The Genetics of Taste

By Colton Valentine

S
he hates broccoli, he can't stand the sight of carrots, and your dad's the only one who eats radishes. It takes little scientific inquiry to identify the variability of food preferences, but what exactly makes someone have a sweet tooth? Can taste proclivities be explained solely through postnatal conditioning or is there a genetic component involved as well? Unfortunately, determining exactly why you love tartar sauce turns out to be quite a tricky process. Yet the implications for understanding these mechanisms are profound and far-reaching. After all, people eat what tastes good, and those who find that alluring flavor in spinach rather than ice cream will likely have healthier diets.

Modern taste science began, as so much science tends to do, by accident. In 1991, Arthur Fox, a DuPont chemist, accidentally released a cloud of phenylthiocarbamide while Fox tasted nothing, his colleague complained about the bitter taste emanating throughout the room [1]. Fox immediately recognized the importance of this differential reaction, and set to work investigating its source. Since then, no taste response has been studied more than that to PTC, but the full implications of Fox's observation are not entirely clear. What we do know is that PTC sensitivity is largely controlled by the gene TAS2R38 and that this single bitterness response affects a wide range of taste preferences [1]. In fact, TAS2R38 has been implicated in everything from vegetable consumption to alcohol dependency [1-5]. Throw the complex idea of a “supertaster,” or one who tastes certain flavors with particularly intensity, into the mix, and you can begin to see the exciting culinary and clinical applications TAS2R38 may have. But let's not get ahead of ourselves.

Supertasters
Chances are you have seen at least an article or two that involves the concept of a supertaster. Cake is more delicious for those with proclivities to sweetness, saltiness, or bitterness interacts with proclivities to sweetness, saltiness, and more. Yet as more research was performed, it became clear that there were actually three distinct phenotypes for the TAS2R gene. Tasters, it turned out, could be divided into two subgroups, where one had an even stronger reaction to PTC [1]. How could this be explained on the genotypic level? The key was in the relative differences between PAV heterozygotes and homozygotes. Anyone possessing a PAV allele was a general taster, but those with two copies, or homozygotes, had a higher sensitivity to PTC than those with a copy of each allele or heterozygotes [1]. Much of the research done in recent years has focused on these homozygotes, but occasionally all “tasters” are lumped together. These are distinctions to keep in mind when analyzing data on TAS2R38.

Introduction to TAS2R Genes
As far as might expect, taste response is not mediated by a single gene, but rather by a large network of interacting mechanisms. One of the best-characterized subsets of taste genes, the TAS2R family controls the human response to bitterness. Within this group of 25 genes, perhaps the most intriguing is TAS2R38, for it controls the response that Fox discovered to PTC (and its safer equivalent PROP). At first it was believed that this gene followed a straightforward Mendelian recessive-dominant inheritance structure. Three polymorphisms form two common versions of the gene: PAV and AVI. PAVs could taste PTC, while AVIs could not [3]. What’s particularly intriguing about this gene is that these two alleles indirectly affect other taste functions, for bitterness interacts with proclivities to sweetness, saltiness, and more.

The implications for understanding the mechanisms of taste are profound and far reaching.

PAV homozygotes who “supertaste” PTC and as a result many bitter foods; most studies investigating TAS2R38 use this terminol- ogy. In popular culture, “supertaster” is often used to describe a more general enhanced-taste phenomenon (cake being more delicious). A link exists between the two, for PAV homozygotes are more likely to have this general tast- ing phenotype, but many other factors influence it as well [6].

Who exactly are these generic “supertasters” then? The primary piece of evidence toward their existence is that the anterior or front regions of supertaster tongues contain more taste papillae, perhaps leading to higher flavor sensitiv- ity [6]. However, PAV allele holders also show an increase in these papillae, though not to the same extent, making the identification between these two groups of supertasters a tricky business [1]. For the purposes of the rest of this article, the term “PAV supertasters” will be used to denote TAS2R38 PAV homozygotes, that is the area where specific research has been conducted. It pays to be wary of any information discussing the general phenomenon of a supertaster, as far more work needs to be done to specifi- cally understand what the concept even means. This is a case where a scientific claim has been largely blown out of propor- tion in pop culture. But enough on semantics; what’s far more interesting is how PAV tasters are attracted to differ- ent foods than AVI nontasters.

Food Proclivities
Since TAS2R38 controls sensitivity to bitterness, much of the food proclivity research has been done on food groups...
related to this sense, especially with respect to vegetables. Several studies have shown a correlation between PROP sensitivity and perceived bitterness in vegetables, yet the exact methods and conclusions vary [1,2]. One group demonstrated several years ago that PROP tasters and supertasters found a variety of vegetables more bitter and less sweet in a lab setting. Even more importantly, when questioned about their dietary practices, those with higher PROP sensitivities reported eating fewer vegetables [2]. It seemed that the sensitivity to bitterness was turning people off from the leafy greens, making them less likely to enjoy the recommended three servings a day.

Another study took this investigation a step farther by tying vegetable intake directly to the TAS2R38 genotype. They found that PROP allele carriers perceived more bitterness in vegetables and consumed relatively fewer servings. Intriguingly, these trends were associated with all vegetables, not simply those considered bitter. This suggests that both PROP homozygotes and heterozygotes were treated as one group. Together these studies raised concerns about PROP tasters being less inclined than nontasters to eat vegetables, which raises a potential health concern.

Yet vegetable intake is not the only shifting food reaction PROP allele carriers have. More recently, data have circulated regarding higher vegetable intake by both nontaster allele carriers who have modulated their intake. This could be another way to envision for or against? One study looked at TAS2R61, a gene that controls sensitivities to several different bitter compounds. Two alleles exist for this gene; N172 or higher sensitivity and K172 or lower sensitivity [10]. According to this study, higher sensitivity alleles may cause greater food intake, yet natural selection might favor those with nontaster alleles because they have access to more food [10]. Perhaps the reason both alleles are still quite common is that a balance between these two selective pressures exists.

**The Alcohol Effect**

An oft-surprising component of TAS2R research is likely to look at the association between PROP or TAS2R38 and alcohol consumption, so natural selection might favor those with nontaster alleles because they have access to more food [10]. Perhaps the reason both alleles are still quite common is that a balance between these two selective pressures exists.

A step farther by tying vegetable intake directly to the TAS2R38 genotype. They found that PROP allele carriers perceived more bitterness in vegetables and consumed relatively fewer servings. Intriguingly, these trends were associated with all vegetables, not simply those considered bitter. This suggests that both PROP homozygotes and heterozygotes were treated as one group. Together these studies raised concerns about PROP tasters being less inclined than nontasters to eat vegetables, which raises a potential health concern.

Yet vegetable intake is not the only shifting food reaction PROP allele carriers have. More recently, data have circulated regarding higher vegetable intake by both nontaster allele carriers who have modulated their intake. This could be another way to envision for or against? One study looked at TAS2R61, a gene that controls sensitivities to several different bitter compounds. Two alleles exist for this gene; N172 or higher sensitivity and K172 or lower sensitivity [10]. According to this study, higher sensitivity alleles may cause greater food intake, yet natural selection might favor those with nontaster alleles because they have access to more food [10]. Perhaps the reason both alleles are still quite common is that a balance between these two selective pressures exists.

**The Alcohol Effect**

An oft-surprising component of TAS2R research is likely to look at the association between PROP or TAS2R38 and alcohol consumption, so natural selection might favor those with nontaster alleles because they have access to more food [10]. Perhaps the reason both alleles are still quite common is that a balance between these two selective pressures exists.

A step farther by tying vegetable intake directly to the TAS2R38 genotype. They found that PROP allele carriers perceived more bitterness in vegetables and consumed relatively fewer servings. Intriguingly, these trends were associated with all vegetables, not simply those considered bitter. This suggests that both PROP homozygotes and heterozygotes were treated as one group. Together these studies raised concerns about PROP tasters being less inclined than nontasters to eat vegetables, which raises a potential health concern.

Yet vegetable intake is not the only shifting food reaction PROP allele carriers have. More recently, data have circulated regarding higher vegetable intake by both nontaster allele carriers who have modulated their intake. This could be another way to envision for or against? One study looked at TAS2R61, a gene that controls sensitivities to several different bitter compounds. Two alleles exist for this gene; N172 or higher sensitivity and K172 or lower sensitivity [10]. According to this study, higher sensitivity alleles may cause greater food intake, yet natural selection might favor those with nontaster alleles because they have access to more food [10]. Perhaps the reason both alleles are still quite common is that a balance between these two selective pressures exists.

A step farther by tying vegetable intake directly to the TAS2R38 genotype. They found that PROP allele carriers perceived more bitterness in vegetables and consumed relatively fewer servings. Intriguingly, these trends were associated with all vegetables, not simply those considered bitter. This suggests that both PROP homozygotes and heterozygotes were treated as one group. Together these studies raised concerns about PROP tasters being less inclined than nontasters to eat vegetables, which raises a potential health concern.

Yet vegetable intake is not the only shifting food reaction PROP allele carriers have. More recently, data have circulated regarding higher vegetable intake by both nontaster allele carriers who have modulated their intake. This could be another way to envision for or against? One study looked at TAS2R61, a gene that controls sensitivities to several different bitter compounds. Two alleles exist for this gene; N172 or higher sensitivity and K172 or lower sensitivity [10]. According to this study, higher sensitivity alleles may cause greater food intake, yet natural selection might favor those with nontaster alleles because they have access to more food [10]. Perhaps the reason both alleles are still quite common is that a balance between these two selective pressures exists.

A step farther by tying vegetable intake directly to the TAS2R38 genotype. They found that PROP allele carriers perceived more bitterness in vegetables and consumed relatively fewer servings. Intriguingly, these trends were associated with all vegetables, not simply those considered bitter. This suggests that both PROP homozygotes and heterozygotes were treated as one group. Together these studies raised concerns about PROP tasters being less inclined than nontasters to eat vegetables, which raises a potential health concern.

Yet vegetable intake is not the only shifting food reaction PROP allele carriers have. More recently, data have circulated regarding higher vegetable intake by both nontaster allele carriers who have modulated their intake. This could be another way to envision for or against? One study looked at TAS2R61, a gene that controls sensitivities to several different bitter compounds. Two alleles exist for this gene; N172 or higher sensitivity and K172 or lower sensitivity [10]. According to this study, higher sensitivity alleles may cause greater food intake, yet natural selection might favor those with nontaster alleles because they have access to more food [10]. Perhaps the reason both alleles are still quite common is that a balance between these two selective pressures exists.

A step farther by tying vegetable intake directly to the TAS2R38 genotype. They found that PROP allele carriers perceived more bitterness in vegetables and consumed relatively fewer servings. Intriguingly, these trends were associated with all vegetables, not simply those considered bitter. This suggests that both PROP homozygotes and heterozygotes were treated as one group. Together these studies raised concerns about PROP tasters being less inclined than nontasters to eat vegetables, which raises a potential health concern.

Yet vegetable intake is not the only shifting food reaction PROP allele carriers have. More recently, data have circulated regarding higher vegetable intake by both nontaster allele carriers who have modulated their intake. This could be another way to envision for or against? One study looked at TAS2R61, a gene that controls sensitivities to several different bitter compounds. Two alleles exist for this gene; N172 or higher sensitivity and K172 or lower sensitivity [10]. According to this study, higher sensitivity alleles may cause greater food intake, yet natural selection might favor those with nontaster alleles because they have access to more food [10]. Perhaps the reason both alleles are still quite common is that a balance between these two selective pressures exists.

A step farther by tying vegetable intake directly to the TAS2R38 genotype. They found that PROP allele carriers perceived more bitterness in vegetables and consumed relatively fewer servings. Intriguingly, these trends were associated with all vegetables, not simply those considered bitter. This suggests that both PROP homozygotes and heterozygotes were treated as one group. Together these studies raised concerns about PROP tasters being less inclined than nontasters to eat vegetables, which raises a potential health concern.

Yet vegetable intake is not the only shifting food reaction PROP allele carriers have. More recently, data have circulated regarding higher vegetable intake by both nontaster allele carriers who have modulated their intake. This could be another way to envision for or against? One study looked at TAS2R61, a gene that controls sensitivities to several different bitter compounds. Two alleles exist for this gene; N172 or higher sensitivity and K172 or lower sensitivity [10]. According to this study, higher sensitivity alleles may cause greater food intake, yet natural selection might favor those with nontaster alleles because they have access to more food [10]. Perhaps the reason both alleles are still quite common is that a balance between these two selective pressures exists.