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# Genetic variation in taste perception: does it have a role in healthy eating?

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Taste is often cited as the factor of greatest significance in food choice, and has been described as the body's 'nutritional gatekeeper'. Variation in taste receptor genes can give rise to differential perception of sweet, umami and bitter tastes, whereas less is known about the genetics of sour and salty taste. Over twenty-five bitter taste receptor genes exist, of which *TAS2R38* is one of the most studied. This gene is broadly tuned to the perception of the bitter-tasting thiourea compounds, which are found in brassica vegetables and other foods with purported health benefits, such as green tea and soya. Variations in this gene contribute to three thiourea taster groups of people: supertasters, medium tasters and nontasters. Differences in taster status have been linked to body weight, alcoholism, preferences for sugar and fat levels in food and fruit and vegetable preferences. However, genetic predispositions to food preferences may be outweighed by environmental influences, and few studies have examined both. The Taste-buddies study aimed at taking a holistic approach, examining both genetic and environmental factors in children and adults. Taster status, age and gender were the most significant influences in food preferences, whereas genotype was less important. Taster perception was associated with BMI in women; nontasters had a higher mean BMI than medium tasters or supertasters. Nutrient intakes were influenced by both phenotype and genotype for the whole group, and in women, the AVI variation of the *TAS2R38* gene was associated with a nutrient intake pattern indicative of healthy eating.

**Food choice: Bitter taste: Phenylthiocarbamide: Propylthiouracil: *TAS2R38*: Fruit and vegetables**

A recent Irish study reported that the prevalence of overweight and obesity in 4–13-year olds is 24·6%[1], while in adults the prevalence is 61%[2]. These statistics reflect the current situation in the UK and most parts of Europe[3], and are approximately 5% behind levels in the USA[2], where the prevalence is predicted to reach almost 90% by 2030[4]. Obesity has been linked, among others, to increased risk of heart disease, cancer and depression[5], with an estimated annual cost of €70 million to the Irish health service[1]; between £2·4 billion and £2·6 billion to the UK and as much as $78·5 billion annually in the USA[4]. Recognized contributory factors to the obesity 'epidemic' include decreased levels of physical activity and an increase in adverse eating behaviours[6] such as high consumption of energy-dense foods and low fruit and vegetable consumption[7]. The reasons for particular food choices are therefore of great importance to dietitians, health-care workers and nutritionists. Fruit and vegetables are vital for good health[8,9], and the WHO recommends a minimum intake of 400 g of fruit and vegetables per day, preferably in five 80 g servings[10]. However, almost 80% of adults fail to reach this amount, with an average daily intake in Ireland of 276 g, and in children an even lower average intake of just over 200 g[11].

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**Abbreviations:** FP, fungiform papillae; MT, medium taster; NT, nontaster; PROP, propylthiouracil; PTC, phenylthiocarbamide; SNP, single nucleotide polymorphisms; ST, supertaster.
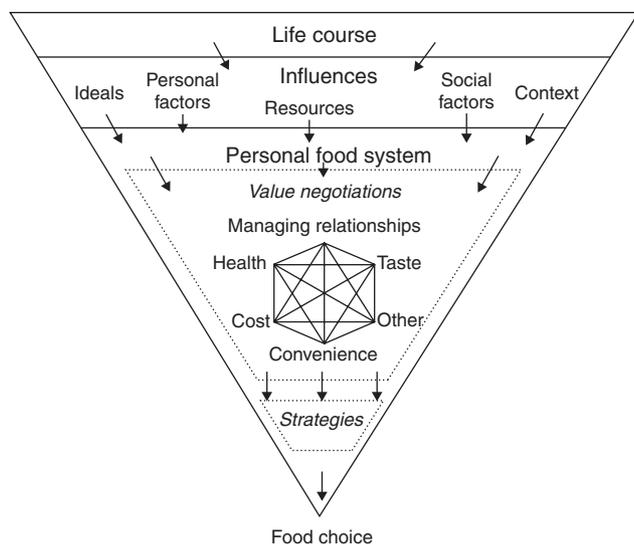*Corresponding author: Emma Feeney, email emma.feeney@ucd.ie

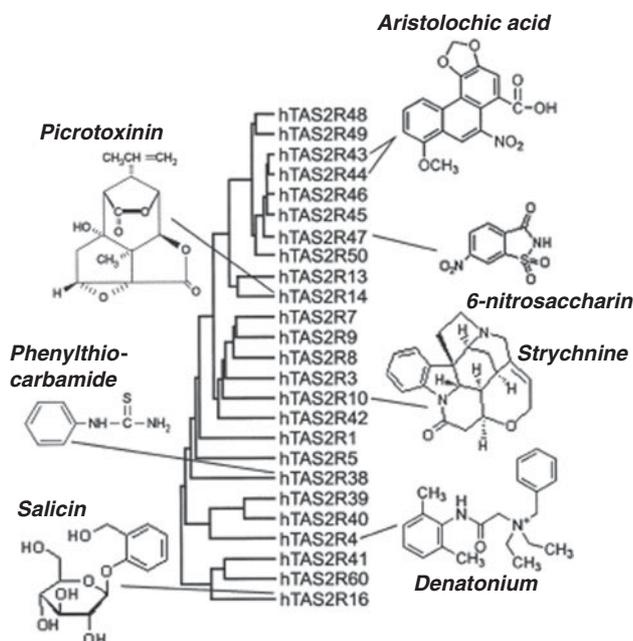Fig. 1. The Food Choice Process Model (taken from Connors et al.[17]).



Fig. 2. Phylogenetic tree of members of the TAS2R gene family and some of the ligands which bind their receptors (taken from Behrens and Meyerhof[99]).

In today's Western society, where most foods are available to most people, food choices are more than a simple matter of availability; they are governed by a multitude of complex processes[12–15]. Some of the factors involved include mood, the environment, health, allergies, convenience, hunger levels, cost, pregnancy, habit, cultural influences, sensory attributes such as colour and smell and of course taste[7,16–18]. One model that demonstrates how the wide range of different factors involved interact is the Food Choice Process Model[17] (Fig. 1).

In this model, experiences over the life course influence the personal factors that govern an individual's unique personal food system. Within this system, people manage five main food-related values. These differ in relative importance from person to person and even between eating situations.

Taste is perceived as a highly influential factor in food choice decisions[12,17,19,20]. It is listed as one of the five main values in the Food Choice Process Model[17], and has also been listed as the top reason for food choice by Americans[12]. Taste preferences have significant impact on eating behaviours[21] and may be influenced by taste perception[22–24]. Perception of taste may vary between individuals depending on genetic variations in certain taste receptor genes. Genetically determined variation in taste sensitivity in human subjects was reported for four of the basic tastes: sweet[25], bitter[26,27], sour[28] and umami, the savoury taste exemplified by the taste of monosodium glutamate[29,30]. This review examines how genetic variation in taste may influence food choice and in turn impact on nutrient intake, focusing, in particular, on variation in bitter taste and the role of the receptor gene *TAS2R38*.

## Evolution of taste and genetic variation in taste sensitivity

Taste is one of the primary means of determining the acceptability of a food and might have been critical to the survival of early human subjects[31]. Sweet and umami taste evolved as 'energetic sensors' to recognize carbohydrate and protein energy sources, whereas bitter taste evolved as a warning against toxin ingestion[31–33]. This is supported by the fact that while just three genes exist in the T1R gene family (which is responsible for the receptors for both sweet and umami taste) over twenty-five genes exist in the T2R gene family of bitter receptors (Fig. 2) which appear to have evolved from gene duplication events that expanded the range of bitter compounds to which human subjects are sensitive[34]. Further, bitterness in food tends to trigger an innate negative response or aversion[35,36] and can be detected at low levels[31,37,38] to protect against accidental ingestion of potential toxins, even in small amounts.

The perception of sweet, umami and bitter tastes are all mediated via G-coupled protein receptors, encoded by the TAS1R and TAS2R taste receptor gene families, while salty and sour tastes are transduced via ion channels[28]. A sixth possible taste, that of fat, is still under debate[39]. Salty tastes are elicited by NaCl as well as other salts and are most likely mediated by a highly specific Na channel in taste receptor cells[100], and are a vital part of ion and water homoeostasis[33]. Because of this, it seems that sensitivity to and preference for salty taste are influenced more strongly by environmental cues, such as surrounding salt concentrations, rather than individual genetic variation[28]. Similarly, there is little variation in the detection threshold of sour taste (which indicates the presence of acids) because of genetic polymorphisms[28]. For these reasons, this review will concentrate on the variation in bitter, sweet and umami taste qualities, which have been demonstrated to some degree to associate with certain genetic variations, with a specific focus on bitter taste perception.

**Table 1.** Single nucleotide polymorphisms (SNP) in TAS1R and TAS2R gene family with known functional variation in sweet, umami and bitter perception

| Gene | SNP | Association and possible mechanism, if known | Taste quality affected |
|---|---|---|---|
| *TAS1R1* | A372T[30] | T associated with high sensitivity. Mechanism unknown | Umami |
| | G1114A[95] | A associated with high sensitivity. Mechanism unknown | Umami |
| | C329T[95] | T associated with low sensitivity. Mechanism unknown | Umami |
| *TAS1R3* | R757C[30,43] | C associated with lower sensitivity. Mechanism unknown | Umami |
| | R247H[30] | H associated with increased sensitivity. Possibly influences binding with L-glutamate resulting in stronger activation of taste system. | Umami |
| | A5T[43,95] | A associated with heightened perception. | Umami |
| | C2269T[95] | T more frequent in nontasters. Mechanism unknown | Umami |
| | C1266T[41] | T alleles result in reduced promoter activity | Sweet |
| | C1572T[41] | T alleles also result in reduced promoter activity in this mutation | Sweet |
| *TAS2R16* | G516T[96] | G associated with low sensitivity | Bitter |
| *TAS2R38* | P49A[44,51] | P associated with high sensitivity, possibly through increased G-protein activation rather than ligand binding[97] | Bitter |
| | A262 V[44,51] | A associated with high sensitivity possibly through increased G-protein activation | Bitter |
| | V296I[44,51] | V associated with high sensitivity | Bitter |
| *TAS2R43* | W35S[60] | W associated with high sensitivity | Bitter |
| *TAS2R44* | W35R[60] | W associated with high sensitivity | Bitter |

The products of the three genes within the TAS1 gene family are responsible in various combinations for both sweet and umami tastes[40]. Sweeteners such as saccharin and naturally occurring sugars such as sucrose, glucose, fructose and alcohol sugars elicit a sweet taste mediated through a heterodimer of G-coupled protein receptors encoded by two genes, *TAS1R2* and *TAS1R3*[41], while a heterodimer encoded by *TAS1R1* and *TAS1R3* forms the basis of the umami receptor[42], responsible for the perception of L-glutamate[43]. A number of single nucleotide polymorphisms (SNP) have been identified in these genes in both the coding and non-coding parts of these genes. Some of these have been linked to variation in taste perception of both umami and sweet tastes (Table 1), although the mechanisms underlying most of these differences remain to be determined. Sequence analysis of the TAS1R gene family has shown that generally *TAS1R3* is the most conserved, and so less variation in the ability to detect umami is envisaged than that of sweet taste[40]. One recent study has identified two non-coding SNP in *TAS1R3*[41], and reported that a large amount of the variability in sucrose variation was explained by differences in promoter activity associated with these SNP. This is one of the first studies to show that non-coding SNP may also affect taste perceptions, and importantly, as the *TAS1R3* subunit also forms part of the umami receptor, these SNP may also contribute to some of the variation observed in umami taste[41], although this is yet to be confirmed.

The TAS2R gene family is the largest family of taste receptors, and encodes the bitter taste receptors. Most of the bitter receptor genes are located on chromosomes 7 and 12, likely as a result of gene duplications. Variation has been observed in a number of bitter receptor genes, and in general, the variation observed in bitter receptors is higher than in most other genes[40], with a total of 151 non-synonymous SNP combinations or haplotypes identified within the members of the TAS2R gene family[40]. It is not yet known if all of these correspond to variation in bitter sensitivity or to different taste receptors, but to date, variations in four of the bitter receptor genes *TAS2R16*, 38, 43 and 44, have been associated with differential bitter taste perception (Table 1). The area of bitterness sensitivity is the most extensively researched of all the taste qualities and dates back to the early 1930s, when 'taste blindness' to the bitter compounds phenylthiocarbamide (PTC) and propylthiouracil (PROP) was first noted[44]. Both compounds contain a thiourea chemical group (N—C═S) and are recognised to a degree by the receptor encoded by the PTC gene *TAS2R38*[44]. PROP is now more commonly used in taste perception studies, as PTC has a slightly sulfurous odour[45,46] and it has been reported to be toxic[47].

In 1992, the phrase 'supertasters' (ST), in relation to bitter taste perception, was coined[48] following the research in PROP perception, when it was discovered that the 'taster' group could be further divided into 'medium tasters' (MT) and 'ST' of PROP, resulting in three groups of taster: ST, MT and nontasters (NT), depending on the perceived intensity of PROP, making up 20, 50 and 30% of the population, respectively[22]. ST perceive PROP as intensely bitter, and generally also dislike the taste. The *TAS2R38* gene is responsible for the majority of the variation in bitter taste sensitivity of PTC, as well as a significant proportion of sensitivity to PROP[44,49,50]. A number of SNP have been identified within this gene (Table 2), three of which give rise to the two main haplotypes observed in over 90% of the Caucasian population[49], PAV (proline–alanine–valine at amino acid positions 49, 262 and 296 respectively), the 'taster' form, and AVI (alanine–valine–isoleucine at amino acid positions 49, 262 and 296 respectively), the 'NT' form[29,51] and can be attributed to much of the variation observed in PTC and PROP sensitivity[51].

**Table 2.** Single nucleotide polymorphisms (SNP) in *TAS2R38*

| Position | | | |
| --- | --- | --- | --- |
| Base pair | Amino acid | Allele | Amino acid encoded |
| **145** | **49** | **C** | **Pro** |
| | | **G** | **Ala** |
| 239 | 80 | A | His |
| | | G | Arg |
| **785** | **262** | **C** | **Ala** |
| | | **T** | **Val** |
| 820 | 274 | C | Arg |
| | | T | Cys |
| **886** | **296** | **C** | **Val** |
| | | **T** | **Ile** |

A total of five SNP have been observed in *TAS2R38*. The three most common, which give rise to the haplotypes PAV and AVI, are shown in bold. The less frequently observed sub-Saharan African SNP are shown in grey.

## Taste and fungiform papillae density

Another mechanism through which genetic variation might affect taste perception is through the density of fungiform papillae (FP), one of the three types of papilla structures which house the tastebuds on the tongue. The bitter *TAS2R38* gene has been suggested to influence FP density, as PROP sensitivity has been generally reported as positively correlated with FP density[52–56] and thus tasters of PROP may exhibit higher densities of trigeminal (touch) fibres on the tongue than NT. This may explain reports that PROP intensity has been associated with perceived intensity of other bitter tastes such as quinine and caffeine[57], and that PROP tasters experience a heightened perception of other taste qualities, including sweetness from sucrose[58] and saccharine[59,60]; and the perception of liquid fat[61], as well as heightened 'oral burn' from spices such as capsaicin[62]. However, the relationship between *TAS2R38*, PROP intensity and FP density is unclear: one study has reported that FP density does not predict PROP intensity in individuals heterozygous at *TAS2R38*[63], while another study concluded that PROP intensity is not correlated to creaminess perception and therefore not related to FP density[64]. It has been suggested that the differences observed in oral sensitivity may be due to differences in central nervous system processes, rather than differences in FP density[65]. Further clarification in this area is needed between FP density, PROP taster status and *TAS2R38*.

## Taste perception and food choice

Taste perception influences food preference, which is one of the strongest mediators of fruit and vegetable consumption in children and adolescents[66,67], and might represent a significant barrier to the consumption of high-fibre-foods and fruit and vegetables in these groups[13,68]. The majority of taste perception studies focus on bitter taste perception and there is little information on the effect of either sweet or umami perception on food preferences. It is known that preference for sweet tastes is partly genetically mediated and in rodents variations in the T1R1 receptor contribute to differential preferences for sweeteners such as saccharin[69,70]. In human subjects, preference for sweet tastes has been linked to a gene or genes located on chromosome 16p 11·2.[71]. Unlike our murine counterparts, this is not because of variations in sweet taste receptor genes, as the *TAS1R2* and *TAS1R3* genes are both found on chromosome 1p 36[71]. Instead this variation might influence sweet preference in some other way; possibly, the authors suggest through differential processing of sweet sensation[71]. Sweet perception may influence food preferences, as individuals with an increased sweet perception tend to have a lower preference for sugar than less sensitive individuals[72]. Sweet perception has been linked to alcoholism[73] and to increased BMI, with a reduced threshold observed in obese children[74]. While it is unclear whether preferences increase the risk of these conditions, or if over-consumption eventually influences preference, a heritable component does appear to be involved.

A variation in the perception of umami taste has also been observed, and this variation has been associated with SNP in umami taste receptor genes, although these SNP are yet to be linked with specific food preferences. Umami taste perception has, however, been linked to obesity, although with mixed results. One report suggested that a heightened umami perception was associated with an increased BMI[74], while another found that obese women have a lower umami sensitivity, and prefer higher concentrations[75]. One possible explanation for these disparate observations is that different mechanisms may be involved in the perception of threshold and suprathreshold (above the level needed for recognition) monosodium glutamate concentrations[75].

Variation in bitter taste perception has been linked to preference for many different foods. Thiourea-containing compounds such as PTC and PROP are bitter-tasting dietary goitrogens, which inhibit the amount of biologically available iodine, and can affect energy balance[76]. Although PROP itself is not found in nature, PROP-related compounds occur in many fruit and vegetables and bitter-tasting foods, including the brassica family of vegetables which contain glucosinolates, and are hydrolysed to isothiocyanates[9]. Isoflavones are bitter-tasting phenolic compounds found in soya and green tea, which might also taste bitter to PROP-sensitive individuals[77]. Therefore, PROP-sensitive individuals may be more sensitive to the bitter tastes of certain 'healthy' foods with known chemopreventive effects[9], which may affect their preferences, and ultimately, health status[16,31,36,72,78]. Furthermore, PROP taster status and differences in oral sensitivities may also affect preference for fat and sugar (NT were reported as more likely to be 'sweet likers' than PROP-sensitive individuals[72]) and to prefer higher-fat foods[61], while ST have shown lower acceptance of whole-grain breads[79]. This may be due to the link with the FP density which can affect oral sensitivity.

The perception of bitter taste has also been associated with a number of adverse health effects, such as a higher risk of alcoholism in PROP NT[80], increased BMI in female NT[50,61,81], a possible increased risk of colon cancer in male ST [82], and in children it has been shown that NT have a higher risk of developing dental caries (tooth decay), presumably as they prefer sugar-containing foods

**Table 3.** Allele frequencies at variant loci *TAS2R38*

| Position in gene | | | | Frequency observed in this study | Comparative frequencies | | | |
|---|---|---|---|---|---|---|---|---|
| Base pair | Amino acid | Allele | Amino acid encoded | Irish Caucasian population | Caucasian population[49] | Americans of European descent[39] | Americans of African descent[39] | British Caucasian[98] |
| 145 | 49 | C | Proline | 0·42 | 0·36 | 0·41 | 0·47 | 0·40 |
| | | G | Alanine | 0·58 | 0·64 | 0·59 | 0·53 | 0·60 |
| 785 | 262 | C | Alanine | 0·47 | 0·38 | 0·47 | 0·66 | 0·45 |
| | | T | Valine | 0·53 | 0·62 | 0·53 | 0·34 | 0·55 |
| 886 | 296 | C | Valine | 0·47 | 0·38 | 0·48 | 0·49 | Did not examine 3rd SNP |
| | | T | Isoleucine | 0·53 | 0·62 | 0·52 | 0·51 | |

SNP, single nucleotide polymorphism.

to PROP MT and ST [83]. However, while it has been conclusively demonstrated that PROP tasters detect more bitterness from glucosinolate-containing vegetables than do NT[84], there is a general lack of agreement between reports on the extent of the link between taster status and food preference or intake. A number of studies have shown no correlation between taster status and food preferences[85–87], while many others have reported links between taster status and preference for and/or consumption of various fruit and vegetables. Lower preference has been reported in PROP tasters (both MT and ST) for citrus fruit[88,89] and this group appears to consume less fruit in general than NT[56]. Lower preference has also been reported for Brussels sprouts, cabbage and spinach[16], asparagus and curly kale[90] and lower overall vegetable consumption[90,91] in PROP-sensitive individuals. One caveat with the majority of these studies is that they have examined differences in preference or intake in small population subgroups, which are not necessarily representative of the true extent of the effect of this trait in the general population. Additionally, various methods have been used to determine PROP taster status, making it difficult to compare them. Another drawback to earlier studies is that the PTC gene has been identified relatively recently[51], meaning that few studies have examined the effects of both *TAS2R38* and PROP taster status on dietary intakes. Finally, while it is clear that genetic predispositions are simply one of the range of factors involved in the determination of food preferences, many studies fail to account for this, and this may have contributed to the mixed set of results yielded from past studies[31,92]. Understanding how taste influences fruit and vegetable preference and consumption may help develop an effective programme to mediate increased intake.

## Current research aims and initial findings

Studies have shown a link between genetic variations and food preferences and intake. While many studies have examined the *TAS2R38* gene or used PROP taster status as a marker of this gene, there is a lack of information examining both food preferences and habitual dietary intake in relation to both PROP taster status and *TAS2R38* genotype. This study aimed at examining the extent of the link between *TAS2R38* genotype and PROP taster phenotype, and to establish the extent of the influence of these

traits separately on nutrient intakes in a group of Irish children and adults. A range of other factors were also considered including gender, age, perceived barriers to healthy eating and general awareness of healthy eating. A total of 525 children (225 male, 300 female) aged 7–12 (mean male age 10·39 (SD 1·84) years; mean female age 10·07 (SD 1·43) years and 165 of their parents (36 males; mean age 44·85 (SD 7·07) years and 129 female; mean age 41·09 (SD 6·06) years were recruited to the study through a number of schools which were contacted in Counties Dublin, Louth and Westmeath in the Republic of Ireland, from an even split of socio-economic areas, as determined by school location.

A proportion (84·2%) of the group (*n* 581) was genotyped for the three common alleles found at *TAS2R38*. This is the first time that this trait has been examined in the Irish population at this scale, and the allele and haplotype frequencies observed were as expected for a mainly Caucasian population, and were comparable to previously reported frequencies (Table 3). When genotypes were examined, the overall percentages of PAV/PAV, PAV/AVI and AVI/AVI in the cohort were similar to the percentages of ST, MT and NT of PROP, determined by the perceived intensity of a PROP-impregnated paper disc, as previously described[93]. However, there was an overlap between categories, such that those PAV/PAV individuals were not necessarily all PROP ST. Multivariate analyses revealed that genotype was a significant predictor of PROP intensity, and the overall contribution of genotype to phenotype was approximately 40% ($R^2$ 0·41, $P<0.001$). This is lower than previous reports (55–85%)[50,51] but may represent a truer estimation of the extent of the influence of this single gene on taste perception in a genetically diverse population, and is consistent with the recent finding that supertasting is dependent on more than simply the *TAS2R38* gene[63].

## Genotype, phenotype, food preferences and nutrient intakes

Participants were shown pictures of a range of different vegetables which they rated on a five-point hedonic scale, as described previously[72]. These were grouped into 'bitter vegetables', from the average preference ratings for broccoli, cauliflower, cabbage and Brussels sprouts, and 'non-bitter vegetables' containing the remaining vegetables,

peas and carrots, based on their glucosinolate contents. Multiple regression analyses were carried out to measure the percentage of variation in preferences owing to various factors. A linear model containing five variables was created: PROP taster status, genotype, sweetness perception, FP density, age and gender. This model was a significant predictor of vegetable preference ($P<0.01$), explaining 15·3 % of the variation in males and 18·9 % in females, with the most significant influences being PROP taster status, age and gender, while genotype was less important.

A 3-d diet history was obtained from both the adults and children through interviews with the researchers using a food atlas as a guide for portion size estimates and probing for any brand information. Dietary data were analysed using WISP © (Tinuviel Software, Llanfechell, Anglesey, UK) for overall macro and micronutrient intakes in the different genotype and phenotype groups. This was performed for boys, girls, men and women separately. While there were no BMI differences in either the men or the children, mean BMI was significantly higher in NT women (28·44, SD 6·96) compared to MT and ST (24·67 (SD 3·94) and 25·33 (SD 4·18)) BMI units, respectively; $P<0.01$. Energy intake was slightly higher in NT women, but not significantly so, and when micronutrients were examined, vitamin $B_6$ intakes were significantly higher in NT ($P<0.05$). Interestingly, there was no significant difference between mean BMI in the three genotype groups in the women, consistent with one previous report that has examined both genotype phenotype and BMI in older females[50] and found that phenotype was more influential.

Upon examination of nutrient intakes by genotype, AVI/AVI females had significantly higher intakes of thiamine, vitamin $B_6$ and folate, and lower intakes of vitamin $B_{12}$ ($P<0.05$). The main dietary sources of folate are green leafy vegetables and fortified cereals, whereas common sources of vitamin $B_{12}$ are animal products such as meat and dairy, as well as fortified breakfast cereals. This nutrient pattern observed in AVI/AVI females is similar to a pattern of intakes observed in the European Prospective Investigation into Cancer and Nutrition study, where a 'health conscious' group had significantly higher thiamine and lower vitamin $B_{12}$ levels compared with others[94]. Additionally, AVI/AVI individuals consumed more fibre. These findings may be extrapolated to show that *TAS2R38* is associated with healthy eating patterns in females. However, there were no significant differences in vitamin C, carotene or biotin intakes, which would be expected if fruit and vegetable intakes differed between genotype groups, and so the differences observed in fibre may be derived from increased whole-grain and lower animal product consumption. Whether certain types of fruit and vegetables intake differ between genotype groups is not clear from these analyses.

## Conclusion

Food choices are affected by a wealth of factors, one of which is taste. Taste perception for various taste qualities may vary genetically and lead to differential preferences for certain types of foods. PROP taster status is linked to *TAS2R38* genotype, and both of them can separately influence food preferences. However, in this study, phenotype (PROP taster status) was a greater source of variation in food preferences than *TAS2R38* genotype, and may also predict BMI, which was higher in NT women. Over 20 % of the variation in food preferences could be contributed to taster status. Notably, this influence was found to vary with gender for some vegetables, which was not expected. Clear differences were observed between both genotype and phenotype groups in certain vegetable preferences, although overall, differences in macro and micronutrients were relatively minor in children and in males. Further, AVI/AVI genotype was associated with increased markers of a healthful diet in adult females, yet conversely, overall energy intakes were also increased in this group. This indicates that while female AVI/AVI individuals may be eating more than their PAV/AVI or PAV/PAV counterparts, their choices may be more healthy. Therefore, in conclusion, while genetic variation in taste perception does indeed appear to have a small role in healthy eating, other factors such as age and gender are also important contributors to both food preferences and dietary intakes.

## References

1. Barron C, Comiskey C & Saris J (2009) Prevalence rates and comparisons of obesity levels in Ireland. *J Nurs* **18**, 799–803.
2. Morgan K, McGee H, Watson D et al. (2008) SLAN 2007: Survey of Lifestyle, Attitudes and Nutrition in Ireland: Main Report. Dublin: Department of Health and Children.
3. Berghöfer A, Pischon T, Reinhold T et al. (2008) Obesity prevalence from a European perspective: a systematic review. *BMC Public Health* **8**, 200.
4. Wang Y, Beydoun MA, Liang L et al. (2008) Will all Americans become overweight or obese? Estimating the progression and cost of the US obesity epidemic. *Obesity* **16**, 2323–2330.
5. Shelton RC & Miller AH (2010) Eating ourselves to death and despair: the contribution of adiposity and inflammation to depression. *Prog Neurobiol* **4**, 275–299.
6. Livingstone M, McCaffrey T & Rennie K (2006) Childhood obesity prevention studies: lessons learned and to be learned. *Public Health Nutr* **9**, 1121–1129.
7. Drewnowski A & Specter S (2004) Poverty and obesity: the role of energy density and energy costs. *Am J Clin Nutr* **79**, 6–16.
8. Van Duyn MAS & Pivonka E (2000) Overview of the health benefits of fruit and vegetable consumption for the

dietetics professional: selected literature. *J Am Diet Assoc* **100**, 1511–1521.

9. Vig A, Rampala G, Thinda TH *et al.* (2009) Bio-protective effects of glucosinolates – a review *LWT. Food Sci Technol* **42**, 1561–1572.

10. WHO (1990) *Diet, Nutrition and the Prevention of Chronic Diseases*. Geneva: WHO.

11. FSAI (2007) *A Review of the Fruit and Vegetable Food Chain*. Cork: Safefood.

12. Glanz K, Basil M, Maibach E *et al.* (1998) Why Americans eat what they do: taste, nutrition, cost, convenience, and weight control concerns as influences on food consumption. *J Am Diet Assoc* **98**, 1118–1126.

13. Neumark-Sztainer D, Story M, Perry C *et al.* (1999) Factors influencing food choices of adolescents findings from focus-group discussions with adolescents. *J Am Diet Assoc* **99**, 929–937.

14. Dibsdall LA, Lambert N, Bobbin RF *et al.* (2003) Low-income consumers' attitudes and behaviour towards access, availability and motivation to eat fruit and vegetables. *Public Health Nutr* **6**, 159–168.

15. Wardle J, Carnell S & Cooke L (2005) Parental control over feeding, and children's fruit and vegetable intake: How are they related? *J Am Diet Assoc* **105**, 227–232.

16. Drewnowski A, Henderson SA, Levine A *et al.* (1999) Taste and food preferences as predictors of dietary practices in young women. *Public Health Nutr* **2**, 513–519.

17. Connors M, Bisogni CA, Sobal J *et al.* (2001) Managing values in personal food systems. *Appetite* **36**, 189–200.

18. Scheibehenne B, Miesler L & Todd PM (2007) Fast and frugal food choices: uncovering individual decision heuristics. *Appetite* **49**, 578–589.

19. Drewnowski A & Rock CL (1995) The influence of genetic taste markers on food acceptance. *Am J Clin Nutr* **62**, 506–511.

20. Kearney M, Kearney JM, Dunne A *et al.* (2000) Socio-demographic determinants of perceived influences on food choice in a nationally representative sample of Irish adults. *Public Health Nutr* **3**, 219–226.

21. Mok L (2010) The effect of variety and dietary restraint on food intake in lean young women: a preliminary study. *J Gen Psychol* **137**, 63–83.

22. Tepper B (2009) Genetic variation in taste sensitivity to 6-n-propylthiouracil and Its relationship to taste perception and food selection. *Ann NY Acad Sci* **1170**, 126–139.

23. Sanematsu K, Horio N, Murata Y *et al.* (2009) Modulation and transmission of sweet taste information for energy homeostasis. *Ann NY Acad Sci* **1170**, 102–106.

24. Hayes JE, Sullivan BS & Duffy VB (2010) Explaining variability in sodium intake through oral sensory phenotype, salt sensation and liking. *Physiol Behav* **100**(4), 369–380.

25. Mainland J & Matsunami H (2009) Taste perception: how sweet it is (to be transcribed by you). *Curr Biol* **19**, R655–R656.

26. Kim UK & Drayna D (2005) Genetics of Individual differences in bitter taste perception: lessons from the PTC gene. *Clin Genet* **67**, 275–280.

27. Sugino Y, Umemoto A & Mizutani S (2002) Insensitivity to the bitter taste of chloramphenicol: an autosomal recessive trait. *Genes Genet Syst* **77**, 59–62.

28. Wise PM, Hansen JL, Reed DR *et al.* (2007) Twin study of the heritability of recognition thresholds for sour and salty taste. *Chem Senses* **32**, 749–754.

29. Drayna D (2005) Human taste genetics. *Annu Rev Genom* **6**, 217–235.

30. Shigemura N, Shirosaki S, Sanematsu K *et al.* (2009) Genetic and molecular basis of individual differences in human umami taste perception. *PLoS ONE* **4**, e6717.

31. Tepper BJ (2008) Nutritional implications of genetic taste variation: the role of PROP sensitivity and other taste phenotypes. *Annu Rev Nutr* **28**, 367–388.

32. Meyers B & Brewer MS (2008) Sweet taste in man: a review. *J Food Sci* **73**, R81–R90.

33. Kim UK, Breslin PA, Reed D *et al.* (2004) Genetics of human taste perception. *J Dent Res* **83**, 448–453.

34. Kaji I, Karaki S-I, Fukami Y *et al.* (2009) Secretory effects of a luminal bitter tastant and expressions of bitter taste receptors, T2Rs, in the human and rat large intestine. *Am J Physiol Gastrointest Liver Physiol* **296**, G971–G981.

35. Glendinning JI (1994) Is the bitter rejection response always adaptive. *Physiol Behav* **56**, 1217–1227.

36. Drewnowski A & Gomez-Carneros C (2000) Bitter taste, phytonutrients, and the consumer: a review. *Am J Clin Nutr* **72**, 1424–1435.

37. des Gachons C, Beauchamp G & Breslin P (2009) The genetics of bitterness and pungency detection and its impact on phytonutrient evaluation. *Ann NY Acad Sci* **1170**, 140–144.

38. Meyerhof W (2005) Elucidation of mammalian bitter taste. *Rev Physiol Biochem Pharmacol* **154**, 37–72.

39. Mangold JE, Payne TJ, Ma JZ *et al.* (2008) Bitter taste receptor gene polymorphisms are an important factor in the development of nicotine dependence in African Americans. *J Med Genet* **45**, 578–582.

40. Kim UK, Wooding S, Riaz N *et al.* (2006) Variation in the human TAS1R taste receptor genes. *Chem Senses* **31**, 599–611.

41. Fushan AA, Simons CT, Slack JP *et al.* (2009) Allelic polymorphism within the TAS1R3 promoter is associated with human taste sensitivity to sucrose. *Curr Biol* **19**, 1288–1293.

42. Li X, Staszewski L, Xu H *et al.* (2002) Human receptors for sweet and umami taste. *Proc Natl Acad Sci USA* **99**, 4692–4696.

43. Chen QY, Alarcon S, Tharp A *et al.* (2009) Perceptual variation in umami taste and polymorphisms in TAS1R taste receptor genes. *Am J Clin Nutr* **90**, 770S–779S.

44. Bufe B, Breslin PAS, Kuhn C *et al.* (2005) The molecular basis of individual differences in phenylthiocarbamide and propylthiouracil bitterness perception. *Curr Biol* **15**, 322–327.

45. Tepper BJ (1998) 6-n-propylthiouracil: a genetic marker for taste, with implications for food preference and dietary habits. *Am J Hum Genet* **63**, 1271–1276.

46. Reed DR, Tanaka T & McDaniel AH (2006) Diverse tastes: genetics of sweet and bitter perception. *Physiol Behav* **88**, 215–226.

47. Wheatcroft PE & Thornburn CC (1972) Toxicity of the taste testing compound phenylthiocarbamide. *Nat New Biol* **235**, 93–94.

48. Bartoshuk LM, Fast K, Karrer TA *et al.* (1992) PROP super tasters and the perception of sweetness and bitterness. *Chem Senses* **17**, 594.

49. Kim U, Wooding S, Ricci D *et al.* (2005) Worldwide haplotype diversity and coding sequence variation at human bitter taste receptor loci. *Hum Mutat* **26**, 199–204.

50. Tepper BJ, Koelliker Y, Zhao L *et al.* (2008) Variation in the bitter-taste receptor gene TAS2R38, and adiposity in a genetically isolated population in southern Italy. *Obesity* **16**, 2289–2295.

51. Kim UK, Jorgenson E, Coon H *et al.* (2003) Positional cloning of the human quantitative trait locus underlying

taste sensitivity to phenylthiocarbamide. *Science* **299**, 1221–1225.

52. Bartoshuk LM, Duffy VB & Miller IJ (1994) PTC/PROP tasting: anatomy, psychophysics, and sex effects. *Physiol Behav* **56**, 1165–1171.

53. Delwiche JF, Buletic Z & Breslin PAS (2001) Relationship of papillae number to bitter intensity of quinine and PROP within and between individuals. *Physiol Behav* **74**, 329–337.

54. Essick GK, Chopra A, Guest S *et al.* (2003) Lingual tactile acuity, taste perception, and the density and diameter of fungiform papillae in female subjects. *Physiol Behav* **80**, 289–302.

55. Tepper BJ & Nurse RJ (1997) Fat perception is related to PROP taster status. *Physiol Behav* **61**, 949–954.

56. Yackinous CA & Guinard JX (2002) Relation between PROP (6-n-propylthiouracil) taster status, taste anatomy and dietary intake measures for young men and women. *Appetite* **38**, 201–209.

57. Hansen JL, Reed DR, Wright MJ *et al.* (2006) Heritability and genetic covariation of sensitivity to PROP, SOA, quinine HCl, and caffeine. *Chem Senses* **5**, 403–413.

58. Lucchina LA, Curtis OF, Putnam P *et al.* (1998) Psychophysical measurement of 6-n-propylthiouracil (PROP) taste perception. *Ann NY Acad Sci* **855**, 816–819.

59. Bartoshuk LM (1979) Bitter taste of saccharin related to the genetic ability to taste the bitter substance 6-n-propylthiouracil. *Science* **205**, 934–935.

60. Pronin AN, Xu H, Tang H *et al.* (2007) Specific alleles of bitter receptor genes influence human sensitivity to the bitterness of aloin and saccharin. *Curr Biol* **17**, 1403–1408.

61. Tepper BJ & Nurse RJ (1998) PROP taster status is related to the perception and preference for fat. *Ann NY Acad Sci* **855**, 802–804.

62. Karrer T & Bartoshuk L (1991) Capsaicin desensitization and recovery on the human tongue. *Physiol Behav* **49**(4), 757–764.

63. Hayes JE, Bartoshuk LM, Kidd JR *et al.* (2008) Supertasting and PROP bitterness depends on more than the TAS2R38 gene. *Chem Senses* **33**, 255–265.

64. Lim J, Urban L & Green BG (2008) Measures of individual differences in taste and creaminess perception. *Chem Senses* **33**, 493–501.

65. Green BG & George P (2004) 'Thermal taste' predicts higher responsiveness to chemical taste and flavor. *Chem Senses* **29**, 617–628.

66. Rasmussen M, Krølner R, Klepp K-I *et al.* (2006) Determinants of fruit and vegetable consumption among children and adolescents: a review of the literature. Part I: quantitative studies. *Int J Behav Nutr Phys Act* **3**, 22.

67. Bere E, Brug J & Klepp K (2008) Why do boys eat less fruit and vegetables than girls? *Public Health Nutr* **11**, 321–325.

68. Berg C, Jonsson I, Conner M *et al.* (2003) Perceptions and reasons for choice of fat- and fibre-containing foods by Swedish schoolchildren. *Appetite* **40**, 61–67.

69. Sclafani A (2006) Oral, Post-Oral and Genetic Interactions in Sweet Appetite. *Physiol Behav* **89**, 525–530.

70. Meyers B & Brewer MS (2008) Sweet taste in man: a review. *J Food Sci* **73**, R81–R90.

71. Keskitalo K, Knaapila A, Kallela M *et al.* (2007) Sweet taste preferences are partly genetically determined: identification of a trait locus on chromosome 16. *Am J Clin Nutr* **86**, 55–63.

72. Looy H & Weingarten HP (1992) Facial expressions and genetic sensitivity to 6-n-propylthiouracil predict hedonic response to sweet. *Physiol Behav* **52**, 75–82.

73. Mennella JA, Pepino MY, Lehmann-Castor SM *et al.* (2010) Sweet preferences and analgesia during childhood: effects of family history of alcoholism and depression. *Addiction* **105**, 666–675.

74. Donaldson LF, Bennett L, Baic S *et al.* (2009) Taste and weight: is there a link? *Am J Clin Nutr* **90**, 800S–803S.

75. Pepino MY, Finkbeiner S, Beauchamp GK *et al.* Obese women have lower monosodium glutamate taste sensitivity and prefer higher concentrations than do normal-weight women. *Obesity* **18**, 959–965.

76. Reed DR, Li S, Li X *et al.* (2004) Polymorphisms in the taste receptor gene (Tas1r3) region are associated with saccharin preference in 30 mouse strains. *J Neurosci* **24**, 938–946.

77. Akella GD, Henderson SA & Drewnowski A (1997) Sensory acceptance of Japanese green tea and soy products is linked to genetic sensitivity to 6-n-propylthiouracil. *Nutr Cancer* **29**, 146–151.

78. Keller KL, Steinmann L, Nurse RJ *et al.* (2002) Genetic taste sensitivity to 6-n-propylthiouracil influences food preference and reported intake in preschool children. *Appetite* **38**, 3–12.

79. Bakke A & Vickers Z (2007) Consumer liking of refined and whole wheat breads. *J Food Sci* **72**, S473–S480.

80. Duffy VB, Davidson AC, Kidd JR *et al.* (2004) Bitter receptor gene (TAS2R38), 6-n-propylthiouracil (PROP) bitterness and alcohol intake. *Alcohol Clin Exp Res* **28**, 1629–1637.

81. Goldstein GL, Daun H & Tepper BJ (2005) Adiposity in middle-aged women is associated with genetic taste blindness to 6-n-propylthiouracil. *Obes Res* **13**, 1017–1023.

82. Basson MD, Bartoshuk LM, Dichello SZ *et al.* (2005) Association between 6-n-propylthiouracil (PROP) bitterness and colonic neoplasms. *Dig Dis Sci* **50**, 483–489.

83. Rupesh S & Nayak U (2006) Genetic sensitivity to the bitter taste of 6-n propylthiouracil: a new risk determinant for dental caries in children. *J Indian Soc Pedod Prev Dent* **24**, 63–68.

84. Sandell MA & Breslin PA (2006) Variability in a taste-receptor gene determines whether we taste toxins in food. *Curr Biol* **16**, R792–R794.

85. Anliker JA, Bartoshuk L, Ferris AM *et al.* (1991) Children's food preferences and genetic sensitivity to the bitter taste of 6-n-propylthiouracil (PROP). *Am J Clin Nutr* **54**, 316–320.

86. Niewind A, Krondi M & Shrott M (1988) Genetic influences on the selection of brassica vegetables by elderly individuals. *Nutr Res* **8**, 13–20.

87. Mattes R & Labov J (1989) Bitter taste responses to phenylthiocarbamide are not related to dietary goitrogen intake in human beings. *J Am Diet Assoc* **89**, 692–694.

88. Drewnowski A, Henderson SA, Shore AB *et al.* (1998) Sensory responses to 6-n-propylthiouracil (PROP) or sucrose solutions and food preferences in young women. *Ann NY Acad Sci* **855**, 797–801.

89. Tepper BJ, Keller KL & Ullrich NV (2003) Genetic variation in taste and preferences for bitter and pungent foods: implications for chronic disease risk. In *Challenges in Taste Chemistry and Biology*, pp. 60–74 [TL Hofmann, editor]. Symposium Series 867. Washington, DC: American Chemical Society.

90. Dinehart ME, Hayes JE, Bartoshuk LM *et al.* (2006) Bitter taste markers explain variability in vegetable sweetness, bitterness, and intake. *Physiol Behav* **87**, 304–313.

91. Jerzsa-Latta M, Krondl M & Coleman P (1990) Use and perceived attributes of cruciferous vegetables in terms

of genetically-mediated taste sensitivity. *Appetite* **15**, 127–134.

92. Mattes R (2004) 6-n-propylthiouracil taster status: dietary modifier, marker or misleader? In *Genetic Variation in Taste Sensitivity*, pp. 229–250 [J Prescott & B. Tepper, editors]. New York: Marcel Dekker Inc.

93. Zhao L, Kirkmeyer SV & Tepper BJ (2003) A paper screening test to assess genetic taste sensitivity to 6-n-propylthiouracil. *Physiol Behav* **78**, 625–633.

94. Olsen A, Halkjaer J, van Gils CH *et al.* (2009) Dietary intake of the water-soluble vitamins $B_1$, $B_2$, $B_6$, $B_{12}$ and C in 10 countries in the European prospective investigation into cancer and nutrition. *Eur J Clin Nutr* **63**, S122–S149.

95. Raliou M, Wiencis A, Pillias A-M *et al.* (2009) Nonsynonymous single nucleotide polymorphisms in human tas1r1, tas1r3, and mGluR1 and individual taste sensitivity to glutamate. *Am J Clin Nutr* **90**, 789S–799S.

96. Soranzo N, Bufe B, Sabeti PC *et al.* (2005) Positive selection on a high-sensitivity allele of the human bitter-taste receptor TAS2R16. *Curr Biol* **15**, 1257–1265.

97. Floriano WB, Hall S, Vaidehi N *et al.* (2006) Modeling the human PTC bitter-taste receptor interactions with bitter tastants. *J Mol Model* **12**, 931–941.

98. Timpson NJ, Heron J, Day IN *et al.* (2007) Refining associations between TAS2R38 diplotypes and the 6-n-propylthiouracil (PROP) taste test: findings from the Avon longitudinal study of parents and children. *BMC Genet* **8**, 51.

99. Behrens M & Meyerhof W (2006) Bitter taste receptors and human bitter taste perception. *Cell Mol Life Sci* **63**, 1501–1509.

100. Beauchamp GK & Stein LJ (2007) Salt taste. In *The Senses: A Comprehensive Reference*, pp. 401–408 [AI Basbaum, A Kaneko, GM Shepherd *et al.*, editors]. Amsterdam: Elsevier.