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Acute effect of oral sensation of sweetness on celiac artery blood flow and gastric myoelectrical activity in humans

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ABSTRACT

Little is known about the effect of sweet taste stimulus on gastrointestinal motility and splanchnic blood flow. We examined whether gastric myoelectrical activity and/or celiac artery blood flow (CABF), which perfuses the stomach, are increased following an oral sensation of sweetness. After overnight fasting, 11 subjects rested for 5 min and sipped, but not swallowed, one of four solutions for 1 min. The fluid was then spat out, and subjects remained at rest for a further 10 min. Fluids were approximately 15 ml of three glucose solutions (4, 16, or 48%) or distilled water. Subjects completed trials with all four solutions in a randomized order. During each trial, gastric myoelectrical activity and CABF were continuously measured using electrogastrography and pulsed Doppler ultrasonography, respectively. None of the four solutions affected gastric myoelectrical activity. CABF was significantly increased after oral stimuli by all three glucose solutions, but not by water. There were no significant differences in the increments in CABF among the three glucose solutions. These results suggest that a sweet taste stimulus above a certain level of intensity acutely increases CABF during cephalic phase, without augmentation of gastric myoelectrical activity.

1. Introduction

The physiological responses to food ingestion occur before the food reaches the stomach. For example, secretion of gastric acids and gastrin as well as saliva is elicited by sight, smell, taste, and image of foods (Feldman and Richardson, 1986). These prior digestive activities triggered by cephalic-vagal stimulation are called cephalic phase responses (CPRs) (Smeets et al., 2010), which play a role in preparing the gastrointestinal system for digestion and absorption of nutrients. There has been considerable research on CPRs with a focus on secretion of digestive juices and gut hormones (Zafra et al., 2006), but CPRs also include non-secretory ones such as gastrointestinal motility (Chen et al., 1996; Katschinski et al., 1992; Manabe et al., 2011; Rogers et al., 1993; Stern et al., 1989) and peripheral circulation (Buss et al., 2012; Hasegawa et al., 2013; Kashima and Hayashi, 2013; Someya and Hayashi, 2008).

After eating, splanchnic blood flow (BF) increases for digestion and absorption. Someya and Hayashi (2008) reported that the celiac artery blood flow (CABF) supplying the upper abdominal organs such as the stomach was increased by chewing a chocolate-flavored cereal bar (without swallowing), but not by chewing tasteless paraffin wax. While these findings suggest that the CPR of CABF can be induced by the taste of foods, it remains unknown what kind of taste triggers such response and what mechanisms mediate it. Sweet taste is a vital sense for animals, being utilized to predict caloric contents of foods, and has been reported to elicit CPRs by itself (Dušková et al., 2013; Grill et al., 1984; Ikuno and Sakaguchi, 1990; Just et al., 2008; Tonosaki et al., 2007). It has also been reported that gastric myoelectrical activity (GMA), which reflects gastric motility, increases after eating and is enhanced during the cephalic phase (Stern et al., 1989). When GMA is enhanced, CABF is increased owing to the exaggerated contractive activity of the smooth muscles. We therefore hypothesized that a quick increase in CABF may be induced by sweet taste, and the response may be accompanied by an enhancement of gastric motility.

Moreover, a postprandial increment in splanchnic BF is dependent on meal size (Sidery and Macdonald, 1994), but it is unknown whether the intensity of taste stimuli affects the amount of change in CABF. Stronger stimuli could possibly induce a greater increase in CABF. Therefore, the aim of this study was to examine whether CABF and GMA acutely increase as initial CPRs following an oral sensation of sweet taste by different concentrations of glucose. We hypothesized that

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sweet taste in the oral cavity could trigger dose-related increases in CABF and GMA.

2. Materials and methods

2.1. Subjects

Eleven healthy young Japanese subjects (5 males and 6 females, aged 21 ± 2 years, BMI 20.4 ± 2.1 kg/m^2; mean ± SD) participated in the experiment. They had no food allergies, no gastrointestinal symptoms, no history of major diseases such as cardiovascular disease, and did not take any medication. They were informed about the study aims and signed a written consent form. The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of the University. Prior to the experimental session, subjects were tested for their sweet taste threshold for glucose to determine if they had normal sweet taste sensitivity by a whole-mouth recognition method.

2.2. Taste solutions for orosensory stimulation

The taste solutions for the orosensory stimuli consisted of different sweetness intensities with glucose concentrations of 4, 16 and 48% (w/v), and distilled water. Glucose solutions were prepared by dissolving D-glucose (Nakalai Tesque Inc., Kyoto, Japan) in distilled water. All solutions were provided to participants at 24.9 ± 0.3 °C, and the sip volume for a stimulus was 15.1 ± 4.3 ml (mean ± SD).

2.3. Protocol

Subjects reported to the laboratory at 08:00–09:00 after overnight fasting for at least 10 h. In addition, subjects refrained from strenuous exercise and ingestion of alcohol and caffeine-rich beverages for at least 1 day before the experimental day. Subjects were kept in a semi-supine position and were asked not to swallow any solution or saliva during each trial. After baseline values were recorded for 5 min, subjects sipped one of the taste solutions and held it in their mouth for 1 min. Then subjects spat it out, and post-stimulation recordings were continued for 10 min. This trial was repeated for each fluid at intervals of at least 10 min with the confirmation which all responses returned to the baseline level. During an interval, subjects thoroughly rinsed their mouth with distilled water until they did not sense a taste. The four trials for each subject were randomly assigned. All experiments were performed in a quiet and temperature controlled room (25.4 ± 0.7 °C, mean ± SD).

2.4. Measurements

Heart rate (HR) was continuously monitored using an electrocardiogram (BP-88S, Nippon Colin Co., Komaki, Japan) throughout the procedure. The beat-to-beat mean arterial pressure (MAP) was estimated on the left middle finger using the model flow method (Finometer PRO, Finapres Medical Systems, Amsterdam, The Netherlands), and the MAP value was calibrated by a mercury manometer measurement using a left arm cuff.

During the examinations, mean blood velocity (MBV) and vessel diameter of the celiac artery (CA) were continuously recorded in order to evaluate CABF, using a pulsed Doppler ultrasound sonography (LOGIQ S6, GE Medical Systems, Tokyo, Japan) with a 3.5-MHz convex probe. CA measurements were taken within 1–1.5 cm from the origin of the artery. After adjustment of the sample volume width to cover the CA diameter at an expiratory phase, the Doppler probe was kept constant on the subject's skin surface. The Doppler beam insonation angle was maintained at ≤60° relative to the CA. According to a previous study in our laboratory (Endo et al., 2008), the Doppler signals for antegrade and retrograde flow and the electrocardiogram signal were digitally sampled online at 20 kHz using an A/D converter (PowerLab 8/30, ADInstruments, Colorado Springs, USA) and then analyzed offline by the Doppler signal processing software (fast Fourier transfer analysis) to calculate second-by-second MBV. Vessel diameter was obtained by analyzing pictures of the vertical section of blood vessels using B-mode ultrasound. CABF was calculated as:

$$\mathrm{CABF\,(ml/min)} = \mathrm{MBV\,(cm/s)} \times [\mathrm{vessel\ diameter\,(cm)}/2]^2 \times \pi \times 60\,(\mathrm{s}).$$

The celiac artery vascular conductance (CAVC) was calculated as:

$$\mathrm{CAVC\,(ml/min/mm\ \ Hg)} = \mathrm{CABF/MAP}.$$

Electrogastrography was used to measure gastric slow waves, which reflect myoelectrical activity of gastric smooth muscle (Koch and Stern, 2004; Yin and Chen, 2013). This method has a non-invasive and non-restraint nature, considering that the human gastrointestinal tract is a very sensitive system. Throughout the procedure, the electrogastrogram (EGG) was recorded continuously with cutaneous electrodes according to a standard method (Koch and Stern, 2004). As the stomach location for each subject was drawn using a B-mode ultrasonography described above, bipolar electrodes were attached on the abdominal surface to overlay the gastric antrum. To prevent artifacts being mixed into EGG signal, subjects were instructed not to move their body, especially the abdomen, during each trial. The EGG signal was sampled at 10 Hz using an A/D converter (PowerLab 8/35) and amplified with low (0.03 Hz) and high (0.08 Hz) frequency cutoffs. The obtained EGG data were performed by running spectral analysis with a window of 512 points to estimate min-by-min peak amplitude (dominant power, EGGDP) and its corresponding frequency (dominant frequency, EGGDF), indicating a magnitude of GMA and a rhythm of GMA, respectively.

After each trial, subjects filled out a questionnaire evaluating the subjective sweetness intensity and the taste preference of the solution, using Japanese translations of "Labeled Magnitude Scale (LMS)" (Green et al., 1996) and "Labeled Hedonic Scale" (Lim et al., 2009), respectively. The scores of LMS were expressed as logarithmic values in accordance with the study by Green et al. (1996).

2.5. Statistical analysis

The data are expressed as mean and SEM. The effects of time and trial on the temporal data were tested by two-way repeated analysis of variance (ANOVA). When a significant effect was obtained, Dunnett's and Tukey's post hoc tests were conducted to reveal the effects of time against baseline and each trial, respectively. The effects of taste solutions on the subjective sensory evaluations were tested by one-way repeated ANOVA, followed by Tukey's post hoc test. The level of statistical significance was set at $P < 0.05$. All analyses were performed using SPSS 18.0 for Windows (SPSS Inc., Chicago, IL, USA).

3. Results

3.1. CABF and CAVC

Fig. 1 shows an example of CABF responses to the oral stimulation in a subject. The CABF stayed increased 10 min after the oral stimulation by the glucose solutions. In contrast, the water did not increase the CABF. Fig. 2A and B show the changes in CABF and CAVC from the baseline values in all subjects. There were no significant differences in the baseline values. The increments in CABF during all four stimulation were not apparent to reach the level of statistical significance ($P > 0.05$). CABF significantly ($P < 0.05$) increased compared with the baseline after oral stimuli by all three glucose solutions, but was not affected by water. The 4% glucose significantly increased CABF throughout the post-

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