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▶ **To cite this version:**

Minghai Fu, Mary-Louise Manchadi, Nadia de Jager, David Val-Laillet, Sylvie Guerin, et al.. Bitter compounds delayed gastric emptying and induced intestinal smooth muscle relaxation in a pig model. 17. International Symposium on Olfaction and Taste (ISOT2016), Jun 2016, Yokohama, Japan. Chemical Senses, 41 (9), 2016, Chemical Senses. hal-01413522

HAL Id: hal-01413522
https://hal.science/hal-01413522

Submitted on 3 Jun 2020

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P1-111 Bitter compounds delayed gastric emptying and induced intestinal smooth muscle relaxation in a pig model

Minghai Fu[1], Mary-Louise Manchadi[1], Nadia De Jager[1], David Val-Laillet[2], Sylvie Guerin[2], Eugeni Roura[1]

[1]The University of Queensland, Brisbane, Australia, [2]INRA, St Gilles, France

Bitter compounds induced innate aversion which was reflected in loss of appetite and decreased fat deposition in a pig model reported by our group. However, the mechanism remains unclear. We hypothesized that bitter compounds would reduce gastric emptying and slow down intestinal peristalsis in the pig. Based on previous in-vivo trial and known molecular mechanisms, caffeine and quinine were the two bitter compounds selected. Both compounds are known agonists of human bitter taste receptors (hT2R) with high homology with the porcine T2 Rs: Tas2r4, Tas2r7, Tas2r10, Tas2r20 and Tas2r39. Real-time PCR was used to study the expression of porcine bitter taste receptor genes in the stomach and duodenum that related to the tested bitter compounds. The results showed high expression both in the stomach and duodenum and that the expression level was most significant in the epithelium relative to the smooth muscle layer. The non-invasive scintigraphy imaging data showed that caffeine and quinine mixed feed significantly ($P<0.01$ and $P<0.05$ respectively) decreased gastric emptying compared to control pigs. In addition, an ex-vivo intestinal smooth muscle contraction study illustrated that the two bitter compounds induced ($P<0.001$) dose-response relaxation in the duodenum. To conclude, dietary bitter compounds significantly reduced gastric emptying and gut motility. These effects may partially explain previous results showing a reduction in appetite and fat deposition in a pig model. The potential of bitter compounds to help prevent obesity in humans is worth further investigation.

P1-112 Expression of umami taste-related genes in the tongue: A pilot study for genetic taste diagnosis

Noriaki Shoji[1], Shizuko Satoh-Kuriwada[1], Masahiro Tsuchiya[1,2], Hisayuki Uneyama[3], Misako Kawai[3], Takashi Sasano[1]

[1]Division of Oral Diagnosis, Tohoku University Graduate School of Dentistry, Sendai, Japan, [2]Tohoku Fukushi University, Sendai, Japan, [3]Institute for Innovation, Ajinomoto Company Inc., Kawasaki, Tokyo, Japan

Umami taste is important for maintaining not only good health but also the quality of life. A wide variety of test is used to discriminate between normal and abnormal umami taste sensation. However, it is not necessarily useful for patients who cannot express their sensation to the tastant, such as patients with dementia, because of the subjective nature of the test. Against this background, we evaluated the expression of the umami receptor genes in the tongue to develop an objective umami taste test. Tissue samples were collected from healthy volunteers by scraping the foliate papillae of the tongue. Immunocytochemistry staining of gustducin, a taste-cell-specific G protein, and gene expression analysis by real-time polymerase chain reaction of *β-actin*, gustducin (*GNAT3*), and umami receptors (*T1R1*, *T1R3*, and *mGluR1*) were performed. Further, amplified PCR products were analyzed by DNA sequencing to confirm the expected sequences. Changes in umami receptor expression following application of umami substances onto the tongue were analysed. Results showed: (i) Gustducin-positive cells were observed in the samples, indicating the presence of taste cells; (ii) Gene expression of *β-actin*, *GNAT3*, *T1R1*, and *T1R3* was detected in all seven samples tested, while that of mGluR1 was detected in four samples; (iii) Sequence analysis by NCBI Blast showed that each polymerase chain reaction product had a 99% rate of identification of its targeted sequence, indicating that the designed specific primers enabled to amplify the targeted gene expression without mispriming and/or primer-dimer artefacts; (iv) Stimulation of the tongue by monosodium glutamate significantly upregulated the gene expression levels of *T1R1* and *T1R3*, indicating that this method can detect alterations in umami-related gene expression. In conclusion, evaluation of the expression of the umami receptor genes *T1R1* and *T1R3* in the tongue could be clinically useful for objective genetic diagnosis of umami taste disorders.

P1-113 The anatomy of mammalian sweet taste receptors

Jean-Baptiste Cheron, Jerome Golebiowski, Serge Antonczak, Sebastien Fiorucci

Institut de Chimie de Nice, Faculte des Sciences, Universite de Nice-Sophia Antipolis, Nice, France

All sweet-tasting compounds are detected by a single G-protein coupled receptor (GPCR), the heterodimer T1R 2-T1R3, for which no experimental structure is available. The sweet taste receptor is a class C GPCR, and the recently published crystallographic structures of metabotropic glutamate receptor (mGluR) 1 and 5 provide a significant step forward for understanding structure-function relationships within this family. In this article, we critically align the sequences of class C GPCRs that recapitulate available structural and site-directed