umami intensity. To ascertain the mechanism by which MSG increased perceived saltiness of NaCl solutions (see exp 1), NaCl-MSG mixtures were compared to NaCl only solutions matched for total Na+ concentration using a 2-AFC methodology. When test solutions (MSG+NaCl and NaCl only) contained eqimolar concentrations of Na, observed saltiness enhancement by MSG was eliminated. This suggests that Na+ from MSG is responsible for the perceived saltiness enhancement observed in exp 1. Finally, advances in flavor chemistry have identified Na-free compounds that evoke umami sensations (Na-FUs). In exp 4 we investigated if perceived salt and umami intensities change if NaCl levels are held constant and a Na-FU compound is added. Similar to exp 1, adding Na-FU to NaCl increased perceived umami sensation but only marginally increased perceived saltiness at low concentration.

#P305 Poster session VII: Chemosensory Psychophysics II

Perceptual variation in umami taste and polymorphisms in *TAS1R* taste receptor genes

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The TAS1R1 and TAS1R3 G-protein coupled receptors (GPCRs) are believed to function in combination as a heteromeric glutamate taste receptor in humans. In this study, we first characterized the general sensitivity to glutamate in a sample population of 242 subjects using discrimination task and extensively tested a subset of ten subjects at extremes of sensitivity. We followed these experiments by sequencing the coding regions of the genomic TAS1R1 and TAS1R3 genes for a separate set of 87 individuals who were tested repeatedly with monopotassium glutamate solutions and asked to rate umami taste on a general Labeled Magnitude Scale. We established that there is considerable variation in umami taste perception. A subset of subjects at the extremes of sensitivity was subsequently studied in a battery of psychophysical tests validated the observation. In genetic sequencing experiment we revealed that TAS1R3, the shared subunit of the TAS1R sweet and umami taste heteromer receptors, contained more variations than did TAS1R1, contrary to earlier reports. We identified one rare nonsynonymous single nucleotide polymorphism (SNP) and four synonymous SNPs in TAS1R3 that have not been previously reported. Statistical analysis showed that the rare 'T' allele of SNP R757C in TAS1R3 led to a doubling of umami ratings of 25 mM MPG. Other suggestive SNPs of TAS1R3 include the 'A' allele of A5T and the 'A' allele of R247H, which both resulted in an almost doubling of umami ratings of 200 mM MPG. In conclusion, there is reliable and valid variation in human umami taste from L-glutamate. Variations in perception of umami taste correlated with variations in the human TAS1R3 gene. Thus, this receptor is likely contributing to human umami taste perception.

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Monosodium Glutamate Taste Recognition Thresholds are not Affected by Modulation of Serotonin or Noradrenaline Levels in Healthy Humans

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This study was designed to determine the effect of altering serotonin and noradrenaline levels on monosodium glutamate (MSG) taste recognition thresholds in humans. We have previously shown that manipulation of these neurotransmitters lowers taste thresholds of specific taste modalities (Heath et al 2006). A preliminary tongue map for MSG taste was generated in 34 healthy adults (6 male, 28 female, age range 19-71) to determine the most sensitive area of the tongue for further testing. In a further 26 adults (13 male, 13 female, age range 19-48) a series of MSG and sodium chloride solutions were presented either to the back or the tip of the tongue in a pseudorandom order. Recognition thresholds were calculated from psychophysical function curves before and 2 hours after a single acute dose of either paroxetine (serotonin selective reuptake inhibitor), reboxetine (noradrenaline reuptake inhibitor), caffeine (active placebo) or placebo (lactose) in a double blind cross-over design. MSG taste recognition thresholds were significantly lower at the back of the tongue compared to the tip (back 14±3mM, tip, 60±13mM, $p<0.01$). Comparison of thresholds at either the back or the tip of the tongue showed MSG recognition thresholds were not affected by any drug, or by placebo, in either region. Sodium chloride thresholds were also unaffected by any intervention, as previously shown. Thus, pharmacological modulation of serotonin or noradrenaline levels in humans has no effect on glutamate taste. These findings, together with our previous study showing that the same interventions modulate bitter and sweet taste, support a modality specific neuromodulatory role for serotonin and noradrenaline in human taste perception. Studies were approved by Bath LREC, UK.

#P307 Poster session VII: Chemosensory Psychophysics II

A receptor focused analysis of experience induced changes in glucose and monosodium glutamate (MSG) taste sensitivity

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Brief treatment with fructose induces increased taste discrimination ability (sensitivity) for glucose, and MSG treatment increases MSG sensitivity in humans. Both induction effects reverse after treatment is stopped (Kobayashi & Kennedy, 2002; Kobayashi *et al.*, 2006, Gonzalez *et al.*, 2008). Hamster chorda tympani and *Drosophila* receptor cell neurophysiological data suggest a peripheral mechanism (Berteretche et al., 2005, Gonzalez et al., 2009). We conducted further human studies with various sweeteners and umami stimuli. The tests were designed according to current knowledge of the binding of stimuli to the sweet and umami receptors, to test hypotheses about the receptors' function in the induction. All sweetener concentrations were isosweet, and the umami concentrations isointense, to the original fructose treatment and glucose or MSG test concentrations, as determined by a gLMS scale and magnitude matching. Treatment and testing paradigms were as in Gonzalez *et*