PROP taster status and self-perceived food adventurousness influence food preferences*, J Am Diet Assoc, 104 (2004), 543-549.

22. A. Kokavec, *Is decreased appetite for food a physiological consequence of alcohol consumption?* Appetite, 51 (2008), 233-243.

23. V. B. Duffy, J. E. Hayes, B. S. Sullivan, and P. Faghri, *Surveying food and beverage liking: A tool for epidemiological studies to connect chemosensation with health outcomes*, Ann NY Acad Sci 1170 (2009), 1-11.

24. A. B. Kampov-Polevoy, C. Eick, G. Boland, E. Khalitov, and F. T. Crews, *Sweet liking, novelty seeking, and gender predict alcoholic status*, Alcohol Clin Exp Res, 28 (2004), 1291-1298.

25. A. B. Kampov-Polevoy, J. C. Garbutt, and D. S. Janowsky, *Association between preference for sweets and excessive alcohol intake: A review of animal and human studies*, Alcohol Alcohol, 34 (1999), 386-395.

26. A. B. Kampov-Polevoy, M. V. Tsoi, E. E. Zvartay, N. G. Neznanov, and E. Khalitov, *Sweet liking and family history of alcoholism in hospitalized alcoholic and non-alcoholic patients*, Alcohol Alcohol, 36 (2001), 165-170.

27. G. Hellekant, V. Danilova, T. Roberts, and Y. Ninomiya, *The taste of ethanol in a primate model: I. Chorda tympani nerve response in Macaca mulatta*, Alcohol, 14 (1997), 473-484.

28. A. N. Gearhardt, W. R. Corbin, and K. D. Brown, *Food addiction: An examination of the diagnostic criteria for dependence*, J Addict Med, 3 (2009), 1-7.

29. S. M. Brasser, N. Castro, and B. Feretic, *Alcohol sensory processing and its relevance for ingestion*, Physiol Behav, 148 (2015), 65-70.

30. K. C. Berridge, *Food reward: Brain substrates of wanting and liking*, Neurosci Biobehav Rev, 20 (1996), 1-25.

31. A. B. Kampov-Polevoy, J. C. Garbutt, and D. Janowsky, *Evidence of preference for a high-concentration sucrose solution in alcoholic men*, Am J Psychiatry, 54 (1997), 269-270.

32. A. S. Levine, C. M. Kotz, and B. A. Gosnell, *Sugars and fats: The neurobiology of preference*, J Nutr, 133 (2003), 831S-834S.

33. L. J. Nolan, and L. M. Scagnelli, *Preference for sweet foods and higher body mass index in patients being treated in long-term methadone maintenance*, Subst Use Misuse, 42 (2007), 1555-1566.

34. A. Scinska, E. Koros, B. Habrat, A. Kukwa, W. Kostowski, and P. Bienkowski, *Bitter and sweet components of ethanol taste in humans*, Drug Alcohol Depend, 60 (2000), 199-206.

35. K. A. Tremblay, J. M. Bona, H. R. Kranzler, *Effects of diagnosis or family history of alcoholism on the taste intensity and hedonic value of sucrose*, Am J Addict, 18 (2009), 494-499.

36. M. Wronski, D. Skrok-Wolska, J. Samochowiec, M. Ziolkowski, L. Swiecicki, P. Bienkowski, et al., *Perceived intensity and pleasantness of sucrose taste in male alcoholics*, Alcohol Alcoholism, 42 (2007), 75-79.

37. V. B. Duffy, *Associations between oral sensation, dietary behaviors and risk of cardiovascular disease (CVD)*, Appetite, 43 (2004), 5-9.

38. B. J. Tepper, *Chapter 8: 6-n-propylthioracil as a genetic marker for fat intake, obesity, and chronic disease risk: Current evidence and future promise*, In Prescott J and Tepper BJ (eds), Genetic Variation in Taste Sensitivity, (2004),155-177. Basel, NY: Marcel Dekker.

39. B. J. Tepper, E. A. White, Y. Koelliker, C. Lanzara, P. d'Adamo, and P. Gasparini, *Genetic variation in taste sensitivity to 6-n-propylthioracil and its relationship to taste perception and food selection*, International Symposium on Olfaction and Taste: Ann NY, Acad Sci, 1170 (2009), 126-139.

40. M. R. Bajec, and G. J. Pickering, *Association of thermal taste and PROP responsiveness with food liking, neophobia, body mass index and waist circumference*, Food Qual Pref, 21 (2010), 589-601.

41. V. B. Duffy, A. C. Davidson, J. R. Kidd, K. K. Kidd, W. C. Speed, A. J. Pakstis, et al., *Bitter receptor gene (TAS2R38), 6-n-propylthiouracil (PROP) bitterness and alcohol intake*, Alcohol Clin Exp Res 28 (2004), 1629-1637.

42. J. E. Hayes, and R. S. J. Keast, *Two decades of supertasting: Where do we stand?* Physiol Behav 104 (2011), 1072-1074.

43. G. J. Pickering, G. Haverstock, D. DiBattista, *Evidence that sensitivity to 6-n-propylthiouracil (PROP) affects perception of retro-nasal aroma intensity*, J Food Agric Environ 4 (2006), 15-22.

44. B. J. Tepper, *Nutritional implications of genetic taste variation: The role of PROP sensitivity and other taste phenotypes*, Annu Rev Nutr 28 (2008), 367-388.

45. G. J. Pickering, and G. Robert, *Perception of mouthfeel sensations elicited by red wine is associated with sensitivity to 6-n-propylthiouracil (PROP)*, J Sens Stud 21 (2006), 249-265.

46. G. J. Pickering, K. Simunkova, D. DiBattista, *Intensity of taste and astringency sensations elicited by red wines is associated with sensitivity to PROP (6-n-propylthiouracil)*, Food Qual Pref, 15 (2004), 147-154.

47. L. R. Intranuovo, and A. S.Powers, *The perceived bitterness of beer and 6-n-propylthiouracil (PROP) sensitivity*, Ann Acad NY Sci, 855 (1998), 813-815.

48. G. J. Pickering, A. K. Jain, R. Bezawada, *Segmentation and drivers of wine liking and consumption in US wine consumers*, Int J Wine Res 6 (2014), 1-11.

49. D. Krahn, J. Grossman, H. Henk, M. Mussey, R. Crosby, and B. Gosnell, *Sweet intake, sweet-liking, urges to eat, and weight change: Relationship to alcohol dependence and abstinence*, Addict Behav 31 (2006), 622-631.

50. M. E. Fischer, K. J. Cruickshanks, J. S. Pankow, N. Pankratz, C. R. Schubert, G. H. Huang, et al., *The associations between 6-n-propylthiouracil (PROP) intensity and taste intensities differ by TAS2R38 haplotype*, J Nutrigenet and Nutrigenom 7 (2014), 143-152.

51. M. L. Pelchat and S. Danowski, *A possible association between PROP-tasting and alcoholism*, Physiol Behav, 51 (1992), 1261-1266.

52. H. R. Kranzler, P. J. Moore, V. M. Hesselbrock, *No association of PROP taster status and paternal history of alcohol dependence*, Alcohol Clin Exp Res, 20 (1996), 1496-1500.

53. H. R. Kranzler, K. Skipsey, V. Modesto-Lowe, *PROP taster status and parental history of alcohol dependence*, Drug Alcohol Depend, 52 (1998), 109-113.

54. G. J. Pickering, and C. W. Cullen, *The influence of taste sensitivity and adventurousness on Generation Y's liking scores for sparkling wine*, Proceedings of the Fourth International Conference of the Academy of Wine Business Research, Siena, Italy, July 17-19 (2008), (CD).

55. D, Reinert, and J. P. Allen, *The alcohol use disorders identification test (AUDIT): A review of recent research*, Alcohol Clin Exp Res, 26 (2002), 272-279.

56. K. L. Barry, and M. F. Fleming, *The alcohol use disorders identification test (AUDIT) and the SMAST-13: Predictive validity in a rural primary care sample*, Alcohol Alcohol, 28 (1993), 33-42.

57. T. M. Crews, and K. J. Sher, *Using adapted short MASTs for assessing parental alcoholism: Reliability and validity*, Alcohol Clin Exp Res, 16 (1992), 576-584.

58. L. Zhao, S. V. Kirkmeyer, and B. J. Tepper, *A paper screening test to assess genetic taste sensitivity to 6-n-propylthiouracil*, Physiol Behav, 78 (2003), 625-633.

59. L. M. Bartoshuk, V. B. Duffy, K. Fast, B. G. Green, J. Prutkin, and D. J. Snyder, *Labeled scales (e.g., category, likert, VAS) and invalid across-group comparisons: What we have learned from genetic variation in taste*, Food Qual Pref, 14 (2002), 125-138.

60. G. J. Pickering, A. K. Jain, and R. Bezawada, *Super-tasting gastronomes? Taste phenotype characterization of foodies and wine experts*, Food Qual Pref, 28 (2012), 95-91.

61. G. J. Pickering, and J. Hayes, *The influence of biological, experiential and psychological factors in wine preference segmentation*, Aust J Grape Wine Res, 23 (2017), 154-161.

62. K. J. Cruickshanks, C. R. Schubert, L. M. Bartoshuk, C.W. Acher, C. T. Baldwin, G. H. Huang, et al. *Measuring taste impairment in epidemiologic studies-The Beaver Dam Offspring Study*, Ann NY Acad Sci 1170 (2009), 543-552.

63. M. Meilgaard, G. V. Civille, and B. T. Carr, *Sensory Evaluation Techniques*, 3rd ed. Boca Raton: CRC Press LLC (1999).

64. D. R. Peryam, and P. J. Pilgrim, *Hedonic scale method for measuring food preferences*, Food Technol, 11 (1957), 9-14.

65. L. M. Bartoshuk, V. B. Duffy, B. G. Green, H. J. Hoffman, C. W. Ko, L. A. Lucchina, et al., *Valid across-group comparisons with labeled scales: The gLMS versus magnitude matching*, Physiol Behav, 82 (2004), 109-114.

66. J. C. Wang, A. L. Hinrichs, S. Bertelsen, H. Stock, J. P. Budde, D. M. Dick, et al., *53 Functional variants in TAS2R38 and TAS2R16 influence alcohol consumption in high-risk families of african-american origin*, Alcohol Clin Exp Res. 31 (2007), 209-215.

67. S. T. DiCarlo, and A. S. Powers, *Propylthiouracil tasting as a possible genetic association marker for two types of alcoholism*, Physiol Behav, 64 (1998), 147-152.

68. R. L. Spencer, and K. E. Hutchison, *Alcohol, aging, and the stress response*, Alcohol Res Health, 23 (1999), 272-283.

69. L. S. Greene, J. A. Desor, O. Mailer, *Heredity and experience: their relative importance in the development of taste preference in man*, J Comp Physiol Psychol, 89 (1975), 279-284.