Astringency: Mechanisms and Perception

Martha R. Bajec and Gary J. Pickering

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Astringency plays an important role in the sensory experience of many foods and beverages, ranging from wine to nuts. Given the recent trend toward fortifying consumables with astringent compounds and the evidence regarding the health benefits of some astringents, the mechanisms and perceptual characteristics of astringency warrant further discussion and investigation. This paper reviews the current state of the literature, including consideration of new methods for describing and measuring astringency, and provides an overview of research concerned with elucidating the physical, physiological, and psychological factors that underlie and mediate perception of this sensation.

Keywords  taste, physiology, behavior, saliva, sensory, oral, astringency

INTRODUCTION

Astringency plays an important role in the sensory experience elicited by a diverse range of foods and beverages (Table 1), including wine (Gawel, 1998), tea (Scharbert et al., 2004), soymilk (Al Mahfuz et al., 2004), coffee (Morales, 1989), fruits (Joslyn and Goldstein, 1964; Ozawa et al., 1987), nuts (Karchesy and Hemingway, 1986) and legumes (Martin-Tanguy et al., 1977; Barahona et al.) Numerous and potent health-promoting benefits of some astringent compounds (polyphenols) found in a range of fruits and vegetables and the processed consumables derived from them, such as tea (Horiba et al., 1991; Imai and Nakachi, 1995; Hollman et al., 1997), red wine (Renaud et al., 1992; Fitzpatrick et al., 1993; Clifford et al., 1996; Bargallo et al., 2006), and most recently, phenol-enriched white wines (Fuhrman et al., 2001; Landraut et al., 2003; Auger et al., 2005), have also been demonstrated. The importance of astringent compounds in the primate diet has been confirmed by the natural feeding behavior of rhesus monkeys (Macaca mulatta), who choose their food based on phenolic content as opposed to total protein or non-structural carbohydrate content (Marks et al., 1988).

While G-protein coupled receptors (GPCRs) and ion channels are generally—although not universally—accepted (Herness and Gilbertson, 1999; Bradbury, 2004) as the molecular basis for sweet, bitter and umami tastes (Naim et al., 1994; Hoon et al., 1999; Adler et al., 2000; Chandrashekar et al., 2000; Matsunami et al., 2004; Li et al., 2002; Nelson et al., 2001, 2002; Ozeck et al., 2004), and salty and sour tastes (Heck et al., 1984; Tennissen and McCutcheon, 1996; Kinnamon et al., 1988; Waldmann and Champigny, 1997; Ugawa et al., 2003), the molecular and physiological mechanisms underlying astringency have not been definitively elucidated (Bakker, 1998). At the perceptual level, it is far from clear whether astringency is best regarded as a single perceptual phenomenon or as a composite term encompassing a number of subtle tactile sensations (astringent “sub-qualities”). Widely differing opinions exist on the current state of knowledge concerning astringency, with some groups claiming that the physical nature of astringency is well understood (Lyman and Green, 1990) while others maintain the opposite (Bakker, 1998; Iiyama et al., 1995; Courregelongue et al., 1999). For instance, while some research strongly suggests that the sensation of astringency is a tactile phenomenon (Breslin et al., 1993), initiated by the binding and precipitation of proteins by polyphenols (Kallithraka et al., 1998), evidence has also been presented supporting the speculation of Aristotle and Galen that astringency is a gustatory sensation (Bartoshuk, 1978). This paper reviews the literature on this debate and others concerning the underlying mechanism(s) responsible for astringency, and provides an overview of the research concerned with elucidating the physical, physiological and psychological factors that mediate its perception.

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Table 1 Common tannin-containing plants used as foodstuffs, forage crops, livestock feeds, beverages, and herbal preparations. Adapted from Haslam and Lilley (1988).

<table>
<thead>
<tr>
<th>Proanthocyanidins (Condensed tannins)</th>
<th>Galloyl and hexahydroxydiphenoyl esters (Hydrolysable tannins)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common name</td>
<td>Species name</td>
</tr>
<tr>
<td>Apple</td>
<td>Malus sp.</td>
</tr>
<tr>
<td>Persimmon</td>
<td>Diospyros kaki</td>
</tr>
<tr>
<td>Grape</td>
<td>Vinus vinifera</td>
</tr>
<tr>
<td>Strawberry</td>
<td>Fragaria sp.</td>
</tr>
<tr>
<td>Blackberry, Dewberry, Raspberry</td>
<td>Rubus sp.</td>
</tr>
<tr>
<td>Plum, Cherry</td>
<td>Prunus sp.</td>
</tr>
<tr>
<td>Bilberry, Cranberry</td>
<td>Vaccinium sp.</td>
</tr>
<tr>
<td>Gooseberry, Black and red currant</td>
<td>Ribes sp.</td>
</tr>
<tr>
<td>Quince</td>
<td>Cydonia sp., Chaenomeles chinensis</td>
</tr>
<tr>
<td>Cocoa bean</td>
<td>Theobroma cacao</td>
</tr>
<tr>
<td>Kola nut</td>
<td>Cola acuminata</td>
</tr>
<tr>
<td>Pear</td>
<td>Pyrus sp.</td>
</tr>
<tr>
<td>Hawthorn</td>
<td>Crataegus sp.</td>
</tr>
<tr>
<td>Rose hip</td>
<td>Rosa sp.</td>
</tr>
<tr>
<td>Chinese gooseberry</td>
<td>Actinidia chinensis</td>
</tr>
<tr>
<td>Yam</td>
<td>Dioscorea alata</td>
</tr>
<tr>
<td>Sorghum</td>
<td>Sorghum sp.</td>
</tr>
<tr>
<td>Barley</td>
<td>Hordeum vulgare</td>
</tr>
<tr>
<td>Sainfoin</td>
<td>Onobrychis viccifolia</td>
</tr>
<tr>
<td>Herbaceous legumes</td>
<td>Lotus sp., Trifolium sp.</td>
</tr>
<tr>
<td>Heather</td>
<td>Calluna vulgaris</td>
</tr>
<tr>
<td>Wattle</td>
<td>Acacia sp.</td>
</tr>
<tr>
<td>Rhubarb</td>
<td>Rhei rhisoma</td>
</tr>
<tr>
<td>Polygonum multiflorum root</td>
<td>Polygonum multiflorum</td>
</tr>
<tr>
<td>Myricaceae bark</td>
<td>Myrica rubra</td>
</tr>
</tbody>
</table>

ASTRINGENCY AND ASTRINGENTS DEFINED

The American Society for Testing and Materials (ASTM) defines astringency as “the complex of sensations due to shrinking, drawing or puckering of the epithelium as a result of exposure to substances such as alums or tannins” (ASTM, 2004). Lee and Lawless (1991) presented evidence suggesting that the tactile attributes of drying, puckering, roughing, and overall astringency may not be totally interchangeable. Since the time-courses of “dry”, “rough”, and astringent sensations are well matched, and the time-courses of puckering, bitterness, and sourness differ subtly from astringency when elicited by compounds commonly accepted as astringent (tannic acid, aluminum sulfate (alum) and tartaric acid), these authors suggest that there may be multiple sub-qualities to astringency. Green (1993) suggests this result implies that puckering, sourness and bitterness are not essential to the sensation of astringency. Lee and Lawless (1991), however, explicitly recommend that future studies of astringency address, and account for the possibility of multiple sub-qualities. Following their own advice, Lawless et al. (1994) developed a lexicon for the description of alum, gallic acid, catechin, citric acid, and their mixtures consisting of the terms drying, roughing, puckering, and astringent. Lawless and Corrigan (1994) provided a graphic interpretation of the relationship between astringent sub-qualities, and their relationship with sourness, and they note that some of what are considered sub-qualities of astringency, such as drying, might be better described as concomitant reactions. Echoing Lawless’ call for semantic agreement in the use of astringency descriptors, Kiehlhorn and Thorngate (1999) in their examination flavan-3-ols conclude “... a refinement of the language used to describe oral sensations is necessitated.”

To further refine discussions and descriptions of perceived astringency, Gawel et al. (2000) used descriptive data and clustering techniques to develop a hierarchical lexicon (“mouth-feel wheel”) to assist in identifying and classifying a wide range of oral sensations elicited by red wine, which included 33 terms or sub-qualities to define astringency (Fig. 1). The majority of the publications considered in this review include, at a minimum, dryness, roughing, and puckering in their definitions of astringency (Gawel, 1998; Simon et al., 1992; Ishikawa and Noble, 1995; Jöbstl et al., 2004), although some substitute constricting for puckering (Breslin et al., 1993), or employ either dryness
Astringency

Figure 1  Red wine mouth-feel wheel. Reproduced from Richard Gawel, A. Oberholster, and I. Leigh Francis (2000); *Australian Journal of Grape and Wine Research* Vol 6(3), 203–207 (Gawel et al., 2000) (with permission from the Australian Society of Viticulture and Oenology).

(Lyman and Green, 1990) or puckering (Bate-Smith, 1954), and others explicitly provide no working definition of astringency (Fischer et al., 1994).

**Astringent Compounds**

Medically, an astringent compound is considered “a drug that causes cells to shrink by precipitating proteins from their surfaces” (CMD, 2007). The current chemical and pharmacological definition of an astringent compound as one that binds and precipitates proteins has not deviated from its Latin root ad stringere, meaning “to bind” (Joslyn and Goldstein, 1964). Astringent compounds can be found in countless products ranging from skin cream to pickles. As described by Joslyn and Goldstein (1964), there are four groups of “true” (i.e., perceptually astringent and capable of reacting with proteins) astringent compounds: salts of multivalent metallic cations (particularly aluminum salts), dehydrating agents (ethanol and acetone), mineral and organic acids, and polyphenols.

Tannins, so named because of their use in the process of tanning animal hides (Hergert, 1989), have long been considered an important component of some plants’ defense mechanisms (Feeny, 1976) where they can act as either a digestive inhibitor or a toxin, depending on the tannin-type and its consumer (Robbins et al., 1991). Whether defense is the primary function of tannins in plants has not yet been determined (Beart et al., 1985; Haslam, 1988). Tannins, also commonly referred to as vegetable polyphenols or polyphenols, are the primary source of astringency in foods and beverages reported to be astringent (Joslyn and Goldstein, 1964; Courregelongue et al., 1999; Bate-Smith, 1954; Arnold et al., 1980). Tannins are categorized as either condensed or hydrolysable, which are composed of proanthocyanidins, and galloyl and hexahydroxylidiphenoyl esters, respectively (Haslam and Lilley, 1988; Bennick, 2002). Tannins have a number of anti-nutritional characteristics including iron absorption inhibition (Disler et al., 1975), esophageal (Warner et al., 1988) and hepatic cancers (Korpassy, 1961), developmental inhibition and anomalies (Featherston and Rogler, 1975; Elkin et al., 1978), and irreversible complexation of digestive
enzymes and dietary proteins (Robbins et al., 1991; Ahmed et al., 1991). However, the review of tannins and human health by Chung et al. (1998) suggests that tannins in moderation are responsible for a number of positive physiological effects. Tannins have also been shown to confer antibacterial, antimicrobial (Scalbert, 1991), anticarcinogenic (Das et al., 1989; Athar et al., 1989), antioxidant (Teissedre et al., 1996), and neuroprotective effects (Sun et al., 1999; Sun et al., 2002; Simonyi et al., 2002).

Traditionally, astringent polyphenols have been defined as having molecular weights between 500 and 3000 Da (Bakker, 1998; Lesscheave and Noble, 2005), but smaller compounds, including 5-O-caffeoylquinic acid, and flavan-3-ol monomers, dimers and trimers, can also elicit astringency (Naish et al., 1993; Peleg et al., 1999). While simple phenols bind proteins weakly (Haslam and Lilley, 1988), it is generally accepted that the greater the degree of polymerization and molecular weight of an astringent compound, the greater its ability to precipitate proteins (Bate-Smith, 1973), and its perceived intensity (Arnold et al., 1980; Peleg et al., 1999). Contrary to this experimental finding, the astringency of ripening fruit and aging red wine is reported to decrease with increased polyphenol polymerization. Ozawa et al. (1987) suggest that the decrease in ripening fruits is not due to changes in polyphenols, but rather to changes in other molecules (e.g. pectin) that inhibit the interaction between polyphenols and mucosal proteins. Taira et al. (1997) provided in vitro evidence of this, as the perceived astringency of persimmons was reduced by the addition of pectin, or by a pectin pre-rinse.

Along with sourness, organic (i.e., acetic, fumeric, quinic, adipic, lactic, malic, tartaric, and citric) (Martin and Pangborn, 1971; Hyde and Pangborn, 1978; Rubico and McDaniel, 1992; Hartwig and McDaniel, 1995; Sowalsky and Noble, 1998), and inorganic acids (hydrochloric and phosphoric) (Rubico and McDaniel, 1992; Hartwig and McDaniel, 1995; Sowalsky and Noble, 1998; Corrigan and Lawless, 1995) can induce sensations of astringency. For organic acids, an inverse, pH-dependent relationship exists between acidity and perceived astringency (Hartwig and McDaniel, 1995; Sowalsky and Noble, 1998; Lawless et al., 1996). Aluminum sulfate (alum) is also an established astringent (Joslyn and Goldstein, 1964; Ward, 1882), and a component of a number of commonly used items including antiperspirants, toothpastes, cosmetics, soaps, eardrops, and topical astringents and styptics.

Ingestion of zinc also induces a primary sensation of astringency (Keast, 2003; Lim and Lawless, 2005), while for iron, copper, and the minerals magnesium and calcium the sensation of astringency is a secondary characteristic (Lim and Lawless, 2005; Lawless et al., 2004). Considering the recent trend toward mineral fortification of foods and beverages (Mitchell, 2004) and the need to make these products palatable (Hurrell, 2002), further research on the astringent properties of minerals is particularly pertinent (Lim and Lawless, 2005).

**MECHANISMS OF ASTRINGENCY PERCEPTION**

**Oral Physiology**

The mammalian tongue houses three types of gustatory papillae: circumvallate, foliate, and fungiform. Polarized, neuroepithelial taste receptor cells (TRCs) in clusters of 50 to 150 are organized into taste buds in each papilla (Beidler, 1978). The apical surface of the taste bud is exposed to the oral cavity through the taste pore, where the microvilli of TRCs make contact with saliva and tastants (Akabas, 1990). Interestingly, TRCs are not static receptor structures. As first demonstrated in the rat, TRCs undergo a progression from basal cells, which are the precursor cell population, through differentiation and death that ranges from 2 days to 3 weeks (Beidler and Smallman, 1965; Hamamichi et al., 2006). TRCs themselves are not neurons; they synapse onto the primary gustatory fibers of the nerves that innervate them, with each gustatory fiber contacting multiple TRCs in multiple taste buds (Scott, 2005).

Besides taste receptors, the oral cavity also houses mechanoreceptors (MRs), which appear to be of equal, if not greater, importance for astringency perception (Weiffenbach, 1993; Trulsson and Essick, 1997). Unlike TRCs, MRs are neurons classified according to the size and character of their receptive field (Kaas, 2004); type I MRs have small and distinct receptive fields, while type II have large, diffuse receptive fields (Jacobs et al., 2002). MRs are further classified depending on whether they are rapidly adapting (RA) or slowly adapting (SA) receptors; RA receptors respond during the dynamic phase of stimulus application and SA receptors respond to both dynamic and static force applications (Jacobs et al., 2002). The MRs of the oral cavity include: Ruffini endings, Merkel cells, Meissner cells (lamellated corpuscles), and free nerve endings (Capra, 1995; Watanabe, 2004). The distribution of MR types varies with oral cavity location. For example, recording from the infraorbital nerve, Johansson et al. (1988) found that about one-third of the MRs at the transitional zone of the upper lip were of the SA I (slow adapting, type I), while Trulsson and Essick (1997), recording from the lingual nerve, found that two-thirds of the MRs stimulated in the lingual mucosa were RA. Trulsson and Essick (1997) suggest that mucosal regions that are deformed during normal functioning (e.g., lips) have a greater proportion of SA afferents, while regions that are mainly used for explorative and manipulative behaviors (e.g., tongue) contain a proportionately greater number of RA fibers. While oral MRs appear to function like those of the skin, they have smaller receptive fields and lower activation thresholds (Trulsson and Essick, 1997).

The facial nerve (cranial nerve (CN) VII), the glossohyaryngeal nerve (CN IX), and the trigeminal nerve (CN V) innervate the oral cavity (Matthews, 2001). Taste buds in the posterior one-third of the tongue receive innervation from the glossohyaryngeal nerve, while those in the anterior two-thirds receive innervation from the chorda tympani branch of the facial nerve.
The members of this 20-protein family are most commonly noted in considerations of astringency as they interact with and precipitate polyphenols (Hagerman, and Butler, 1981). PRPs are characterized by their highly repetitive structure of approximately 19 residues of proline, glycine and glutamine repeated 5–15 times, which alone accounts for 70–80% of the total amino acid content of PRPs (Kauffman and Keller, 1979). The three types of PRPs, basic, acidic, and glycosylated account for 23%, 30%, and 17%, respectively, of the total protein in parotid saliva, with PRPs overall comprising 70% of salivary proteins (Kauffman and Keller, 1979; Bennick, 1982). Although all the PRPs’ functions have yet to be fully elucidated, acidic PRPs may have a role in calcium homeostasis and bacterial binding (Bennick et al., 1981; Amano et al., 1996), and glycosylated PRPs provide lubrication (Hatton et al., 1985) and prevent bacterial agglutination (Bergey et al., 1986). The 11, 6–9 kDa basic PRPs have demonstrated anti-viral activity, and a high affinity for binding tannins (Lu and Bennick, 1998; Hagerman and Butler, 1981; Mehansho et al., 1983; Kauffman et al., 1991).

Treatment of rats and mice with the β-adrenergic receptor agonist isoproterenol causes hypertrophy of the parotid and submandibular glands, and an increase in the production of PRPs (Muenzer et al., 1979, 1979). Interestingly, feeding rats and mice a sorghum diet high in tannins has the same results as isoproterenol injection, but the effects are restricted to the parotid gland (Mehansho et al., 1983; 1985), indicating that PRP levels are mediated by the concentration of tannins in these rodents’ diet. In parallel with the PRP increase, rats fed a high-tannin diet gained weight, implying that the increase in PRP secretion has a positive nutritional effect on the animal (Mehansho et al., 1983). The results of Mehansho et al. (1983) and Asquith et al. (1985) suggest that the increased PRP secretion in mice and rats fed high-tannin diets results from β-adrenergic receptor activation.

While hamsters also responded to isoproterenol treatment with increased PRP levels, their response to a high-tannin diet was quite different than that observed in rats and mice (Mehansho et al., 1987). Hamsters did not respond to a high-tannin diet with a compensatory increase in PRP levels, rather they displayed severely retarded growth and/or death. After six months on a high-tannin diet hamsters were the same size as they were at 3 days old, but when they were switched to a low-tannin diet they grew at close to the same rate as younger animals on a normal diet (Mehansho et al., 1987). The variation in tannin-handling capabilities between rats, mice, and hamsters did not result from differences in either their β-adrenergic receptor complement or their adenylylate cyclase activity (Mehansho et al., 1987). These results, along with those of Robbins et al. (1991) suggest that similar animals with seemingly homologous proteins cannot be expected to react in the same way to dietary tannin stress.

It has been suggested that other mammalian omnivores and herbivores produce PRPs constitutively, at a concentration that reflects the approximate level of polyphenols in their diets (Luck et al., 1994; McArthur et al., 1995). This suggestion is substantiated by the fact that mammalian herbivores whose diets do not naturally include tannin-containing foods, do not produce...
tannin-binding salivary proteins (Austin et al., 1989). High performance liquid chromatography (HPLC) analysis of saliva following the ingestion of highly astringent wine suggests that humans might also mediate PRP levels based on polyphenol consumption, as, for some subjects, late-eluting peaks observed post-ingestion increased in area (Kallithraka et al., 1998). The results of Askiyan (2005) appear to confirm this finding; while some salivary proteins decrease in concentration following ingestion of astringent red wine, others, in the molecular weight range of the PRPs, are reported to increase.

Twelve low molecular weight histatins, or histidine-rich proteins (HRPs) have been isolated (Troxler et al., 1990; Oppenheim et al., 1988), and are only found in saliva (Sabatini et al., 1989). HRP1, 3, and 5, found in parotid and submandibular secretions, are the predominant members of the family, accounting for 80% of all HRP s present in saliva (Lamkin and Oppenheim, 1993). Interestingly, a lower concentration of HRP s is found in whole saliva than in pure parotid and submandibular glandular secretions due to their degradation in whole saliva (Baum et al., 1976). Aside from their roles in the maintenance and protection of tooth enamel, HRP s participate in non-immune oral defense through their potent anti-microbial action (Lamkin and Oppenheim, 1993; Oppenheim et al., 1986; 2007).

\( \alpha \)-amylase catalyzes the hydrolysis of \( \alpha (1,4) \)glycosidic bonds of polysaccharides, and is found in organisms ranging from insects to humans. Salivary \( \alpha \)-amylase is composed of two families; the glycylated A family, and the non-glycylated B family. The A family comprise isoenzymes 1, 3, and 5, and family B comprise isoenzymes 2, 4 and 6 (Oppenheim et al., 2007; Keller et al., 1971). \( \alpha \)-amylase secretion from the parotid gland increases with stimulation by tastes (Froehlich et al., 1987), and its bacterial-binding capacity suggests it may contribute to bacterial clearance, and it has been detected in dental plaque (Scannapieco et al., 1993).

Lactoferrin (Lf), a relatively minor component of saliva (Dodds et al., 2005), is a member of the transferrin family of non-heme iron-binding proteins (Levay and Viljoen, 1995) that is present in all salivary glands (Reitamo et al., 1980). While multiple isoforms of Lf exist, the iron-binding Lf is an 80 kDa, single chain protein, whose tertiary structure consists of two ferric- and glycan-binding globular lobes (Levay and Viljoen, 1995; Lonnerdal and Iyer, 1995). Lf acts as an antibacterial via nutritional immunity, whereby it makes iron unavailable as a food source (Humphrey and Williamson, 2001). Lf also has direct bacteriostatic effects; for some bacteria these effects depend on Lf being iron-free, while for others the effects depend on Lf being bound by iron (Aguijera et al., 1998; Fine and Furgang, 2002).

Mucin-glycoproteins, or mucins, are principally responsible for the viscoelastic properties of all mucosal secretions, including saliva (Schenkels et al., 1995; Tabak, 1990). The submandibular and sublingual glands, along with some of the minor salivary glands, secrete the two main salivary mucins, MG1 and MG2 (Tabak, 1995). MG1 is a large multi-subunit superstructure, with a high-carbohydrate content and hydrophobic pockets, whose molecular weight is in excess of 1000 kDa (Tabak, 1990; Loomis et al., 1987). MG2 is a lower molecular weight (200–250 kDa) single polypeptide chain, enriched in threonine, serine, proline, and alanine (Loomis et al., 1987). As might be expected, MG1 provides better lubrication than MG2 (Aguirre et al., 1989), and binds tightly to teeth contributing to the protective enamel pellicle (Humphrey and Williamson, 2001). Interestingly, MG2 is easily displaced from enamel, but has demonstrated important functions in the aggregation and clearance of oral microorganisms (Schenkels et al., 1995).

### Polyphenol-Protein Binding

Some have suggested that the binding of polyphenols by proteins is a defense mechanism that inhibits harmful tannins before they become bioavailable and affect the gastrointestinal tract (Lu and Bennick, 1998; Hagerman and Butler, 1981; Mehansho et al., 1987; McAurthur et al., 1995; Mehansho et al., 1987, 1995). Another plausible explanation is tannin detection. Based on the particle sizes resulting from mastication, and the unlikelihood that mastication would release all of the tannins located in intracellular vacuoles thus leaving some tannins to traverse the gastrointestinal system unbound by proteins, it is also theorized that the interaction of tannins with salivary proteins and the sensation of astringency are part of a mechanism for the detection of potentially harmful astringent compounds (Prinz and Lucas, 2000).

Regardless of whether it is a defense or a detection mechanism, the protein-binding ability of polyphenols is well documented, and has been demonstrated with a variety of proteins besides salivary PRPs including casein (Jöbstl et al., 2004; Luck et al., 1994), gelatin (Hagerman and Butler, 1981; Oh et al., 1980; Yokotsuka and Singleton, 1995; Siebert et al., 1996; Edelmann and Lendl, 2002), bovine serum albumin (BSA) (Hagerman and Butler, 1981; 1980; 1980), haemaglobin (Bate-Smith, 1973), and Lf, and 

\( \alpha \)-amylase are also capable of polyphenol-binding (Gambuti et al., 2006; de Freitas and Mateus, 2001; 2001), and that, along with the PRPs and HRPs, these proteins are involved with the sensation of astringency.

PRPs appear to have a higher affinity for condensed tannins than for hydrolysable tannins, and for polymers over monomers (Yokotsuka and Singleton, 1995; Baxter et al., 1997). Similarly, larger PRPs have a greater affinity for tannins than smaller PRPs or peptide fragments (Hagerman and Butler, 1981; Charlton et al., 2001). The greater affinity of larger, polymerized polyphenols for proteins, and vice versa, has been attributed to the multi-dentate nature of polyphenols, which allows a single polyphenol to bind multiple residues of the protein (Jöbstl et al., 2004; Baxter et al., 1997; Charlton et al., 2002). In the case of hydrolysable tannins, the affinity of tannin-protein binding is directly related to the degree of galloylation, as pentagalloylgucose binds proteins with greater affinity than monogalloylgucose (Baxter et al., 1997; Charlton et al., 2002; Kawamoto et al., 1995).
The effect of galloylation on binding affinity reaches a plateau with the pentagalloylated molecules, as the affinity of hepta- and octagalloylglucose for PRPs is of the same order as tetra- and pentagalloylglucose (McManus et al., 1985; Bacon and Rhodes, 2000).

Protein-tannin complexes have been described as both soluble and insoluble, and recent data suggests that complex solubility is dependent on a number of variables. Using BSA and a condensed tannin, Hagerman and Robbins (1987) demonstrated that under optimal protein:polyphenol ratios and pH conditions, protein-polyphenol complexes are insoluble. However, in the presence of excess protein, the protein-polyphenol complexes that form are soluble as there is not enough tannin to sufficiently crosslink proteins and form aggregates (Hagerman and Robbins, 1987). Luck et al. (1994) confirmed these results using gelatin and a hydrolysable tannin, but using salivary PRPs they were unable to resolve the polyphenol-protein complex, regardless of how much protein was added. These findings suggest that the stability of polyphenol-protein complexes depends not only on the environmental conditions of the reaction (Hagerman and Robbins, 1987; Kawamoto and Nakatsubo, 1997), but also on the types of polyphenol and protein used.

The first studies of polyphenol-protein binding used condensed tannins in their examinations, and the results suggested mainly hydrogen bonding between the hydroxyl groups of the polyphenols and the carbonyl groups of the proteins (Hagerman and Butler, 1981, 1980, 1980). Subsequent studies have confirmed that for condensed tannins, hydrogen bonding is the driving force of the interaction (Oh et al., 1980; Hagerman et al., 1998; Simon et al., 2003), but in some cases, it appears that hydrophobic interactions may be the basis for the complexation of tannins with protein (Jöbstl et al., 2004; Luck et al., 1994; Baxter et al., 1997; Charlton et al., 2002; Hagerman et al., 1998).

Hagerman et al. (1998) suggest that polyphenol polarity is the main predictor of the type of association that will occur between polyphenols and proteins (i.e., hydrogen bond vs. hydrophobic interaction), with polar polyphenols forming hydrogen bonds and nonpolar polyphenols forming hydrophobic interactions.

Charlton et al. (2002) put forth a 3-stage model of the binding and precipitation of PRPs by polyphenols. Jöbstl et al. (2004) have confirmed and expanded the model (Fig. 2). In step 1, the binding of multiple multidentate polyphenols to several sites on the protein causes the previously randomly coiled protein to coil around the polyphenol, making the protein more compact. In the second stage, the polyphenol fractions of the protein-phenol complexes cross-link forming polyphenol bridges and creating protein dimers, and finally (step 3), the dimers aggregate to form large complexes and precipitate. The initial polyphenol-protein interaction results from the binding of the hydrophobic face of the polyphenol's aromatic ring with the pyrrolidine ring of the protein’s proline residues (Charlton et al., 2002). Jöbstl et al. (2004) suggest that this 3-stage model is consistent with the time-course of astringency, but this assertion has yet to be confirmed.

Besides their antibacterial and antifungal roles, HRPs have also been identified as polyphenol-binding proteins, which suggests a possible role for them in the perception of astringency (Yan and Bennick, 1995; Naurato et al., 1999). While HRP1, HRP3, HRP5, and HRP7 are capable of binding tannins, the amount of tannin bound appears to vary with the type of tannin used (i.e., condensed vs. hydrolysable) (Naurato et al., 1999). Yan and Bennick (1995) demonstrated that HRP5 was more efficient than PRP1 at precipitating tannic acid, a hydrolysable tannin, and condensed tannin at a pH of 7.4, but PRP1 was a more effective precipitator of both polyphenol preparations at a pH of 3.0.

Although it does so with lower affinity than the PRPs, α-amylase readily binds both tannin types, which inhibits its enzymatic activity (de Freitas and Mateus, 2001; Kandra et al., 2004; Zajacz et al., 2006). The α-amylase-tannin interaction is reversible, leaving α-amylase’s activity intact after its release from the tannin, and is inhibited by both HRP5 and an acidic PRP (Yan and Bennick, 1995; Oh et al., 1980). Similarly, mucins have been shown to bind polyphenols (Gambuti et al., 2006; Monteleone et al., 2004; Conelli et al., 2006), and, along with α-amylase, Lf and two glycosylated PRPs, decrease in the saliva following the ingestion of astringent wine (Gambuti et al., 2006).

Together, these results strongly suggest that tannin-binding is a redundant function of salivary proteins, which confirms its physiological importance (Bennick, 2002).

**ASTRINGENCY AS A TACTILE SENSATION**

In 1954, Bate-Smith (1954) first suggested that astringency is a feeling not a taste, and since then the postulated tactile nature of astringency has been accepted as a paradigm. Joslyn and
Goldstein (1964) furthered the tactile theory of astringency by asserting that “(T)he precipitation of tissue proteins is accompanied by the shrinkage of tissue due to a loss of water and a decrease in the permeability of this tissue to water and solutes.” They further postulated that astringency might be the result of a constriction or closure of the salivary ducts, or inhibition of the salivary gland causing a decrease in available saliva. Given that salivary flow rate increases in response to an astringent compound in complex and model solutions (Lyman and Green 1990; Fischer et al., 1994; Hyde and Pangborn, 1978), this is not tenable.

Evidence supporting a reduction in salivary PRPs following ingestion of an astringent solution came from Kallithraka et al. (1998), who attributed the decrease in tentative PRPs following wine intake to their precipitation resulting from their complexation with phenols. While some have suggested that the precipitation of salivary and epithelial proteins leads to a constriction of the oral epithelium (Joslyn and Goldstein, 1964; Lyman and Green 1990), or that astringent substances change the oral epithelium causing it to feel rough (Jellinek, 1985), a 2-stage model where the polyphenol-protein interaction precedes the binding of the complex to the epithelial proteins has also been put forth (Guinard et al., 1998) and expanded to include recent polyphenol-polyphenol binding data (Jöbstl et al., 2004). The current “lubrication” theory of astringency asserts that after astringent compounds strip the oral cavity of mucosal and epithelial proteins that confer lubrication, the increased friction between the surfaces of the oral cavity stimulates mechanoreceptors (Lyman and Green, 1990). The interaction of proteins and polyphenols in solution results in the development of a haze or cloudiness (Monteleone et al., 2004; de Freitas and Mateus, 2001), which can be observed in complex matrices such as beer and wine (Siebert et al., 1996), and simple mixtures of saliva and tannic acid (Horne et al., 2002). A negative correlation between perceived astringency ratings and haze developing capacity was observed when individuals’ saliva was mixed with tannic acid (Horne et al., 2002), suggesting that a higher level of salivary proteins available to bind polyphenols results in a decrease in perceived astringency. Conversely, the results of Kallithraka et al. (2001) suggest that protein binding and precipitation are not directly related to the perception of astringency, as the time course of chemical astringency (i.e., protein binding) was not correlated to its perception. This result corroborates the findings of Guinard et al. (1998) who found no correlation between the perception of astringency and salivary protein composition.

The only direct physiological data indicating that astringency is a tactile sensation mediated by non-gustatory mechanisms comes from research presented by Breslin et al. (2005) and Lim and Lawless (2005). These studies demonstrate that aluminum sulfate (Breslin et al., 1993) and copper sulfate (Lim and Lawless, 2005) elicit the sensation of astringency when applied to the area between the gum and the upper lip, an area of the mouth generally accepted to be devoid of taste receptors (Jones, 1954). Green (1993) suggests that the MRs responsible for astringency may be RA afferents that have been identified in the chorda tympani and lingual nerve (Trulsson and Essick, 1997; Biedenbach and Chan, 1971).

Besides this direct evidence, the theory of astringency as a tactile sensation is based on characteristic differences between astringency and the five accepted gustatory sensations. One line of argument concerns adaptation, “…the disappearance of taste impressions under continuous stimulation” (Moskowitz, 1978), likely resulting from receptor-dependent mechanisms such as desensitization (O’Mahony, 1986; Bohm et al., 1997; Meyerhof et al., 2005). Adaptation of each of the five tastes has been demonstrated (Abrahams et al., 1937; Krakauer and Dallenbach, 1937; McBurney and Lucas, 1966; Meiselman, 1968; O’Mahony and Wong, 1989; Gent and McBurney, 1978). It has been suggested that, since the perceived intensity of an astringent stimulus increases with repeated ingestion (Lyman and Green 1990; Courregelounge et al., 1999; Guinard et al., 1986) and other tastes decrease in intensity with repeated ingestion, astringency cannot be a gustatory sensation (Green, 1993). In contradiction to this line of reasoning, the perceived intensity of bitterness, which is an accepted gustatory sensation, has also been shown to increase with sustained or repeated ingestions (Lyman and Green 1990; McNulty and Moskowitz, 1974; Guinard et al., 1986). While Lyman and Green (1990) note of their results that “(T)he lack of adaptation may have been due in part to the intermittent (once per minute) pattern of stimulation, which may have allowed at least partial recovery from adaptation”, they do not indicate how the recovery from adaptation might result in an increase in intensity with repeated sampling.

The ability of augmented oral lubrication to decrease the astringency intensity of polyphenols and alum has also been put forth as evidence that astringency is a tactile phenomenon (Lyman and Green 1990; Courregelounge et al., 1999; Breslin et al., 1993; Smith et al., 1996; Smith and Noble, 1998; Peleg and Noble, 1999; Brannan et al., 2001). A number of compounds have been employed to increase the viscosity of astringent solutions, such as carboxymethylcellulose (CMC) (Courregelounge et al., 1999; Smith et al., 1996; Smith and Noble, 1998; Peleg and Noble, 1999), actual and artificial saliva (Breslin et al., 1993), temperature (Peleg and Noble, 1999), and sucrose (Lyman and Green 1990; Courregelounge et al., 1999; Breslin et al., 1993; Ishikawa and Noble, 1995; Smith et al., 1996). As discussed below, the effect of sucrose on astringency is likely not solely due to its function as a thickener. If friction between oral surfaces leads to the activation of MRs and the perception of astringency, one might expect that a universal lubricant like oil would reduce perceived astringency, but this does not always appear to be the case. While a mixture of corn oil and xanthan gum very effectively decreased the perceived astringency of alum (Breslin et al., 1993; Brannan et al., 2001), corn oil alone had no effect on the astringency elicited by soymilk (Courregelounge et al., 1999). The variation in these results may be due to differences in the astringent mechanisms of soymilk and alum. However, these mechanisms may also suggest that the ability of viscous agents to bind tannins may be of greater importance in mediating astringency than their capacity as simple lubricants. Matrix viscosity...
has been shown to affect the intensity of accepted gustatory sensations and complex flavours (Moskowitz and Arabie, 1970; Christensen, 1980; Malkki et al., 1993; Walker and Prescott, 2000; Hollowood et al., 2002), but not bitterness (Smith et al., 1996). These results can be taken to suggest that viscosity, by some as yet unknown mechanism, is a general modulator of taste and flavour rather than as evidence that astringency is a tactile phenomenon.

**ASTRINGENCY AS A TASTE**

The results of Kawamura et al. (1969) directly demonstrate that tannic acid interacts with the oral epithelium, and provides evidence that tannic acid does not directly interact with MRs. They also show that, not only is astringency an unpalatable sensation in rats, tannic acid stimulates fibers in the glossopharyngeal nerve and the chorda tympani, but not the lingual nerve. Based on their findings that tannic, tartaric, and gallic acids elicit a rapid and reversible response in the chorda tympani, but not the lingual nerve, Schiffman et al. (1992) concluded that astringency is a taste sensation.

A direct conclusion regarding the gustatory nature of astringency is difficult to make based on the electrophysiological data of Kawamura et al. (1969) and Schiffman et al. (1992). While their results clearly indicate that the chorda tympani and glossopharyngeal nerve are responsive to astringent compounds, the basis for their conclusion that astringency is a taste may not be valid. Schiffman et al. (1992) describe the lingual nerve as responsive to tactile, thermal, and pain sensations, but this nerve is also responsive to chemical stimulation (Wang et al., 1993) and Schiffman et al. (1992) themselves demonstrate that it is responsive to some of the high-concentration, low-pH astringents presented. They conclude that since astringent compounds that stimulated the chorda tympani did not stimulate the lingual nerve, MRs cannot be involved in the perception of astringency.

These studies clearly demonstrate that the collaborative innervation of the anterior two-thirds of the tongue by the chorda tympani and the lingual nerve confers the ability to respond to a wide array of chemical stimuli, but they fall short of providing definitive proof that astringency is a taste. It is interesting to note that while the tactile theory of astringency postulates that its tactile nature stems from the increased friction between oral surfaces after the loss of lubrication, both Kawamura et al. (1969) and Schiffman et al. (1992) claim that the unresponsiveness of the lingual nerve, and thus MRs, to the direct application of astringent stimuli is evidence that astringency is not tactile.

The interaction of astringent compounds with ion channels has also been presented as evidence of the gustatory basis of astringency. Simon et al. (1992) further verified that astringents are capable of interacting with proteins through their demonstration that tannic acid and aluminum salts inhibit amiloride-sensitive Na+ channels in preparations of isolated canine lingual epithelia. Similarly, the ability of tannic acid and catechin to alter the membrane potential of a lipid taste sensor has been presented as support for astringency being a gustatory sensation (Iiyama et al., 1995). These authors found that the effectiveness of catechin and tannic acid in modulating membrane potential was similar to that of bitter and sour tastants. The cellular effects of astringent substances have been found to culminate in cortical signaling (Critchley and Rolls, 1996). Single-cell recordings from neurons located in the orbitofrontal region, which includes the secondary taste cortex, clearly illustrate that a subpopulation of neurons, the “tannic acid best” neurons, are responsive to as little as 1 mM tannic acid. Six of the 74 cells examined in two male behaving rhesus macaques responded to oral application of the astringent with a significant increase in frequency of action potential firing. An increase firing frequency for tannic acid best neurons was not observed when hydrochloric acid was applied, suggesting that these neurons are specific for tannic acid, or some component of it (Critchley and Rolls, 1996).

All of the studies discussed here conclude that astringency should be considered a distinct taste quality, like sweet, sour, salty, bitter, and umami. Alternatively, we suggest that while these results indicate that astringent compounds are capable of interacting with cellular receptors, this does not discount the tactile theory of astringency. Taken together, the findings discussed above suggest that, for some astringent compounds, the sensation of astringency may be the result of both taste and tactile mechanisms working together.

**TIME-COURSE AND MEASUREMENT**

In contrast to taste sensations, the perception of astringency builds slowly in intensity after ingestion and persists for a longer duration. Thus, for many psychophysical studies and for product development research, time-intensity (TI) methods, where the perceived intensity of the sensation of interest is recorded for a specified duration, may be more appropriate for fully describing astringency responses (Noble, 1995).

The perceived intensity of astringency increases linearly to a maximum at 13–15 seconds post-ingestion, regardless of the concentration of the astringent compound (Ishikawa and Noble, 1995; Guinard et al., 1986). Using an experimental design considered comparable to normal wine consumption patterns, Guinard et al. (1986) demonstrated that the maximum intensity and the time to maximum (i.e., the time required to reach the maximum intensity) of perceived astringency is unchanged with repeated white wine ingestion (i.e., ingestion, swallowing, return to astringency intensity of zero, repeat), but the total duration of the astringent sensation increased with repeated ingestion. When wine was ingested repeatedly with only a 20- or 40-second interval between ingestions, a clear, significant increase in maximum intensity was observed (Guinard et al., 1986). Subsequently, Lyman and Green (1990) demonstrated that the intensity of a solution of tannic acid would continue to increase with repetitive intake (i.e., 10 ml in the mouth for 10 seconds per minute) for 20 minutes.
According to Lee and Lawless (1991) the perception of astringency elicited by 750 mg/l tannic acid was not entirely extinguished six minutes post-expectoration. In contrast, the results of Guinard et al. (1986), based on ingestion of white wine with 500 mg/l added tannic acid, indicate that astringency reaches intensity levels close to those pre-ingestion within 70 seconds after expectoration. Using 1000 mg/l tannic acid in water, Valentova et al. (2002) found that some residual astringency was perceivable at 100 seconds post-ingestion, and the results of Fischer et al. (1994) place extinction at approximately 120 seconds, both of which are in accord with Guinard et al. (1986).

Overall, these results reinforce the importance of experimental design and protocol when conducting sensory trials with astringents in order to account for the risk of carry-over and additive effects. One practical approach to help minimize these risks in psychophysical studies is the use of pectin mouth-rinse between samples (Colonna et al., 2004). Presumably, pectin is competing with PRPs for polyphenol-binding, and is becoming increasingly employed to reduce carry-over and additive effects (Pickering and Robert, 2006; Pickering et al., 2006).

The phenomenon of multiple astringency sub-qualities—at least at the perceptual level—discussed earlier raises concerns about the limitation of traditional one-dimensional visual-analog approaches (e.g., “rate the astringency intensity on the line scale”) in capturing the full range of sensations experienced, particularly for products that elicit complex tactile sensations, such as red wine. The red wine “mouthfeel-wheel” (Gawel et al., 2000) is one recent innovation to assist enologists to more precisely and comprehensively describe and measure astringency (Fig. 1). While this multi-tiered lexicon includes some terms that appear more hedonic or composite in nature (e.g., “aggressive,” “rich,” “activity”), it is nonetheless proving valuable for describing the full range of astringent sensations elicited by red wines (Pickering and Robert, 2006; Geddes et al., 2001; De Miglio et al., 2002; Francis et al., 2002; Vidal et al., 2003; De Miglio, 2005).

**MODULATORS OF ASTRINGENCY**

**pH**

While acids are themselves astringent (Rubico and McDaniel, 1992; Hartwig and McDaniel, 1995; Sowalsky and Noble, 1998; Corrigan and Lawless, 1995), pH also affects the perceived intensity of astringents. Dealcoholized white wine with 1% ethanol, 1500 mg/l tannic acid, and a pH of 3.0 was found to be more astringent than the same wine with a pH of 3.6 (Fischer et al., 1994). Similar results have been obtained for red wines across an increasing pH series (pH 2.2, 2.4, 2.6, and 2.8) (De Miglio, 2005), and when different acids are used (Kallithraka et al., 1997). Guinard et al. (1986) did not find the inverse relationship of pH and astringency in their high-phenol red wines, and suggest that this is because of the high starting astringency of the wines, which may have effectively precipitated the majority of salivary proteins negating a change in astringency by the addition of acids. In cranberry juice, a decrease in pH increased the perceived astringency, regardless of the temperature or viscosity of the juice (Peleg and Noble, 1999). Even simple aqueous solutions of phenolic compounds (i.e., grape seed tannins, tannic acid, catechin, gallic acid), and model solutions are affected by a decrease in pH (Kallithraka et al., 1997; Guinard et al., 1986; Peleg et al., 1998). The increased intensity of perceived astringency is likely due to the decrease in charged phenolate ions, which are unable to form hydrogen bonds with proteins, at low pH (Sowalsky and Noble, 1998; Guinard et al., 1986).

Interestingly, Peleg et al. (1998) found that the astringency of alum decreased with the addition of acid, and attribute this to the chelation of the aluminum ions in alum by acids, reducing its availability to interact with salivary proteins. These results indicate that alum and phenolic astringents cannot be used interchangeably in psychophysical studies (Peleg et al., 1998), and present the possibility that alum and tannic acid might elicit the sensation of astringency through different mechanisms.

**Gustatory Sensations and Cross-modality Effects**

Four basic gustatory sensations have been accepted as “tastes,” sweet, sour, bitter, and salty. A fifth taste, umami, while controversial is also generally accepted (Bradbury, 2004). Taste transduction occurs through either GPCRs (sweet, bitter, and umami) or ion channels (salty and sour), and while it is still unclear whether astringency should be classified as a taste or tactile sensation, taste qualities have been shown to physically and psychologically interact with and influence perception of astringency.

Lea and Arnold (1978) characterized bitterness and mouth dryness as “twin sensations” since the two are often confused and almost all phenolic compounds that elicit astringency are also bitter. Other studies have also noted similarities between bitterness and astringency, and the ability to elicit both with the same compounds (Ishikawa and Noble, 1995; Peleg et al., 1999). However, polymeric tannins are more astringent than bitter while monomeric tannins are more bitter than astringent (Robichaud and Noble, 1990). Lee and Lawless (1991) dissected the sensations elicited by tannic acid and found that bitterness, along with sourness, a taste induced by acids, could be distinguished from astringency and separately rated, confirming discrete differences between them. The results of Bertino and Lawless (1993) further confirmed that panelists are able to distinguish between astringent qualities (e.g., dry, puckery, astringent) and gustatory sensations (e.g., bitter, sour, salty).

While evidence has been presented suggesting that all true tastes moderate astringency intensity (Brannan et al., 2001), most research in this regard has focused on sweetness (Lyman and Green 1990; Courregelongue et al., 1999; Breslin et al., 1993; Ishikawa and Noble, 1995; Smith et al., 1996; Brannan et al., 2001; Speegle, 2002). The astringency of tannic acid (Lyman and Green 1990) and red wine (Ishikawa and Noble, 1995) is one recent innovation to assist enologists to more precisely and comprehensively describe and measure astringency (Fig. 1). While this multi-tiered lexicon includes some terms that appear more hedonic or composite in nature (e.g., “aggressive,” “rich,” “activity”), it is nonetheless proving valuable for describing the full range of astringent sensations elicited by red wines (Pickering and Robert, 2006; Geddes et al., 2001; De Miglio et al., 2002; Francis et al., 2002; Vidal et al., 2003; De Miglio, 2005).
to tannic acid. The presence of sucrose, possibly by interfering with the binding of tannins and salivary proteins. Equi-sweet solutions comprised of the non-nutritive sweetener aspartame have also been effective in reducing oral astringency (Speegle, 2002), but not to the same extent as sucrose (Lyman and Green, 1990). These authors suggest that the viscosity of the aspartame solution, which was markedly lower than that of the sucrose solution, was the reason it was not as effective as sucrose in reducing astringency. Using similar concentrations of aspartame and sucrose, Smith et al. (1996) presented contradictory evidence; they found that the sweetener did not affect the astringency of grape seed tannin (GST) solutions.

**VARIATION IN THE PERCEPTION OF ASTRINGENCY**

### Salivary Flow Rate

An individual’s salivary flow rate may affect their perception of astringency. Using white wine fortified with tannic acid, Fischer et al. (1994) demonstrated that subjects classified as having high and medium salivary flow rates perceived astringency sooner, for shorter duration, and with less intensity than those classified as having low salivary flow rates. The results of Ishikawa and Noble (1995), who divided participants into low (mean = 1.92 g/min) and high (mean = 3.73 g/min) salivary flow rates, corroborated those findings using a red wine matrix. Guinard et al. (1998) found no affect of salivary flow rate on the perception of astringency elicited by tannic acid fortified white wine. It has been postulated that more rapid re-lubrication (through a number of possible mechanisms) of the oral cavity occurs in individuals with higher flow rates, thus reducing the duration and intensity of the perceived astringency (Ishikawa and Noble, 1995). In contrast, Peleg et al. (1999) found that individuals with a high salivary flow rate perceived the intensity of astringent polyphenols as more intense than those with a low flow rate, while all other TI parameters (i.e., time to maximum, total duration, time (from ingestion) to decay to 60% and 30% of maximum intensity) examined were unaffected by flow rate categorization. In their examination of organic acid astringency, Sowalsky and Noble (1998) found no difference in the astringency ratings of malic, lactic, tartaric, and citric acid between low, medium, and high salivary flow groups, which is in accordance with the results of Smith et al. (1996).

Foods and beverages, including alcoholic beverages, are typical sialogogues (Martin and Pangborn, 1971; Guinard et al., 1998; Guinard et al., 1997), and astringent compounds have also been shown to alter salivary flow rates (Hyde and Pangborn, 1978). Wine (Hyde and Pangborn, 1978), and wine augmented with tannic acid (Fischer et al., 1994; Guinard et al., 1998) have been shown to increase the rate at which saliva flows into the oral cavity, although Lyman and Green (1990) found no difference in salivary volume when ingestion of water was compared to tannic acid.

**PROP Status**

Sensitivity to 6-n-propylthiouracil (PROP) and phenylthiocarbamid (PTC) is genetically inherited and has been thought to follow an incomplete dominance mode of inheritance (Guo and Reed, 2001), allowing the classification of individuals into three groups reflecting their “PROP taster status” (PTS): non-tasters (NTs), medium-tasters (MTs) and super-tasters (STs) (Bartoshuk, 2000). Recent molecular data indicates that there are three broad categories of PROP/PTC receptors encoded by the hTAS2R38 gene; those that are sensitive to PROP/PTC, those with intermediate sensitivity, and those with little or no sensitivity (Bufl et al., 2005). STs experience PROP as intensely bitter, MTs perceive PROP but less intensely than STs, and NTs cannot taste PROP or experience it as a very mild sensation.

PTS serves as an index of general sensitivity to oral stimuli; the perceptual differences between PTS groups extend to other bitterants (Bartoshuk, 1979; Bartoshuk et al., 1988, 1993, 1996; Delwiche et al., 2001); salty compounds (Bartoshuk et al., 1998), sweet compounds (Gent and Bartoshuk, 1983), and substances that produce oral irritation/pain (Cunningham, 2000; Karrer and Bartoshuk, 1991) and tactile sensations (Duffy et al., 1996; Tepper and Nurse, 1997).

PTS is correlated with gender (Bartoshuk et al., 1994), food preferences (Drewnowski et al., 1997, 1999), alcoholism (Peclacht and Danowski, 1992; DiCarlo and Powers, 1998), and a number of diseases (Shepard and Gartler, 1960; Milunicova et al., 1969; Ahuja et al., 1977; Schlosberg and Baruch, 1992; Ali et al., 1994), demonstrating its importance in physiological function. The underlying basis for the perceptual differences between PTS groups appears itself to be physiological. Miller and Reedy (Miller and Reedy, 1990) found that individuals with a greater number of fungiform papillae have a greater number of taste pores, and rated PROP higher in intensity than those with low papillae and taste pore numbers. Later work (Reedy et al., 1993) confirmed that PROP STs have more fungiform papillae and taste pores than MTs and NTs. An interesting correlate has also been found between PTS and the ability to perceive tactile stimuli and differentiate tactile stimuli based on small differences; STs are better able to perceive small particles placed on the tongue than NTs (Tepper and Nurse, 1997; Chopra et al., 2002), recognize raised alphabet letters by tongue (Essick et al., 2003), and have a lower threshold for tactile stimulation (i.e., Von Frey filament stimulation) (Yackinous and Guinard, 2001). As the diameter of fungiform papillae is smaller and their density greater in STs, this, in conjunction with greater trigeminal innervation, might account for their greater tactile acuity.

While numerous studies have examined the interaction, a direct relationship between PROP status and astringency is not clear, as, to-date, results have been conflicting. Contrary to findings that PROP taster status does not affect astringency (Ishikawa and Noble, 1995; Sowalsky and Noble, 1998), Pickering et al. (2004) demonstrated that PROP STs and MTs found the astringency of red wines significantly more intense than NTs, but in the same study reported that the astringency intensity of alum
was not PTS-dependent. Later, Pickering et al. (2006) reported that STs perceive the intensity of alum with greater intensity than NTs, and suggest that the contradiction of this result with their previous findings may be attributable to the use of a substantially higher concentration in the former study. The lack of a PTS effect on perceived astringency in other studies may have been due to the technique used to categorize individuals and separate the groups, with more sensitive suprathreshold measures being used by Pickering et al. (2006). Imm and Lawless (1996) and Courregelonque et al. (1999) also found that PTS impacted the perception of astringency, with NTs perceiving the astringency of alum and soymilk, respectively, higher than PROP tasters.

Interestingly, Pickering and Roberts (2006) found that STs rated the “overall astringency” of red wines lower than NTs, but those same STs rated the textural sub-qualities of the wines higher (Table 2). They suggest that previous findings of higher astringency ratings by STs—where this was the only tactile attribute measured by the subjects—may be due, in part, to a dumping effect (Clark and Lawless, 1994). Given the greater tactile acuity of STs, presumably due to their more densely packed fungiform papillae and/or greater trigeminal innervation, they may be better equipped to discriminate and rate finer qualities of astringency and other tactile sensations.

While the majority of studies indicate that PTS is a factor in an individual’s perception of astringency, we recommend that future studies also incorporate a physiological measure (such as papillae density) to validate/assist with PTS classifications, as assignment of PTS based solely on PROP intensity ratings may lead to errors in categorization.

**CONCLUSION**

Taken together, the literature demonstrates that astringency is a complex, multifaceted sensation whose examination is complicated by a number of variables. While many studies have examined astringency, the lack of a clear, accepted definition that delineates the oral sensations it encompasses makes it difficult to effectively compare results. The potential interaction of astringency and basic tastes in many complex foods and beverages suggests that the physiological and psychological mechanisms underlying the perception of astringency should be further studied using simple, single-component stimuli. The use of single component stimuli would also give researchers greater ability to draw causal relationships between the stimuli used and the actual sensations perceived. Aroma is an interesting potential mediator of astringency that has recently received attention (Pickering et al., 2006); investigation into the possible role of olfaction may provide important insights into central and peripheral processes affecting astringency perception.

The literature suggests that distinct astringent compounds may utilize different pathways in eliciting astringency and that both mechanical and chemical stimulation may contribute to the sensation. Given the time course of neuronal and perceptual processes reviewed here, a possible mechanism for astringency could involve the ingestion of astringent compounds initially detected by the central nervous system, providing an assessment of the compounds and the current state of the oral and gastrointestinal environment to initiate the appropriate response through the peripheral nervous and endocrine systems (i.e., sequestering of astringent compounds by salivary proteins). Given the importance of astringent compounds to food preference (Marks et al., 1988; Kawamura et al., 1969; Glendinning, 1992) and health (Chung et al., 1998), elucidation of the underlying mechanisms, and delineation of genetic and physiological contributions to the perception of astringency are necessary and timely.

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