# The influence of genetic taste markers on food acceptance[1–3]

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**ABSTRACT** Genetically mediated sensitivity to the bitter taste of phenylthiocarbamide (PTC) and 6-*n*-propylthiouracil (Prop) has long been associated with enhanced sensitivity to other sweet and bitter compounds. New studies suggest that tasters and supertasters of Prop may also differ from nontasters in their taste preferences and in their patterns of food rejection and food acceptance. One question is whether the acceptability of bitter-tasting vegetables is influenced by Prop taster status. Cruciferous vegetables are among the major dietary sources of potentially chemoprotective agents in cancer control, and their consumption is reported to alter cancer risk. Strategies aimed at dietary change in individuals or groups should consider the role of genetic taste markers and their potential influences on food preferences and dietary habits. *Am J Clin Nutr* 1995;62:506–11.

**KEY WORDS** Phenylthiocarbamide, 6-*n*-propylthiouracil, genetic taste markers, bitter taste, hedonic responses, food rejection, cruciferous vegetables, cancer risk

## INTRODUCTION

The sense of taste is a powerful predictor of food selection. Human infants show an innate pleasure response to sweet taste, but dislike bitterness and so reject bitter-tasting foods (1). Whereas sweetness serves as a sensory cue for food energy, bitterness often predicts toxicity. Plant-derived alkaloids and glycosides both taste bitter. Because many poisons in the environment also have a bitter taste, being able to identify and reject bitter substances might have a certain evolutionary advantage (1).

The sensitivity to bitter taste is a heritable trait (2, 3). It has long been known that two substances, phenylthiocarbamide (PTC) and 6-*n*-propylthiouracil (Prop) taste bitter to some people but are tasteless to others (4, 5). More recent studies in taste psychophysics have distinguished between Prop nontasters, regular tasters, and so-called supertasters (6). That distinction was not only behavioral. Anatomical studies confirmed that supertasters, most of whom are women, had the most fungiform taste buds and the highest density of fungiform papillae (6).

The genetically mediated ability to taste PTC and Prop has long been associated with enhanced sensitivity to other sweet and bitter compounds (5). New studies suggest that Prop taster or supertaster status may also influence taste preferences and even food acceptance (7, 8). There have been further suggestions of additional links between Prop tasting and hormonal factors (9), and between Prop nontasting and alcoholism (10). These studies provide new evidence for a taste-mediated link between genetics, food preferences, and perhaps dietary habits. Exploring these novel connections and their implications for clinical nutrition is the purpose of this review.

## THE GENETICS OF TASTE

Studies on the genetics of taste perception began in 1931 with the accidental finding that crystals of PTC tasted very bitter to some people but not to others (4). Studies of families showed that approximately one-third of the respondents were insensitive or "blind" to the bitter taste of PTC (11, 12). PTC taste sensitivity was initially thought to follow a simple Mendelian pattern, with taste deficiency inherited through a single genetic locus as an autosomal recessive trait (11, 12). PTC tasters were reported to be homozygous or heterozygous for the dominant gene, whereas nontasters carried two recessive genes (4, 13). According to early studies, when neither parent could taste PTC, none of the children could taste it.

Studies in genetic modeling now suggest that the traditional single locus, two-allele model of PTC sensitivity may not provide the best fit to the data. Reports of taster offspring from nontaster parents (14) suggested a model with incomplete penetrance, and there have been reports of multilocus and multiallele models (15). In one study based on PTC taste data for 1152 individuals from 120 families the best fit was obtained with a two-locus model in which one locus controlled PTC taste perception and the other locus controlled a more general taste acuity (16). This model is in accord with taste studies showing that PTC nontasters are divisible into two groups (17). One group is characterized by a specific PTC taste deficit, whereas the other shows a low sensitivity to a wide range of taste stimuli (17).

PTC taste blindness served initially as a marker of genetic inheritance. Numerous studies explored the frequency of the nontaster gene in different populations around the globe (18,

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19). Generally, the highest proportions of PTC nontasters were found in Europeans and North Americans (26–30%), whereas the nontasting gene was far less common among Asians (6–10%), Africans, and Native Americans. More recent taste studies have focused on adolescent growth and development (9). According to one report, girls who could taste PTC reached maturation ≈3.8 mo earlier than did nontasters, suggesting that the PTC polymorphism may involve the hypothalamic-pituitary-gonadal axis and the sex hormone pathways.

## BITTER TASTE OF PTC AND PROP

The earliest studies on the genetics of taste perception used PTC crystals (4, 11, 12). Their use was later followed by PTC-impregnated filter paper and PTC solutions in water (20–22). The current preference is for solutions of another bitter compound, Prop (23). Prop lacks the faint sulfur smell of the most concentrated PTC solutions and is in general less poisonous (3).

Taste thresholds are now determined by using a modified up-and-down procedure that involves forced-choice judgments and successive serial dilutions of the bitter compounds. The Prop series traditionally started with solution 14, containing 1.021 g Prop/L (corresponding to 0.006 mol/L), with progressive dilutions down to solution 1 (3). Current methods call for 15 Prop solutions ranging in concentration from 0.000001 to 0.0032 mol/L that increase in one-quarter log increments on the molar scale (24, 25).

The subjects were generally presented with samples of Prop solutions and distilled water and were asked to judge which of the two samples had the bitter taste (22, 23). Wrong answers led to the presentation of more concentrated solutions, whereas correct answers led to a second presentation of the same solution. Reversal points were defined as the concentration at which a string of correct responses turned to an incorrect response or vice versa. After discarding the first reversal, the calculated Prop threshold was the geometric mean of the subsequent six reversal points (22, 23).

Whereas taste thresholds for most compounds follow a Gaussian (inverted U shape) distribution, thresholds for PTC and Prop showed a bimodal distribution, dividing the subject population into distinct groups of tasters and nontasters (3, 26). As shown in **Figure 1**, the antimode that separated tasters from nontasters fell between Prop solutions 9 and 10. Bartoshuk (23) defined Prop tasters as having thresholds of $< 1.0 \times 10^{-4}$ mol/L (equivalent to solution 8) and nontasters as having thresholds $> 2.0 \times 10^{-4}$ mol/L, equivalent to solution 9 (21, 23). Subsequent distinctions between nontasters, tasters, and supertasters were based both on Prop detection thresholds and on the ratio of intensity ratings for suprathreshold solutions of Prop and sodium chloride (6). According to Bartoshuk, about one-quarter of Americans are nontasters, one-quarter are supertasters, and one-half are regular tasters.

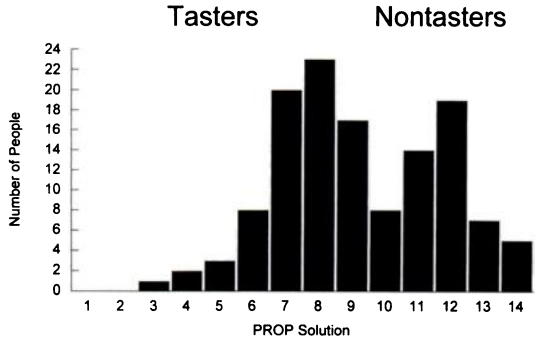


**FIGURE 1.** Taste thresholds for 6-*n*-propylthiouracil (Prop). Threshold distribution is bimodal, separating the population into tasters and nontasters. (Data from reference 27.)

## PROP AND TASTE RESPONSIVENESS

Inherited taste blindness was initially thought to involve only those bitter compounds that carried the -N-C=S group (3). However, later studies showed that Prop sensitivity predicted taste thresholds to numerous other substances, including some present in everyday foods (21, 28).

Prop tasters perceived low concentrations of caffeine as more bitter than did nontasters. The concentration of caffeine found in a cup of brewed coffee (0.004 mol caffeine/L) was below threshold for most nontasters but above threshold for most tasters (28). At concentrations close to those used in soft drinks (0.0015 mol/L), saccharin tasted more bitter to tasters than to nontasters (21). Tasters of PTC and Prop also perceived sodium benzoate and potassium chloride to be more bitter than did nontasters (23). Sodium benzoate is a widely used preservative, whereas potassium chloride is the most frequently used salt substitute for people consuming low-sodium diets.

Not only bitter compounds have been studied. Further investigations (22) showed that some concentrations of sucrose, neohesperidin dihydrochalcone (an intense sweetener), and saccharin tasted more intensely sweet to Prop tasters than to nontasters. Oral perception of pain was also more intense among Prop tasters. The application of capsaicin, the active ingredient of chili peppers, to the apex of the tongue produces a reproducible burning sensation and a salivation reflex. Prop tasters rated capsaicin burn as more intense than did nontasters (29), perhaps because pain fibers are closely associated with taste buds (30).

## PROP AND TASTE PREFERENCES

Although the impact of Prop taster status on taste sensitivity was the focus of numerous reports, the connection between Prop taste sensitivity and sweet taste preference was explored in only one study (7). Respondents in that study were divided into sweet likers and dislikers. Subjects whose hedonic preferences for sugar solutions rose with increasing sweetness were defined as likers, whereas those whose hedonic responses declined with increasing sweetness were defined as dislikers. Of the 144 adult subjects (51 men and 93 women), 110 were Prop tasters and 34 were nontasters, as determined by the solution method. Prop tasters were more likely to dislike the taste of sweet solutions (81/110), whereas nontasters were almost always sweet likers (31/34). The latter relation was also observed among 72 children aged 7–10 y.

Looy and Weingarten's study (7) provided the first link between a genetic taste marker and sweet taste preferences. However, note that hedonic preferences are subject to bias. Sweet dislikers are usually dieting women, and a reported

dislike of sweet solutions has been observed in obesity and in anorexia nervosa (31). Given that Prop supertasters are also mostly women, the link between sweet taste preferences and Prop taster status might be influenced by sex (32). Furthermore, preferences for sweet solutions may not predict liking for sweet or sweet and high-fat foods.

## BITTER TASTE AND FOOD REJECTION

Whereas many factors, including taste, contribute to food acceptance, unpleasant taste is often the chief criterion for food rejection (1). In fact, the very existence of PTC-tasting genes in humans was thought to be linked to the genetic advantage of being able to detect and avoid bitter poisons (33). The most frequently cited example of such taste rejection was the reported avoidance by PTC tasters of bitter antithyroid compounds found in raw cruciferous vegetables, broccoli, cabbage, and Brussels sprouts (3).

Goitrin (1,5 vinyl-2-thiooxazolidone) and isothiocyanates are bitter PTC-related compounds caused by hydrolysis of glucosinolates naturally present in raw cabbage (34). Their consumption has been associated with higher prevalence of endemic goiter (35). However, the antithyroid activity of these plant-derived compounds is no longer the chief focus of research interest. Recent studies suggest that isothiocyanates derived from cruciferous vegetables may have a chemopreventive action in cancer control (36). Similarly, flavonoids such as naringin, the bitter component of grapefruit juice, may be among the biologically active compounds (37). However, bitter-tasting foods are frequently disliked, and bitter taste is one reason for low acceptance of cruciferous vegetables.

Linking PTC and Prop taste sensitivity to a greater number of food dislikes was the aim of numerous early studies (2). Fox (4) first observed that PTC tasters showed consistently low "like" responses to foods and beverages. Fischer and Griffin (38) noted that people sensitive to Prop tended to report more dislikes for common foods than did nontasters. In that study, 48 subjects completed a checklist of 118 foods. The list included green and cruciferous vegetables (cabbage, Brussels sprouts, spinach, and kale) as well as rhubarb, sauerkraut, beer, coffee, and various sharp cheeses (39, 27). Greater taste sensitivity to Prop was linked to a higher percentage of foods disliked. In contrast, sensitivity to sucrose (sweet), sodium chloride (salty), or hydrochloric acid (sour), was not connected to food likes or dislikes (39, 27).

Glanville and Kaplan (40) examined preferences for names of sharp- or mild-tasting foods as a function of Prop taster status in a group of 187 adults, average age 38 y. The sample included 39 husband-wife pairs, 16 pairs of monozygotic twins, and 10 pairs of dizygotic twins. Thresholds for Prop were established by using the Harris-Kalmus procedure (20). The subjects then completed a food preference checklist. Among checklist items were preferences for black coffee or coffee with milk or sugar, liking for mild or sharp-tasting cheeses, and sharp salad dressings, and liking for such foods as grapefruit juice, lemon juice, sauerkraut, vinegar, and horseradish. Prop taste sensitivity was again linked to a higher number of foods disliked. As shown in **Figure 2**, there was also a significant correlation between low Prop taste thresholds (that is, higher sensitivity) and stated preferences for mild rather than for sharp-tasting foods (40).

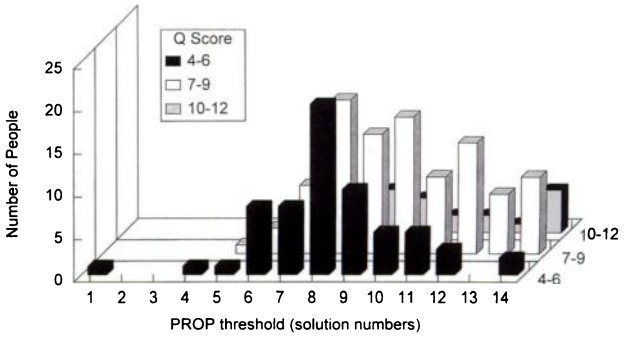


**FIGURE 2.** PROP taste thresholds and scores on food-preference questionnaire. Higher Q scores denote preferences for sharp-tasting foods. Respondents who preferred sharp- and bitter-tasting foods were more likely to be nontasters of PROP (from reference 40).

Another study explored PTC taste sensitivity and preferences for food names among 98 monozygotic and 67 dizygotic twins in Hungary (41). A checklist of 69 different foods was used. Tasters reported lower preferences for salami, anchovy paste, brown bread, beer, kale, and whipped cream (41). On the other hand, a study of 13 monozygotic and 10 dizygotic twin pairs (42), although confirming that sensitivity to PTC was a heritable trait, showed only minimal effects of heritability on self-reported food preferences.

Although it might appear that greater sensitivity to bitter taste was consistently linked with more food dislikes, some of the methods used were highly imprecise. Many studies used the inaccurate PTC crystal method to assess taster status and virtually all studies used preference checklists of food names as a proxy measure of food selection. Rather than measure consumption, food checklists merely assess positive or negative attitudes toward a given food name (43). Biases toward food acceptance or rejection can be influenced by many factors, including prior experience, sociocultural variables, as well as by attitudes and beliefs regarding health, body weight, and dieting.

Perhaps as a result, the reported data were inconsistent. In one study it was reported that, contrary to expectations, taste thresholds to PTC were not linked to the dietary intakes of foods containing antithyroid substances (44). In that study, 282 subjects were separated into 146 tasters and 113 nontasters on the basis of their responses to PTC-impregnated filter paper rather than to PTC solutions. Dietary behaviors were assessed by using a list of 25 foods, including raw cabbage, carrots, celery, strawberries, spinach, oysters, peaches, and pears. Frequency of consumption was rated on a nine-point scale that ranged from "tried once or twice" to "eaten more than once per day." There was no connection between PTC taster status and the avoidance of sources of dietary goitrogens. Although the authors acknowledged the imprecision of methods used to assess both dietary habits and taster status, they argued that the use of a large number of subjects should have produced significant results (44).

Later studies reported only minimal effects of PTC taster status on the consumption of cruciferous vegetables (45, 46). PTC taster status was assessed by using solutions and a forced-choice procedure. The first study (45), conducted with 32 women aged 53–76 y, showed that PTC tasters rated the overall

flavor intensity of cooked cabbage samples higher than did nontasters. However, taste preferences and reported vegetable use were unaffected by PTC taster status (45).

Subjects in the second study (46) were 18 tasters and 18 nontasters, all women. Vegetable consumption was determined using a modified food-frequency questionnaire that included 11 cruciferous vegetables: broccoli, Brussels sprouts, red cabbage, white cabbage, cauliflower, collards, kale, kohlrabi, radishes, white turnips, and watercress. Also included were two noncruciferous but bitter vegetables: spinach and endive.

The questionnaire instrument probed for daily, weekly, monthly, or less than monthly use of vegetables. However, because most of the subjects consumed vegetables rarely if at all, the frequency scale had to be collapsed into two categories only, use and non-use. Not surprisingly, given the small number of subjects and a dichotomous dependent variable, the data showed no major trends. Although PTC nontasters were more likely than tasters to use raw radishes, turnips, and watercress, the perception of bitter taste did not offer an explanation for these results. The authors suggested that social and cultural factors may outweigh taste in determining food selection (46).

Only one published study examined the effect of genetic sensitivity to the bitter taste of Prop on taste preferences for a range of different foods (8). This study was conducted in 30 children, aged 5–7 y, whose food preferences might be expected to be least biased by health beliefs and nutritional concerns. The authors' hypothesis was that taster children would be more likely to avoid strong- and sharp-tasting foods. Taste sensitivity to Prop was assessed by using the accurate solution method, including the tasting of both threshold and suprathreshold solutions to help establish taster status.

Sensory tests of 11 foods and beverages used two methods of measuring preference: the order of food selection and a hedonic rating based on a five-point category scale. Both techniques had been validated in previous studies with even younger children. These sophisticated techniques were supplemented with a verbally administered food-preference questionnaire (8).

Even so, the results were inconclusive. The most sensitive test of food preference was the order of selection of the food items. Tasters selected sharp cheddar cheese later than did nontasters, but selected coffee and milk earlier. No difference was observed for spinach or for raw and cooked broccoli, although tasters reportedly always chose vegetables later than did nontasters. Tasters also gave lower hedonic ratings to cheese and spinach than did nontasters and rated cheese lower on the food preference list. The authors suggest that the dislike of sharp cheese might be based on the bitter taste of calcium; however, cheese does contain other bitter-tasting compounds.

In general, the heritability of food preferences is said to be surprisingly low. In one study of parent-child resemblances in food preference (47), a sample of 118 college students and their parents completed preference checklists for 12 different foods. Parent-child correlations were low, averaging 0.16. However, substantially higher correlations were obtained for hot sauce on foods, black coffee, and beer. It may be that studies on family resemblances in food preference should focus more on bitter and hot and spicy foods, and possibly on alcohol.

## PROP NONTASTING AND ALCOHOLISM

Taste blindness to PTC and Prop might be a genetic marker for alcoholism. The heritable component in alcoholism has been described in numerous studies. However, although some investigators have observed a higher proportion of Prop nontasters among alcoholics, others have not (48).

The link between Prop taster status and alcoholism is thought to be mediated through hedonic preference. Supertasters perceived 10% ethyl alcohol to be more bitter than did nontasters (49). Whole-mouth tests of ethanol solutions (10–50%) showed that supertasters perceived more bitterness and experienced more irritation than did nontasters. To dispel the concern that the association between Prop nontasting and alcoholism may be caused by the excessive use of alcohol, Prop taster status was examined in adult children of alcoholics (10). A much higher proportion of nontasters of Prop was found among children of alcoholics than among children of nonalcoholics, suggesting a genetic link between Prop tasting and alcoholism.

## IMPLICATIONS FOR CLINICAL NUTRITION

The dietary pattern that appears to be most likely to prevent cancer is a diet rich in vegetables and fruit (50, 51). Although increased risk for cancer has been linked to elevated fat consumption, another characteristic of high-fat diets, namely less frequent consumption of vegetables and fruit, may be the link that more accurately explains this connection (52). Diets high in fat are typically low in fiber, antioxidant vitamins, folate, and other plant compounds that have biological activity—the phytochemicals (52, 53).

One group of these compounds, the phytoestrogens, may reduce risk for hormone-dependent cancers by binding to hormone receptors, and ultimately, reducing the circulating concentrations of the metabolically active form of the hormone (54). The phytoestrogen lignans are found in vegetables and some fruit, such as bitter-tasting berries. Another type of phytochemical that may reduce cancer risk is the indoles, found in the cruciferous vegetables (54, 55). As mentioned above, flavonoids such as hesperidin, naringin, and narirutin are provided by foods such as citrus fruits, onions, and kale. These compounds function as antioxidants and also induce cytochrome P-450 enzymes that may metabolize harmful carcinogens and other cancer-promoting factors.

Carotenoids are probably the most widely recognized phytochemicals, found in deeply pigmented vegetables and fruit such as carrots, spinach, and broccoli (51). Nearly 600 of these biologically active compounds exist; vegetables and fruit are the major food sources in the US diet. In addition to anticarcinogenic functions, carotenoids may also reduce risk for cardiovascular disease and other chronic health problems (51, 56). For example, Seddon et al (57) found the carotenoids lutein and zeaxanthin, which are primarily obtained from dark-green leafy vegetables such as spinach and greens, to be associated with decreased risk for age-related macular degeneration.

Attempts to increase vegetable consumption in the United States have focused on nutrition education and behavioral change. Yet little is known about the formation of likes and dislikes for vegetables, or about the role of genetic taste markers in determining food preferences and food aversions.

Generally, survey studies show that vegetables, particularly cruciferous vegetables, are among the most disliked of foods.

The effect of genetic factors on food selection has acquired a new importance in cancer research. Whereas cruciferous vegetables were once regarded as being hazardous to health, they are now recognized as major sources of possibly chemoprotective dietary factors. If PTC and Prop taster status helps to determine food preferences and food consumption, it might turn out to be a genetic marker for some of the major diet-related chronic diseases. However, the taste-mediated connection between genetics and nutrition is still tenuous, awaiting more research on genetic factors that contribute to the patterns of food selection.

## REFERENCES


1. Rozin P, Vollmecke TA. Food likes and dislikes. Annu Rev Nutr 1986;6:433–56.
2. Drewnowski A. Genetics of taste and smell. In: Simopoulos AP, Childs B, eds. Genetic variation in nutrition. World Rev Nutr Diet 1990;63:194–208.
3. Kalmus H. Genetics of taste. In: Beidler LM, ed. Handbook of sensory physiology. Berlin: Springer-Verlag, 1971:165–79.
4. Fox AF. The relationship between chemical constitution and taste. Proc Natl Acad Sci U S A 1932;18:115–20.
5. Bartoshuk LM. Separate worlds of taste. Psychology Today 1980; 14(4):48–63.
6. Reedy FE, Bartoshuk LM, Miller IJ, Duffy VB, Yanagisawa K. Relationship among papillae, taste pores and 6-*n*-propylthiouracil (PROP) suprathreshold taste sensitivity. Chemical Senses 1993;18: 618–9 (abstr).
7. Looy H, Weingarten HP. Facial expressions and genetic sensitivity to 6-*n*-propylthiouracil predict hedonic response to sweet. Physiol Behav 1992;52:75–82.
8. Anliker JA, Bartoshuk L, Ferris AM, Hooks LD. Children's food preferences and genetic sensitivity to the bitter taste of 6-*n*-propylthiouracil (PROP). Am J Clin Nutr 1991;54:316–20.
9. Whissell-Buechy D, Wills C. Male and female correlations for taster (PTC) phenotypes and rate of adolescent development. Ann Hum Biol 1989;16:131–46.
10. Pelchat ML, Danowski S. A possible genetic association between PROP-tasting and alcoholism. Physiol Behav 1992;51:1261–6.
11. Snyder LH. Inherited taste deficiency. Science 1931;74:151–2.
12. Blakeslee AF. Genetics of sensory thresholds: taste for phenylthiocarbamide. Proc Natl Acad Sci U S A 1931;18:120–30.
13. Fischer R. Genetics and gustatory chemoreception in man and other primates. In: Kare M, Maller O, eds. The chemical senses and nutrition. Baltimore: Johns Hopkins University Press, 1967:61–71.
14. Das SR. Inheritance of the PTC taste character in man: an analysis of 126 Rarhi Brahmin families of West Bengal. Ann Hum Genet 1958; 22:200–12.
15. Morton CC, Cantor RM, Corey LA, Nance WE. A genetic analysis of taste threshold for phenylthiocarbamide. Acta Genet Med Gemellol (Roma) 1981;30:51–7.
16. Olson JM, Boehnke M, Neiswanger K, et al. Alternative genetic models for the inheritance of the phenylthiocarbamide (PTC) taste deficiency. Genet Epidemiol 1989;6:423–34.
17. Frank RA, Korchmar DL. Gustatory processing differences in PTC tasters and non-tasters: a reaction time analysis. Physiol Behav 1985; 35:239–42.
18. Allison AC, Blumberg BS. Ability to taste phenylthiocarbamide among American Eskimos and other populations. Hum Biol 1959;31: 352–9.
19. Bhalla V. Variations in taste threshold for PTC in populations of Tibet and Ladakh. Hum Hered 1972;22:453–8.
20. Harris H, Kalmus H. The measurement of taste sensitivity to phenylthiourea (PTC). Ann Eugen (Lond) 1949;15:24–31.
21. Blakeslee AF, Salmon MR. Genetics of sensory thresholds: individual taste reactions for different substances. Proc Natl Acad Sci U S A 1935;21:84–90.
22. Fischer R, Griffin F. Pharmacogenetic aspects of gustation. Arztl-Forsch 1964;14:675–86.
23. Bartoshuk LM. Bitter taste of saccharin related to the genetic ability to taste the bitter substance 6-*n*-propylthiouracil. Science 1979;205:934–5.
24. Gent JF, Bartoshuk LM. Sweetness of sucrose, neohesperidin dihydrochalcone, and saccharin is related to genetic ability to taste the bitter substance 6-n-propylthiouracil. Chemical Senses 1983;7: 265–72.
25. Bartoshuk LM, Rifkin B, Marks LE, et al. Bitterness of KCl and benzoate: related to genetic status for sensitivity to PTC/PROP. Chemical Senses 1988;13:517–28.
26. Fischer R. Gustatory, behavioral and pharmacological manifestations of chemoreception in man. In: Ohloff G, Thomas AF, eds. Gustation and olfaction. London: Academic Press, 1971:187–237.
27. Fischer R, Griffin F, Kaplan AR. Taste thresholds, cigarette smoking and food dislikes. Med Exp Int Exp Med 1963;9:151–67.
28. Hall MJ, Bartoshuk LM, Cain WS, et al. PTC taste blindness and the taste of caffeine. Nature 1975;253:442–3.
29. Karrer T, Bartoshuk L. Capsaicin desensitization and recovery on the human tongue. Physiol Behav 1991;49:757–64.
30. Whitehead MC, Beeman CS, Kinsella BA. Distribution of taste and general sensory nerve endings in fungiform papillae of the hamster. Am J Anat 1985;173:185–201.
31. Drewnowski A. Sweetness and obesity. In: Dobbing J, ed. Sweetness. Berlin: Springer-Verlag, 1987:177–92.
32. Duffy VB, Bartoshuk LM, Weingarten HP. PROP (6-*n*-propylthiouracil) supertasters: sex and sweet preferences. Appetite 1995;24:186 (abstr).
33. Boyd WC. Taste reactions to anti-thyroid substances. Science 1950; 112:153(letter).
34. Fenwick GR, Heaney RK, Mulling WJ. Glucosinolates and their breakdown products in food and food plants. CRC Crit Rev Food Sci Nutr 1983;18:123–201.
35. Hetzel BS, Potter BJ, Dulberg EM. The iodine deficiency disorders: nature, pathogenesis and epidemiology. World Rev Nutr Diet 1990; 62:59–119.
36. Stoner GD, Morrissey DT, Heur YH, Daniel EM, Galati AJ, Wagner SA. Inhibitory effects of phenethyl isothiocyanate on *N*-nitrosobenzylmethylamine carcinogenesis in the rat esophagus. Cancer Res 1991; 51:2063–8.
37. Kuhnau J. The flavonoids: their role in human nutrition. World Rev Nutr Diet 1976;24:117–91.
38. Fischer R, Griffin F, England S, et al. Taste thresholds and food dislikes. Nature 1961;191:1328.
39. Fischer R, Griffin F. "Taste-blindness" and variations in taste threshold in relation to thyroid metabolism. J Neuropsychol 1961;3:98–104.
40. Glanville EV, Kaplan AR. Food preference and sensitivity of taste for bitter compounds. Nature 1965;205:851–2.
41. Forrai G, Bankovi G. Taste perception for phenylthiocarbamide and food choice—a Hungarian twin study. Acta Physiol Hung 1984;64: 33–40.
42. Krondl M, Coleman P, Wade J, Milner J. A twin study examining the genetic influence on food selection. Hum Nutr Appl Nutr 1983; 37A:189–98.
43. Frank RA, van der Klaauw NJ. The contribution of chemosensory factors to individual differences in reported food preferences. Appetite 1994;22:101–23.
44. Mattes R, Labov J. Bitter taste responses to phenyl thiocarbamide are not related to dietary goitrogen intake in human beings. J Am Diet Assoc 1989;89:692–4.
45. Niewind A, Krondl M, Shrott M. Genetic influences on the selection of Brassica vegetables by elderly individuals. Nutr Res 1988;8:13–20.

46. Jerzsa-Latta M, Krondl M, Coleman P. Use and perceived attributes of cruciferous vegetables in terms of genetically-mediated taste sensitivity. Appetite 1990;15:127–34.
47. Rozin P. Family resemblance in food and other domains: the family paradox and the role of parental congruence. Appetite 1991;16: 93–102.
48. Swinson RP. Genetic markers and alcoholism. Recent Dev Alcohol 1983;1:9–24.
49. Bartoshuk LM, Conner E, Grubin D, et al. PROP supertasters and the perception of ethyl alcohol. Chemical Senses 1993;18:526–7 (abstr).
50. National Research Council. Diet, nutrition and cancer. Washington, DC: National Academy Press, 1982.
51. Ziegler RG. Vegetables, fruits, and carotenoids and the risk of cancer. Am J Clin Nutr 1991;53(suppl):251S–9S.
52. Subar AF, Ziegler RG, Patterson BH, Ursin G, Graubard B. US dietary patterns associated with fat intake: the 1987 National Health Interview Survey. Am J Public Health 1994;84:359–66.
53. Weisburger JH. Nutritional approach to cancer prevention with emphasis on vitamins, antioxidants, and carotenoids. Am J Clin Nutr 1991;53:226S–37S.
54. Adlercreutz H, Mousavi Y, Hockerstedt K. Diet and breast cancer. Acta Oncologica 1992;31:175–81.
55. Liu H, Wormke M, Safe SH, Bjeldanes LF. Indolo[3,2-$\beta$]carbazole: a dietary-derived factor that exhibits both antiestrogenic and estrogenic activity. J Natl Cancer Inst 1994;86:1758–65.
56. Morris DL, Kritchevsky SB, Davis CE. Serum carotenoids and coronary heart disease. JAMA 1994;272:1439–41.
57. Seddon JM, Ajani UA, Sperduto RD, et al. Dietary carotenoids, vitamins A, C, and E, and advanced age-related macular degeneration. JAMA 1994;272:1413–20.