

## Sweet and bitter tastes of alcoholic beverages mediate alcohol intake in of-age undergraduates

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### Abstract

Alcoholic beverages are complex stimuli, giving rise to sensations that promote or inhibit intake. Previous research has shown associations between 6-*n*-propylthiouracil (PROP) bitterness, one marker of genetic variation in taste, and alcohol behaviors. We tested the PROP bitterness and alcohol intake relationship as mediated by tastes of sampled alcoholic beverages. Forty-nine undergraduates (mean age=22 years) participated. According to the Alcohol Use Disorders Identification Test (AUDIT), only 3 of 49 subjects reported patterns indicating problematic drinking. Participants used the general Labeled Magnitude Scale to rate PROP bitterness and tastes from and preference for Pilsner beer, blended scotch whiskey, instant espresso and unsweetened grapefruit juice. Alcohol intake was reported over a typical week. Regression analysis tested the hypothesis that PROP bitterness influenced alcohol bitterness and sweetness, which in turn predicted alcohol intake. Those who tasted less PROP bitterness tasted all beverages as less bitter and more preferred. Sweetness of scotch was significantly greater in those who tasted PROP as least bitter. For scotch, greater sweetness and less bitterness from sampled scotch were direct predictors of greater alcohol intake. For beer, preference ratings were better predictors of alcohol intake than the bitter or sweet tastes of the sampled beer. These findings support that PROP bitterness predicts both positive and negative tastes from alcoholic beverages and that those tastes may predict alcohol intake. The college environment may attenuate direct effects of PROP bitterness and intake. Here, PROP bitterness does not predict alcohol intake directly, but acts instead through sweet and bitter tastes of alcoholic beverages.

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### 1. Introduction

Alcoholic beverages give rise to both unpleasurable and pleasurable tastes. Humans are born disliking bitterness [1] and bitterness is typically considered aversive [2]. Specific to beverages, increased bitterness associates with decreased liking of green tea, grapefruit juice [3] and beer [4]. Humans may acquire a preference for initially aversive stimuli whether or not they are addictive [5] through a

range of mechanisms including mere exposure, dissipation of neophobia, physiological consequences of ingestion, Pavlovian conditioning, and social valuation [6]. In contrast, humans are born with an innate preference for sweet, first illustrated over 30 years ago by Steiner (see Ref. [1] for a recent review). Alcoholic beverages can evoke sweet sensations and thus a pleasant response via the ethanol directly [7] or by residual sugars or sweet mixers in the beverages. Moreover, sweet sensations may promote intake for reasons beyond simple positive sensory factors as sweet tastants have been shown to trigger beta-endorphin release [8,9]. Some human and animal data support associations between sweet preference and alcohol intake (see Ref. [10] for a review) while others do not [11,12].

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Consumers report that “taste” is the single most important factor in food and beverage selection [13], although this colloquial usage should be taken to refer to the overall orosensory experience. Specific to alcohol selection, disliking of the smell or taste of alcoholic beverages was the first or second most reported reason for abstinence among adolescents from religious traditions that do not proscribe drinking [14]. Thus alcohol use may be hindered if the beverages are perceived as too bitter and supported if the beverages are perceived as sweet. However, because pure ethanol elicits sensations in multiple chemosensory modalities, it is uncertain if taste alone drives intake.

Studying the effect of taste on alcohol preference and intake is complicated by the fact that individuals vary in the level of pleasurable and unpleasurable sensations from alcohol. One source of variation is genetic, characterized by the perceived bitterness of PROP. Supertasters, individuals who perceive the most intense bitterness from PROP [15], experience more irritation [16,17] and greater bitterness from ethanol than do nontasters [17]. Likewise, supertasters experience the most bitterness from some beers [4]. Prescott and Swain-Cambell [16] speculated that differential response to the irritancy of ethanol leads nontasters to develop a preference for alcohol much more rapidly and/or frequently than supertasters. Three studies support this speculation: one found that nontasters were more likely to be high consumers of beer than were tasters [18]; another found that nontasters drank more beer in the first year of beer drinking [4]; and the third report found that nontasters consumed more alcoholic beverages than did supertasters [19]. The relationship between PROP tasting and alcoholism risk is less clear as some studies find that PROP

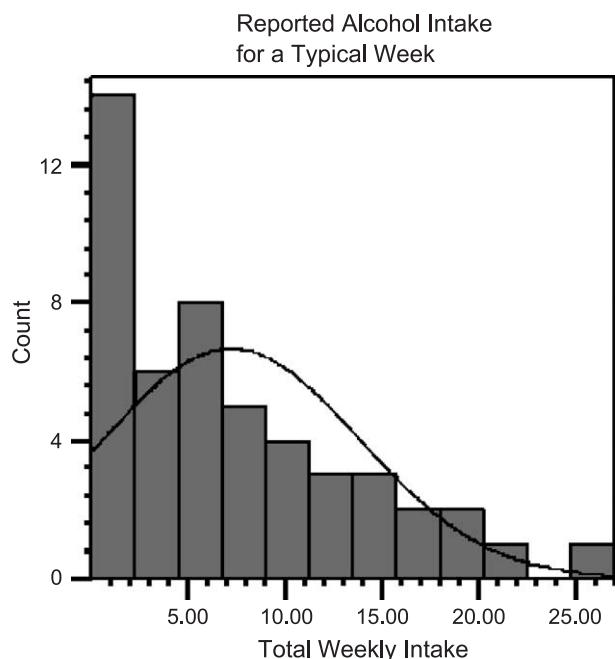


Fig. 1. Typical weekly alcohol intake in 49 undergraduates.

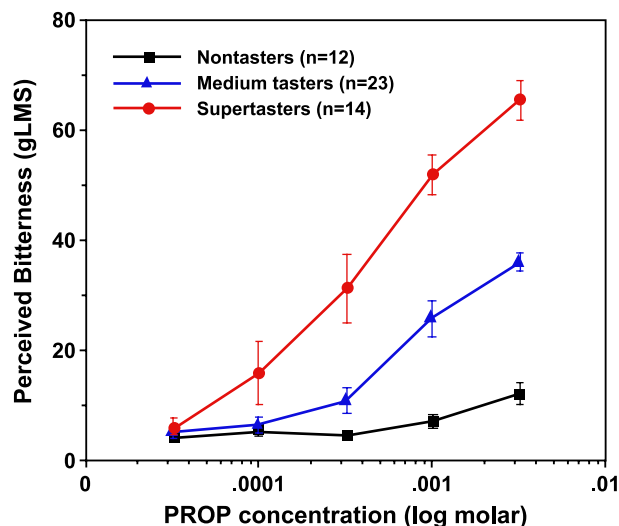


Fig. 2. The bitterness of a PROP concentration series as measured on the gLMS. Subjects were classified according to bitterness of 3.2 mM PROP, resulting in 12 nontasters ( $\leq 22$ ), 23 medium tasters (between 23 and 53), and 14 supertasters ( $\geq 53$ ).

nontasters are more likely to have a family history of alcoholism [20,21] while others fail to find this relationship [22,23].

Recently, Kim and colleagues [24] showed that the TAS2R38 gene on chromosome 7 (7q36)—a member of the bitter taste receptor family—associated with the ability to taste phenylthiocarbamide. PTC is chemically related to PROP; both share a common N=C=S chemical group. There are two common molecular forms of the TAS2R38 gene defined by three nucleotide polymorphisms that result in three amino acid substitutions Proline–Alanine–Valine (PAV) [24,25]. The PAV molecular form is common in humans and is associated with “tasting”; the other common form, Alanine–Valine–Isoleucine (AVI), is associated with “nontasting.” Individuals who are heterozygous for the TAS2R38 gene (PAV/AVI) and those homozygous PAV/PAV taste PROP as more bitter than AVI/AVI homozygotes [26]. Data support associations between the TAS2R38 gene and alcohol intake; individuals who are AVI/AVI consume alcoholic beverages significantly more frequently than PAV/AVI heterozygotes or PAV/PAV homozygotes [27].

Gene–environment interactions may alter the amount of variation in alcohol intake explained by genetic factors. Dick et al. [28] found that environment can heavily moderate genetic effects on adolescent alcohol usage, with a fivefold difference in genetic effects when comparing environmental extremes. The ability of PROP bitterness to predict alcohol intake may be more or less pronounced in social and physical environments that are highly supportive of alcohol consumption. The effects may be less pronounced in environments that make alcoholic beverages readily available, such as public places that account for 80% of young adults’ total alcohol intake [29]. PROP effects also may be less pronounced if social pressure to over-

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