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Mechanisms of umami taste perception: From molecular level to brain imaging

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ABSTRACT

Due to unique characteristics, umami substances have gained much attention in the food industry during the past decade as potential replacers to sodium or fat to increase food palatability. Umami is not only known to increase appetite, but also to increase satiety, and hence could be used to control food intake. Therefore, it is important to understand the mechanism(s) involved in umami taste perception. This review discusses current knowledge of the mechanism(s) of umami perception from receptor level to human brain imaging. New findings regarding the molecular mechanisms for detecting umami tastes and their pathway(s), and the peripheral and central coding to umami taste are reviewed. The representation of umami in the human brain and the individual variation in detecting umami taste and associations with genotype are discussed. The presence of umami taste receptors in the gastrointestinal tract, and the interactions between the brain and gut are highlighted. The review concludes that more research is required into umami taste perception to include not only oral umami taste perception, but also the wider “whole body” signaling mechanisms, to explore the interaction between the brain and gut in response to umami perception and ingestion.

Introduction

There are five widely recognized and accepted basic tastes (salty, sweet, bitter, sour and umami) (Lindemann 2001). In addition, fat (Deepankumar et al. 2019; Khan et al. 2019; Roper and Chaudhari 2017) and kokumi (Rhyu et al. 2020; Ueda et al. 1990) are among proposed candidates for a sixth basic taste. Taste acts as a nutrient-toxin detection system in humans. For instance, sweet indicates carbohydrates as a source of energy, salt informs intake of sodium and dietary electrolyte balance, and umami reflects amino acids in protein (Chandrashekar et al. 2006), whereas the aversive taste of sour (acidic taste) and bitter indicate unripe or overripe foods, and potentially harmful poisons (Kinnamon 2012; Lee and Cohen 2015). However, individual variation in taste perception and food choice play an important role in dietary choices and food intake.

Umami is described as a ‘savory’ taste, and was first discovered in 1908 by K. Ikeda, but only in 2009 recognized as a basic taste quality (Kurita 2009). The prototypical compound that elicits umami taste in humans is monosodium glutamate (MSG), a substance found naturally in some foods including meat, vegetables, seafood and cheese, and is known to increase the palatability of food (Yamaguchi and Ninomiya 2000). Umami taste can be enhanced due to the synergistic effect when glutamate is combined with 5’-nucleotide monophosphates, especially inosine 5’-monophosphate (IMP) and guanosine 5’-monophosphate (GMP) (Li 2009; Zhang et al. 2008). This characteristic of umami taste is widely used in the food industry to enhance palatability of foods. Umami taste plays a key role in the flavor profile of many foods, and these geographically and perceptually diverse foods are formed from similar underlying flavor active compounds. Examples include meat stocks and tomato products in Europe, Italian Parmesan, soy sauce and fish sauces from South East Asia and dashi and a range of other seaweed derived products from Japan (Fuke and Shimizu 1993). We are all exposed to umami even from a very young age because free glutamate is present in abundance in breast milk (Mastorakou et al. 2019).

Although the taste of umami compounds in simple aqueous solution is not especially pleasant, in savory food stuffs addition of umami compounds commonly results in increased ratings of flavor and acceptability (Barylko-Pikielna and Kostyra 2007). The prototypical umami tantant
MSG has also been described as contributing to perceived ‘mouth-fullness’ and ‘thickness’ of foods (Yamaguchi and Ninomiya 2000). There are wide ranging examples of the positive flavor properties of umami in complex savory foods, including vegetable, fish and meat soups and sausages amongst others (Barylko-Pikielna and Kostyra 2007; Gould et al. 2008; McGough et al. 2012; Sinesio et al. 2010; Yeomans et al. 2008). In addition to its flavor enhancement properties, studies exploring taste-taste interactions have demonstrated the ability of umami to modulate perception of other basic tastes, including a variable effect on sweetness but importantly suppression of bitterness and enhancement of other basic tastes, including a variable effect on sweetness (Keast and Breslin 2003; Wang, Zhou, and Liu 2020). These observations extend to more complex flavor mixes and food matrices (Fuke and Ueda 1996). For example, umami compounds in crisps were shown to enhance saltiness and potato chip flavor (Zhang and Peterson 2018) and even enable reduction in sodium content by up to 30% with no loss of palatability (Kongstad and Peterson 2018) or possibly, due to its ability to reduce sodium (Yamaguchi & Takahashi, 2006; Hayabuchi et al. 2017). However, the exact mechanism(s) of umami perception is not fully explained. Understanding the receptors and transduction pathways that mediate umami taste, and the mechanisms of peripheral and central perception in humans is the first step to enable the development of nutritious healthy food products. A number of review articles have been published on umami perception since its discovery, however most of these publications have focused on the molecular mechanism of umami perception (Jyotaki, Shigemura, and Ninomiya 2009; Zhang et al. 2019). A limited number of articles discussed the central mechanism of umami taste perception in the human brain (Rolls 2009), and variations in umami sensitivity (Shigemura, Shirosaki, Ohkuri, et al. 2009). This review article bridges current knowledge and new findings obtained from molecular-genetic, electrophysiological and human brain imaging studies regarding the peripheral and central mechanisms for detecting umami taste.

Overview of the physiology of taste perception

Taste or gustatory sensation is detected by taste receptors cells (TRCs), which are mainly located in taste buds throughout the oral cavity including the tongue, palate and throat. Taste buds are onion-like structures, primarily located on the tongue papillae, each containing 50–100 taste cells. When food or drink is consumed, a small proportion of the food material dissolves in saliva, diffuses into the taste pore and interacts with the surface of the taste cells. Each of the five taste qualities have an identified receptor/ion channel on the surface of the cells and a unique transduction mechanism. Salt and sour tastants permeate the cell membrane via voltage-gated ion channels, whilst sweet, umami and bitter tastants bind to G-protein coupled receptors (GPCRs) on the cell membrane (Roper and Chaudhari 2017). However, some studies suggest there is no strict one-cell-one taste receptor expression rule, since some taste receptor cells respond to two or more (Tomchik et al. 2007) different tastes. Thus, taste is represented by a complex combinatorial code across specifically and broadly-tuned peripheral neurons ‘across-fiber model’ (Tomchik et al. 2007). Others suggest that receptor cells and peripheral neurons are tuned to specific taste modalities ‘labelled-line model’ (Chandrashekar et al. 2006).

The interactions between TRCs and tastants cause electrical potential changes in the taste cells, which triggers a cascade of electrical impulses that are transmitted through the peripheral afferent nerve fibers (gustatory afferent axons) to the primary gustatory cortex in the brain. The electrical signals generated in the TRCs transmit to the brain through thin elongated fibers that wind themselves around TRCs. These signals are conveyed by three cranial nerves: the chorda tympani branch of facial nerve (cranial nerve VII), innervating the anterior two third of the tongue, the glossopharyngeal nerve (cranial nerve IX), innervating the posterior part of the tongue, and the vagus nerve (cranial nerve X), innervating the epiglottis and larynx. These nerves transmit information about the identity and quantity of the chemical nature of the tastants, as well as somatosensory information, in association with the trigeminal nerve (cranial nerve V).

The cranial nerves enter the brainstem and converge at the rostral portion of the nucleus of the solitary tract (NST) in the medulla. In primates, taste signals project from the NST to the cortex via two main pathways. Fig. 1 illustrates a schematic diagram of the peripheral and central taste processing and pathways. In the first taste pathway, the signal projects directly to the ventral posterior medial (VPM) nucleus of the thalamus, which contains a topographic representation of the oral cavity (Cerkevich, Qi, and Kaas 2013; Simon et al. 2006). Signals then pass from the VPM and terminate in the dorsal part of the anterior insula in the frontal operculum, which is the primary gustatory cortex (PGC), and in the somatosensory cortex in the post-central gyrus. The insula is thought to be involved in conscious taste perception, including identifying taste qualities (Yamamoto, Matsuo, and Kawamura 1980). The PGC then projects to the amygdala and to the caudolateral orbitofrontal cortex (OFC), the secondary taste cortex. The OFC has been shown from human neuroimaging studies to act as a higher order taste center involved in encoding the reward values of the taste stimulus (Kringelbach, O’Doherty, Rolls, & Andrews, 2003). It is believed to be modulated by motivational state, responding to hunger and not to satiety (Van der Laan et al. 2011). The amygdala also plays a major role in pleasure and reward of food stimuli. The second taste pathway is from the NST to the nucleus of the pons in the cerebellum which then projects to the amygdala and lateral hypothalamus. The lateral hypothalamus has an important role in the regulation...
of homeostasis, ingestive behavior, reward and motivation (Fu et al. 2019). The amygdala projects to other reward areas including the striatum and the anterior cingulate cortex, which plays a role in the hedonic response to emotions induced by taste (De Araujo, Kringelbach, Rolls, and Hobden 2003).

Mechanisms of umami taste perception

Molecular mechanisms of umami perception

TRCs are divided into three anatomically distinct types, Type I (Roper and Chaudhari 2017), Type II (Clapp et al. 2004; DeFazio et al. 2006), and Type III (Chaudhari 2014). Evidence from molecular biology studies revealed that umami compounds are detected in the oral cavity by receptors expressed in Type II (Clapp et al. 2004; DeFazio et al. 2006) taste cells through multiple receptors. Some of these receptors are not yet completely characterized, however the heterodimer taste receptor Type 1, member 1 (T1R1) and 3 (T1R3) is the best understood of the umami taste receptors (Nelson et al. 2001; Li et al. 2002). T1R1 is expressed selectively in fungiform papillae, located on the anterior two third of the tongue, and rarely detected in circumvallate papillae on the posterior part of the tongue (Nelson et al. 2001; Hoon et al. 1999; Kitagawa et al. 2001), whereas T1R3 is expressed in both fungiform and circumvallate papillae (Nelson et al. 2001; Kitagawa et al. 2001). Since T1R1 and T1R3 are co-expressed in taste bud cells of fungiform papillae (Nelson et al. 2001), umami receptors (T1R1/T1R3) seem to function in the anterior part of the tongue. Zhao et al. (2003) demonstrated that umami taste is strictly dependent on T1R receptors. In this study, knockout of either T1R1 or T1R3 completely eliminated the responses to oral glutamate, which suggests that the heterodimer is the only umami receptor (Zhao et al. 2003). However, in another study, knockout of T1R3 only eliminated the nucleotide potentiation of glutamate taste responses, with little effect on responses to glutamate alone (Delay et al. 2006; Kusuhara et al. 2013; Damak et al. 2003), while other studies demonstrated that T1R1 knockout mice are fully responsive to umami stimuli (Delay et al. 2006; Damak et al. 2003). Although the reason for these discrepancies is not known, these data indicate that other receptors and mechanisms are likely involved in umami taste perception. A recent study (Choudhuri, Delay, and Delay 2015) used calcium imaging of isolated taste cells in T1R3 receptor knockout mice confirmed the existence of multiple glutamate receptors and also suggested that some taste cells can even respond to nucleotides in the absence of glutamate. However, it is important to note that in isolated taste cells stimuli are not restricted to the apical membrane as they are in in-vivo settings, and this may account for some of the differences observed.

The metabotropic glutamate receptors (mGluR4 and mGluR1) (Nicholson, Lindon, and Holmes 1999; Lopez et al. 2007; Nelson et al. 2002; Neugebauer 2008; Nicholson et al. 2017; Chaudhari, Landin, and Roper 2000; Thomsen 1997) are the likely candidates in umami detection, as both of these receptors are located in taste buds of circumvallate and foliate papillae of the posterior part of the tongue in mice (Chaudhari et al. 1996) and have been shown to be activated by glutamate (Chaudhari, Landin, and Roper 2000; San Gabriel et al. 2009). In addition, taste nerve recordings from mGluR4 knockout mice (Chandrashekar et al. 2006) revealed decreased responses to glutamate, confirming that a fraction of the afferent nerve response to glutamate in wild-type mice is attributable to mGluR4 (Yasumatsu et al. 2015). To our knowledge, knockout of mGluR1 has not been tested in taste experiments, however, a selective antagonist to mGluR1, 1-aminooindan-1,5-dicarboxylic acid, reduced responses to glutamate in chorda tympani and
glossopharyngeal nerve recordings (Yasumatsu et al. 2015), supporting the involvement of mGluR1 receptors in umami perception.

**Signaling pathway of umami taste transduction**

Evidence from immunocytochemical and molecular studies demonstrate that the stimulation of umami receptor (T1R1 + T1R3) by umami stimuli activated G-protein subunits, Gz, which leads to the release of Gβγ subunits, modulating cyclic adenosine monophosphate (cAMP) levels (Nelson et al. 2001; Li et al. 2002). The Gβγ subunit stimulates the phospholipase Cβ2 (PLCβ2) pathway (Iwata, Yoshida, and Ninomiya 2014; Kinnamon 2009). This transduction part of Gβγ in the pathway appears to be dominant and necessary for umami transduction (Damak et al. 2003). This in turn produces inositol-1,4,5-triphosphate (IP3) and diacylglycerol (DAG). While the function of DAG remains unclear, IP3 binds to the Ttype III IP3 receptor (IP3R3) inducing Ca2+ release from the Ca2+ stores (Liu et al. 2008; O-Uchi et al. 2018). The increase in Ca2+ activates the transient receptor potential of TRPM5 (transient receptor potential cation channel subfamily M member 5) (Clapham 2003; Dutta Banik et al. 2018; Eddy et al. 2012; Cao et al. 2013; Autzen et al. 2018; Yin et al. 2018) which leads to the depolarization of the taste cell. Finally, the taste cell evokes action potentials via voltage-gated Na+ channels and releases ATP which transforms chemical signals into electrical signals to activate taste nerve fibers as illustrated in Fig. 2 (Cisneros-Mejorado et al. 2015; Huang et al. 2007; Kinnamon 2013).

Evidence of the involvement of this pathway in umami taste transduction comes from several factors, including the reduced or eliminated nerve responses to umami taste in PLCβ2 (Zhang et al. 2003), IP3R3 (Hisatsune et al. 2007), and TRPM5 (Damak et al. 2006) knockout mice in a similar manner to that of the T1R3 knockout (Damak et al. 2003). In addition, the pharmacological inhibitors of PLCβ2 and Ca2+ ATPase, which maintain intracellular Ca2+ stores, eliminate responses to glutamate and nucleotides applied selectively to the taste pore in Ca2+ imaging studies of a lingual slice preparation (Maruyama et al. 2006). However, it should be noted that multiple signaling pathways have been suggested to be involved in umami taste perception, with possible additional undiscovered receptors, and/or interactions among the receptors. In a recent study, Dutta Banik et al. (2020) illustrated a new population of taste cells that can detect different taste qualities including umami (Dutta Banik et al. 2020). These broadly responsive cells are a subset of Type III taste cells and they use the PLCβ3 signaling pathway to respond to umami stimuli (Dutta Banik et al. 2020).
were greatly reduced, and unaffected in other fibers (M1, M2, E- and N-types) compared with wild-type mice (Yasumatsu et al. 2012). In addition, neural evidence also shows that whole CT responses to glutamate stimuli in T1R3 and/or T1R1 knockout mice elicit relatively robust signals in CT, however for the synergistic amplification of the glutamate response by IMP these responses are dramatically attenuated, if not abolished. These findings demonstrate and support the importance of T1R3 and T1R1 for the synergistic effect between glutamate (MPG and MSG) and IMP in the CT nerve. In a recent electrophysiological study (Kalyanasundar et al. 2020), single-unit taste responses from the nucleus of the solitary tract (NST) of the medulla were recorded in T1R double-knockout mice lacking functional T1R1 + T1R3 receptors and its wild-type background strains. The results revealed a major suppression of central responses to synergistic action of MSG and IMP in mice lacking T1R1 + T1R3, supporting previous evidence that the T1R family of receptors plays a crucial role in transducing taste stimuli including umami. However, in line with previous studies (Yasumatsu et al. 2015; Yasumatsu et al. 2012), a degree of responsiveness to umami tastants remained, suggesting that other receptors also convey umami taste information.

Strong evidence supports the involvement of mGluR1 and mGluR4 receptors in umami perception (Yasumatsu et al. 2015; Yasumatsu et al. 2012). These receptors are located in the posterior area on the tongue. Interestingly, the reduction in glutamate response observed in CT nerve in T1R3 and/or T1R1 knockout mice did not occur in the GL nerve, suggesting the involvement of mGluRs receptors. In support of this, mGluR4 knockout mice displayed reduced responses to umami stimuli (MPG) in both the CT and GL nerves (Yasumatsu et al. 2015; Yasumatsu et al. 2012). Furthermore, findings from Yasumatsu et al. (2012) and (2015) suggest that mGluR1 and mGluR4 may function as an umami receptor in M1- and M2-type taste fibers, respectively.

The existence of multiple umami receptors seems to reflect different functions in the detection and preference of umami compounds. In T1R3 and T1R1 knockout mice, glutamate taste discrimination appeared to be unaffected (Delay et al. 2006; Kusuhara et al. 2013), indicating that T1R1/T1R3 does not influence umami taste discrimination, and this is more likely dependent on mGluR1 and mGluR4 receptors. However, there is no direct evidence, to date, elucidating the role of mGluRs pathway in discrimination of umami taste, and further studies are required to support this hypothesis. In contrast, preference to umami compounds was greatly diminished in T1R3 knockout mice (Damak et al. 2003; Zhao et al. 2003), indicating that T1R3-dependent umami receptors (T1R1/T1R3) have a critical role in the behavioral preference for umami compounds, most likely driven via S1-type taste fibers. Interestingly, in double T1R1/T1R3 knockout mice, appetitive motivation responses were unaltered (Blonde and Spector 2017) even at higher concentrations of MSG (Blonde, Travers, and Spector 2018), hence this could be driven by other receptors and neural pathways.

**Representation of umami taste in the human cortex (Central coding)**

Over the past two decades, advances in neuroimaging techniques, particularly functional magnetic resonance imaging (fMRI), have provided valuable insight into central food-related pathways in the human brain. In a typical taste fMRI study, taste stimuli are delivered for a period of time followed by a rest period, known as “block design”, and the neuronal representation or functional mapping of brain activity to food stimuli is indirectly measured through the hemodynamic changes associated with neural activation, typically using blood oxygenated level dependent (BOLD) technique or BOLD contrast (Ogawa et al. 1990).

Brain mechanisms underlying the perception of oral umami taste have been addressed in human fMRI studies. Umami compounds such as MSG and IMP, and their mix, have been shown to activate the primary taste cortex, the insula-opercular taste cortex, and the secondary taste cortex, the OFC (De Araujo, Kringelbach, Rolls, and Hobden 2003) (Fig. 3A), suggesting that umami taste is similarly recognized as the other basic taste qualities. Supporting this theory is evidence from single-neuron recordings in non-
human primates (macaques), which have shown that umami tastants activate neurons in the primary and secondary taste cortices (Baylis and Rolls 1991; Rolls 2000; Rolls et al. 1996). Moreover, in a number of fMRI studies the cortical representation of umami and salt showed overlap in a number of food-related brain areas, including the mid insula (a taste/somatosensory area) (Han et al. 2018; Nakamura et al. 2011), frontal operculum (taste processing area) and pre-and post-central gyri as well as rolandic operculum (oral somatosensory areas) (Han et al. 2018). These findings suggest that umami and salty taste perception share a common processing system. However, it is important to note that these studies were acquired with relatively low spatial resolution, and recent imaging studies in rodents have identified a gustotopic organization "tastemap" in the primary taste cortex (insula) with distinct regions/neurons in the insula selectively responsive to each of the five basic tastes (Chen et al. 2011). Furthermore, in a recent neuroimaging study, the human cortex showed that each taste quality have spatially different but overlapping cortical activation, with umami taste overlapping with salt and sour taste in the anterior part of the insula, and sweet and bitter tastes overlapping in the posterior part of the insula (Prinster et al. 2017). However, these differences could also be due to variation in tastant concentration and/or perceived pleasantness, or differences in individuals taste phenotype or genotype. More work is needed to underpin the neuronal representation of umami taste in human brain.

One of the main characteristics of umami taste is the synergistic effect when IMP is added to umami compounds (MSG). De Araujo et al. (2003) showed that the brain's response to MSG/IMP mixture was significantly higher in the lateral OFC compared to the sum of the MSG alone and IMP alone, a supra-linear response as illustrated in Fig. 3B (De Araujo, Kringelbach, Rolls, and Hobden 2003). The other significant feature of umami taste is the ability to enhance the palatability of foods. The hedonic representation of umami taste has been shown to be encoded in the dorsal part of the anterior cingulate cortex, distinct from the hedonic representation to the sweet taste of glucose samples, which was found to activate the pregenual cingulate cortex (De Araujo, Kringelbach, Rolls, and Hobden 2003).

Individual differences to umami perception

Behavioral and psychophysical studies demonstrate human variability in the perception of umami taste, with some individuals noted as not being able to taste umami (Puputti et al. 2018). Lugaz, Pillias, and Faurion (2002) reported that subjects could be classified into 'tasters', who perceived MSG at a relatively low concentrations, 'hypo-tasters', who perceived MSG at a relatively high concentrations, and 'non-tasters' who were unable to perceive MSG (Lugaz, Pillias, and Faurion 2002). In this study, 81% of subjects were classified as tasters, 10% hypo-tasters, and 3.5% non-tasters, with taste thresholds of umami differing ≈ 5-fold between tasters and hypo-tasters. In a more recent study, Singh, Schuster, and Seo (2010) demonstrated that 3.9% of European adult participants were unable to distinguish MSG versus NaCl, suggesting a reduced ability to taste umami (Singh, Schuster, and Seo 2010). Of the cohort studied, 3.2% of the German participants and 4.6% of the Norwegian participants were potential non-tasters of MSG (Singh, Schuster, and Seo 2010). Interestingly, the distribution of tasters, hypo-tasters and non-tasters was significantly different between Norwegian and German populations. Specifically, the prevalence of hypo-tasters (versus taster) was significantly higher in the Norwegian population than in the German population, whilst no significant difference between the two populations was obtained in terms of the non-taster prevalence.

The variability in human umami taste perception is still poorly understood, however a genetic mechanism is possibly behind these variations, with links to the heterodimeric receptor T1R1 and T1R3 (Raliou et al. 2009; Shi gemura, Shir osaki, Sanematsu, et al. 2009). Different studies conducted a comprehensive evaluation of single-nucleotide polymorphisms (SNPs) and haplotypes in human TASI R1 and TASI R3 genes and revealed several SNPs within the extracellular domain of T1R1 and T1R3 (Chen et al. 2009; Flaherty and Lim 2017; Puputti et al. 2018; Satoh-Kuriwada et al. 2014; Shi gemura, Shir osaki, Sanematsu, et al. 2009; Simmons and Estes 2008; Singh, Schuster, and Seo 2010). Raliou et al. (2009) found variations in genes T1AS R1 and TASI R3 in human fungiform papillae and suggested that these receptor variations contributed to the individual differences in glutamate sensitivity in the studied population (European Caucasian) (Raliou et al. 2009). In line with this study, Shigemura et al. (2009) demonstrated a strong correlation between the recognition thresholds of umami taste perception and genetic variations amongst a Japanese population (Shigemura, Shir osaki, Sanematsu, et al. 2009). These results support the association between inter-population differences in umami perception and genotype. Interestingly, Kim et al. (2006), demonstrated that the frequencies in SNPs show little variation between Asian, African, European and native American populations (Kim et al. 2006). These results suggest that variations observed in umami perception are more likely to be due to individual differences rather than population differences. However, association of umami taste phenotypes with variations in umami taste receptor genes remain unclear at this point, and further studies are needed. In addition, other factors have been shown to have an impact on umami perception including age and body weight (Puputti et al. 2019), or previous exposure/familiarity to umami taste (Han et al. 2018).

The effect of individual variations in umami taste perception on the brain's response to umami taste was addressed in a recent fMRI study (Han et al. 2018). Umami 'high tasters' (high umami identification ability) showed larger activation in the primary gustatory cortex (frontal operculum), the OFC and postcentral gyrus compared to low umami tasters in response to MSG. Interestingly, umami 'low tasters' showed stronger activation in the posterior insula, thalamus and hippocampus indicating that attention and association/memory related brain structures play a
significant role in the perception of umami taste. This study also demonstrated that umami ‘high tasters’ have heightened brain responses to salt taste compared with umami ‘low tasters’, further supporting the association between salt and umami perception.

Concluding remarks and future perspectives

Accumulating evidence demonstrates that several receptors are involved in umami detection in taste buds on the tongue. These receptors include 2 glutamate-selective G protein–coupled receptors, the taste bud–expressed heterodimer T1R1 + T1R3 and mGluR4 and mGluR1. However, umami taste detection may involve additional taste receptors expressed in different subsets of taste cells yet undiscovered. The receptor diversity of umami detection may underlie the complex perception of umami, with different mixtures of umami substances (amino acids, peptides, and nucleotides) yielding distinct taste qualities. Moreover, detection of umami taste has been shown to vary across individuals in a number of studies, with a strong link to genetic variations. Functional MRI techniques have revolutionized the research in taste perception and revealed the cortical representation to umami taste, as highlighted in this review. However, there is still much research needed to understand the neural underpinnings of taste perception in the human brain, and the effect of individual variability in taste perception.

Umami taste is characterized by a synergistic effect when combined with 5′-nucleotide monophosphates, and this unique characteristic has been utilized to increase the palatability of food. However, umami taste is not only known to increase palatability and appetite but has also been demonstrated to increase satiety (Masic and Yeomans 2014), and hence could be used to control food intake. The regulation of hunger and satiety is controlled by the neuronal communication between the brain and the gut (brain-gut axis). The focus of this review is to understand the perception of oral umami, however, it is important to note that an increasing number of studies have detected umami taste receptor in non-taste tissues, including the gastrointestinal (GI) tract (Crowe et al. 2020; Dyer et al. 2005; Jang et al. 2007; Nunez-Salces et al. 2020). The interactions between the brain and gut are not yet fully understood, and there are limited studies in human and animals investigating the signaling between GI taste receptors and the brain. Further studies should be conducted to elucidate these interactions and the possible role of umami taste to increase satiation and assess brain-satiety areas. Understanding these interactions will pave the way to develop healthy food and tackle obesity. In an exploratory/pilot human fMRI study, Meyer-Gerspach et al. (2016) assessed the brain responses of GI taste receptors. In this study an intra-gastric administration of the five basic tastes was applied. Interestingly MSG activated several brain regions including the primary taste cortex (Meyer-Gerspach et al. 2016). This effect was more pronounced for bitter and umami than sweet, salt and sour tastes.

To conclude, whilst the current scientific knowledge around umami taste perception has stepped forwards remarkably in recent years, it is clear from this review that more research is required, including a more holistic overview of not only oral umami taste perception, but also the wider “whole body” signaling mechanisms, to explore the interactions between the mouth, brain and gut in response to umami perception and ingestion.

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