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Experience-Induced Changes in Sugar Taste Discrimination

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Abstract

An apparent plasticity in glucose sensitivity was first noted while studying human taste variants, but the experimental design did not rule out regression to the mean. Since then, a human taste induction hypothesis that sensitivity for a taste stimulus increases with repeated exposure to it has been supported first by experience-induced changes in taste identification of monosodium glutamate and, subsequently, in sensory detection of glutaraldehyde, as well as in psychophysical and functional magnetic resonance imaging responses to novel taste stimuli. Yet, whether such plasticity occurs for the highly familiar taste of sugar remained unconfirmed. Therefore, we tested the taste induction hypothesis for sugar using a counterbalanced design, consisting of 3 pretreatment and 2 treatment conditions. The effects over time also were followed with an additional group of participants. The results showed that 1) experience with fructose significantly increases sensitivity for the taste of a sugar, glucose; 2) there are no significant differences in the sugar sensitivity between groups of randomly assigned participants before treatments; 3) a single session of 5 brief tastings of glucose has an effect on glucose sensitivity when tested 11 or 12 days later; and 4) without continued treatment, the increased sensitivity reverses within 33 or 34 days.

Key words: human psychophysics, plasticity, sugar, taste

Introduction

Plasticity in glucose discrimination was first noted while identifying human taste variants (Eylam and Kennedy 1998a). As the result of screening tests, 12 of 92 participants, who required higher than average concentrations to taste glucose, were categorized as ''nontasters'' for the simple sugar glucose but ''average tasters'' for the glucose isomer, fructose. But when those nontasters were tested further to obtain comprehensive taste profiles, they developed increased discrimination abilities (sensitivity) for glucose. Thus, an experience inducible mechanism was suggested (Eylam 1998; Eylam and Kennedy 1998a, 1998b). However, those longitudinal studies left open the possibility that the observed changes were simply a regression to the mean. A similar plasticity in human olfaction had been found earlier: after repeated exposure to androstenone, ''androstenone anosmics'' could detect androstenone (Wysocki et al. 1989). Also, repeated exposure of the olfactory epithelium to pemenone increased sensitivity to androstenone (Stevens and O'Connell 1995).

Since then, the human taste induction hypothesis that sensitivity for a stimulus increases with repeated exposure to it has been supported by various measures in 4 other studies. First, experience-induced changes were shown in taste identification of monosodium glutamate (MSG) (Kobayashi and Kennedy 2002). Participants treated with MSG in food briefly each day for 10 days identified MSG in solution at lower concentrations than participants treated with food without MSG (Kobayashi and Kennedy 2002; Kobayashi et al. 2006). Such changes in MSG identification abilities reverse when treatment is ceased (Kobayashi et al. 2006). Taste induction also was verified by measuring detection thresholds for glutaraldehyde in drinking water (Cain and Schmidt 2002). Some participants who were asked to taste 3 samples of glutaraldehyde and water and then choose the strongest flavor were able to detect glutaraldehyde during their first session. Three subsequent tasting sessions also revealed progressive improvement. Yet, of participants who could not detect the glutaraldehyde in the first session, some did detect it as soon as the second tasting session 5 days later (Cain and Schmidt 2002). Finally, functional magnetic resonance imaging (fMRI) and psychophysical responses were measured concurrently in participants repeatedly exposed to the novel stimuli, aspartame, quinine hydrochloride, D-threonine, glycyrrhizic acid, and 5′-guanosine monophosphate. The results showed that fMRI pixel activation and psychophysical measures of isointensity and magnitude estimation

increased concurrently over several weeks of exposure to the stimuli (Faurion et al. 2002). The previously shown plasticity in gustation and olfaction involved stimuli not readily recognized in the everyday experience of most Americans.

Here, we show that the taste induction occurs with a highly recognized stimulus—sugar. We tested the taste induction hypothesis for sugar, using a counterbalanced design consisting of 3 pretreatment test conditions and 2 treatment conditions. We also followed the effects over time in an additional group of participants. The results showed that treatment with fructose significantly increased discrimination abilities for glucose, and after treatment ceased, there was a significant reversal of increased discrimination abilities.

Materials and methods

Participants

The Clark University Committee for the Rights of Human Participants in Research and Training Programs and the Smith College Institutional Review Board approved this research. All participants were volunteers and members of the Clark University community or acquaintances of the experimenters. Each participant was informed orally and in writing that the intent of the experiment was to study the effects of experience on human responses to sweet tastes. Of the 167 participants from Experiments I and II, 99 were females and 68 were males, with a mean age of 24.49 (±11.25 standard deviation [SD]) years. Participants were asked not to participate if they were diabetic.

Solutions

Solutions were prepared with distilled water (Poland Springs, Hollis, Maine; refractive index 1.3330, conductivity 2.0×10^{-6} mega Ω). Fructose and glucose were American Chemical Society reagent grade (Sigma-Aldrich, St Louis, MO). Food coloring, used to provide some distraction, was obtained from a local supermarket (Betty Crocker Signature Brands, Ocala, FL). Each 500 ml of solution contained 5 drops of food coloring.

The "sham pretest" solutions were distilled water, colored green. The "glucose pretest" and testing solutions were 17.5, 27, 42, 65, and 100 mM glucose, colored green. These concentrations represent the mean positive identification for glucose and ±1 and 2 SD. This range includes a low concentration that almost no participants would be expected to recognize and a high concentration that almost all participants would be expected to recognize (Eylam and Kennedy 1988a; Kennedy et al. 1997). The test solutions and water were stored in a refrigerator at 5 °C for ≤2 weeks and then brought to room temperature (21 ± 1 °C) before testing. Participants were instructed to store treatment solutions in a refrigerator and bring solutions to room temperature at home before treating their tongues with the solution.

The treatment solution for the experimental groups was fructose 43 mM, a concentration which is the mean for positive identification (Eylam and Kennedy 1998a). The treatment solution for the control groups was distilled water.

Procedure

Experiment I

The counterbalanced design is shown in Figure 1. The 3 pretest conditions were "no pretest," "sham pretest," or "glucose pretest." The no pretest group immediately started their at-home treatment for 10 days. The sham pretest or glucose pretest groups were tested in the laboratory using the sip and spit method in a criterion-free, forced choice procedure (Kennedy et al. 1997; Eylam and Kennedy 1998a). They were presented with 5 pairs of solutions, colored green. Each pair in the sham pretest consisted of 2 cups of water (10 ml each), and each pair in the glucose pretest consisted of 1 cup of water and 1 cup of a specific concentration of glucose, with the glucose and water placed randomly within the pair. Among the pairs, the glucose concentrations were presented in ascending order to minimize any induction effects during the testing procedure. Before tasting each solution, participants rinsed their mouth with a comfortable amount of distilled water. The experimenters sat behind the participant during testing to reduce any psychological effects of their body language on the participants and controlled the temporal sequence of the experiment by verbal instructions according to a digital stopwatch. Each solution took 30 s: 10 s to taste, 10 s to spit, 10 s to rinse; additionally 10 s were given for the participant to mark the data sheet. There were 1 min and 10 s intervals between the first cup in 1 pair of solutions

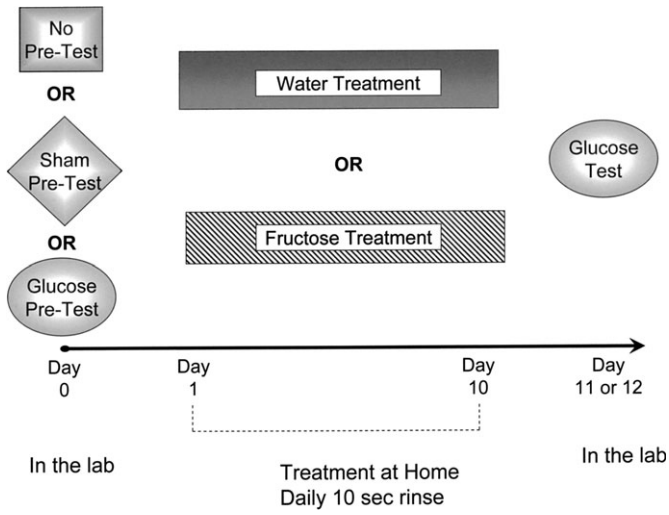

Figure 1 Schematic diagram for the design of Experiment I. Participants (153) were randomly assigned to 1 of 3 pretest conditions—no pretest, sham pretest, or glucose pretest—and 1 of 2 treatment conditions—fructose or water—for a total of 6 pretest/treatment groups. Participants were glucose pretested and sham pretested in the laboratory. After 10 days of fructose or water treatment at home, all participants returned to the laboratory on Day 11 or 12 for a glucose discrimination test.

and the first cup in the next pair. This timing was chosen to minimize the effects of sensory adaptation but to not allow the participant to forget the taste of the previous solution (Kennedy et al. 1997). After tasting both solutions in the pair, participants were then asked, "Which is the sugar?" Participants then marked their answers on a paper data sheet.

The treatment procedure was adapted from previous research in our laboratory (Sullivan et al. 1999). Participants were given a bottle containing 500 ml of treatment solution. The treatment solutions were assigned to the participants randomly, although the numbers of males and females were kept approximately equal in the experimental and control groups. Participants were instructed to take a comfortable amount of treatment solution into their mouths, move the liquid around so as to thoroughly bathe their tongues for 10 s, and then expectorate it once daily for 10 consecutive days at home. They were told not to concern themselves with the taste of the treatment solution because the taste of the treatment solution was not relevant to the experiment.

Following 10 days of at-home treatment, on Day 11 or 12, participants were tested in the laboratory. They were told not to eat, drink, or brush their teeth 1 h prior to testing time. Each participant was asked how the treatment went, and those who reported not completing all the treatment sessions were not used as participants. All participants then were tested as in the glucose pretest.

Experiment II

An additional 14 participants were followed over time (Figure 2). They were first glucose pretested in the laboratory. All then treated themselves with 43 mM fructose at home for 10 days and returned to the laboratory for a glucose test on Day 11 or 12. Then again in increments of 11 or 12 days after treatment ceased, that is, on Day 23 or 24 and on Day 33 or 34, participants returned to the laboratory and were tested as before, in the glucose pretest.

Data analysis

Threshold discrimination categories 1–6 were assigned to the data according to the lowest concentration of glucose identified correctly as well as correct identification of all the higher concentrations (Kobayashi and Kennedy 2002). The discrimination category was assigned only when the correct discrimination was made consistently: if a participant chose the correct solution at a lower glucose concentration, but then chose the water at the next higher glucose concentration, which was an incorrect answer, it was assumed that the participant only guessed and was assigned the higher threshold category. A threshold category of 1 meant that the participant had correctly discriminated as "sugar" the lowest concentration of glucose (17.5 mM) and all the higher concentrations of glucose (27, 42, 65, and 100 mM). Threshold category 2 meant that the participant discriminated the second lowest concentration (27 mM) correctly as well as all the higher concentrations. A participant assigned a 5 was one who discriminated the highest concentration (100 mM) correctly with the assumption that this correct discrimination was not random. The highest threshold category was 6; this category meant that the participant had not discriminated any of the concentrations correctly or had inconsistently discriminated the concentrations throughout the test.

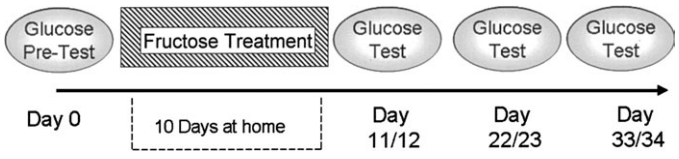

Figure 2 Timeline for Experiment II. To determine the time course of the induction, 14 additional participants were glucose pretested and fructose treated. They then were given glucose discrimination tests on Days 11 or 12, 22 or 23, and 33 or 34.

Whether ordinal data should be analyzed by parametric or nonparametric statistics is controversial (e.g., Stevens 1951; Gaito 1980; Lawless and Heyman 1998). Therefore, data were analyzed with parametric 1- and 2-way analysis of variances (ANOVAs) and *t*-tests and confirmed with nonparametric Kruskal–Wallis and Wilcoxon tests. The percentile data also were confirmed with a median test. For Experiment II, a repeated measures multivariate analysis of variance (MANOVA) was used and confirmed by the nonparametric Kramer test (Kramer et al. 1974). All results were significant, or not, by both parametric and nonparametric analyses. In the case of the single 2-way ANOVA, a nonparametric test for confirmation was not readily available. All analyses were done with JMP v.5, except for the Kramer Test, which was calculated by hand.

Results

Treatment with fructose increased glucose discrimination ability. There was a significant difference among the 6 groups of Experiment I in the ability to discriminate glucose from water after treatment ($F_{4,148}$ = 3.75, P = 0.006, 2-way ANOVA) (Figure 3). As predicted by the taste induction hypothesis, the mean and median posttreatment threshold categories for the combined "fructose-treated" groups (n = 80) were significantly lower than the mean and median posttreatment threshold categories for the combined "water-treated" groups (n = 73) (t_{151}= 2.92, P = 0.004, t-test; P = 0.005, Wilcoxon; P = 0.01, median test) (Figure 4). There was no significant difference in glucose discrimination ability between male and female participants (P = 0.9, 2-way ANOVA effect test).

The "pretests" increased the posttreatment glucose discrimination ability of water-treated participants (Figure 3). For the no pretest groups, fructose-treated participants had significantly lower mean and median threshold categories than water-treated participants did after treatment

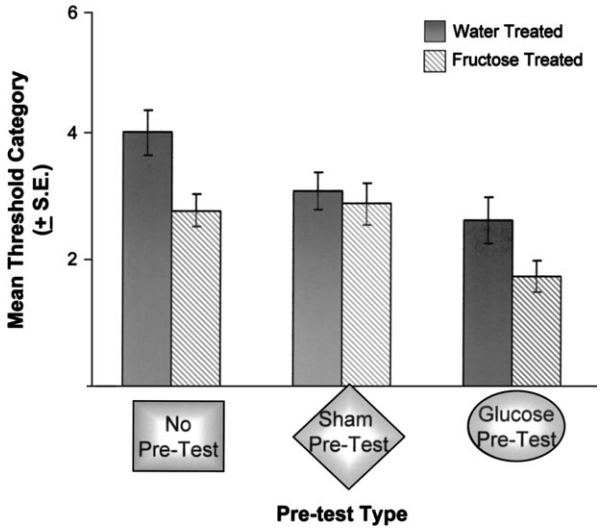

Figure 3 Mean glucose discrimination threshold categories for the 6 pretest/treatment groups in Experiment I (N = 153). There was a significant difference in glucose discrimination ability among the groups when tested at 11 or 12 days (P = 0.03, 2-way ANOVA). There was no significant difference among the 3 fructose-treated groups (P = 0.6, ANOVA). There was a significant difference in glucose sensitivity between the no pretest, fructose-treated group and the no pretest, water-treated group (P = 0.001, t-test). There also was a significant difference among the 3 water-treated groups (P = 0.01, ANOVA). The glucose pretest group and the sham pretest group each discriminated glucose at significantly lower concentrations than the no pretest group (P = 0.004, P = 0.040, t-tests).

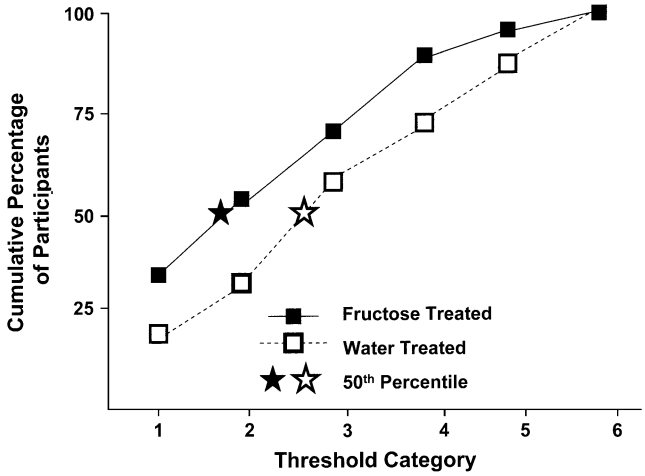

Figure 4 Cumulative frequency percentages for all participants in Experiment I at the various threshold categories (1–6) for glucose discrimination. There was a significant difference in glucose discrimination ability between all participants treated with fructose (n = 80) (solid squares and line) and all those treated with water (n = 73) (empty squares and dotted line) (P = 0.004, t-test). The threshold categories for the 50th percentile of participants treated with fructose (solid star) were significantly lower than those treated with water (open star) (P = 0.005, Wilcoxon; P = 0.01, median test).

(t_{67} = 3.37, P = 0.001, t-test; P = 0.001, Wilcoxon). However, for the sham pretest and glucose pretest groups, the small differences after treatment between fructose-treated and water-treated participants were not statistically significant (t_{40} = 0.50, P = 0.62, t_{40} = 0.48, P = 0.63, respectively, t-tests; P = 0.8, P = 0.7, respectively, Wilcoxon). Figure 3 shows that while 1) threshold categories of the 2 pretest, fructose-treated groups were at least as low as that of the no pretest, fructose-treated group; 2) the threshold categories of the 2 pretest, water-treated groups were nearly as low as the 2 pretest, fructose-treated groups; and 3) the threshold categories of the 2 pretest, water-treated groups were considerably lower than that of the no pretest, water-treated group. Moreover, the mean threshold category of the sham pretest, fructose-treated group was similar to that of the no pretest, fructose-treated group, but the threshold category of the glucose pretest, fructose-treated group was lower. Statistically, posttreatment threshold categories differed among the 3 water-treated groups ($F_{2,70}$ = 4.66, P = 0.01, ANOVA; P = 0.01, Kruskal–Wallis), with significant differences between the no pretest and sham pretest, water-treated groups (t_{54} = 2.04, P = 0.05; P = 0.05) and between the no pretest and glucose pretest, water-treated groups (t_{109} = 2.64, P = 0.001; P = 0.01), but no significant difference between the sham pretest and the glucose pretest, water-treated groups (t_{37} = 0.84, P = 0.41; P = 0.43) (t-test; Wilcoxon). Despite the significant difference among the 3 water-treated groups, there was no difference among the 3 fructose-treated groups ($F_{2,77}$ = 0.5, P = 0.6, ANOVA; P = 0.54, Kruskal–Wallis).

Random assignment of participants from the Clark University community to treatment groups resulted in groups that began with the same mean glucose discrimination ability before treatment. There was no statistically significant difference in mean and median threshold categories before treatment between the glucose pretest, fructose-treated group (2.96 ± 0.29 standard error [SE]) and the glucose pretest, water-treated group (2.35 ± 0.35 SE) in Experiment I (n = 37) (t_{40} = 1.34, P = 0.19, t-test; P = 0.08, Wilcoxon). Also, there was no significant difference for mean and median threshold categories before treatment among the 3 glucose pretest groups from Experiment I and Experiment II ($F_{2,53}$ = 1.97, P = 0.15, ANOVA; P = 0.09, Kruskal–Wallis).

There were significant changes in glucose discrimination ability of the 14 additional participants over the time course of Experiment II (N = 14) (P = 0.04, repeated measures MANOVA; $P \leq 0.01$, Kramer Test) (Figure 5). After fructose treatment was stopped, mean and median threshold categories at Day 11 or 12 and Day 22 or 23 each were significantly lower in comparison with the pretreatment mean (t_{13} = 2.59, P = 0.02, t_{13} = 3.45, P = 0.004, respectively; $P \leq 0.01$). Glucose discrimination ability had returned to the pretreatment level by Day 33 or 34, that is, the mean and median threshold category at Day 33 or 34 was not different from the pretreatment mean and median (t_{13} = 0.46, P = 0.65; $P > 0.05$) (paired t-tests; Kramer Test). Individually, the glucose discrimination abilities of 14% (2) of participants had returned to their own pretreatment levels at Day 22 or

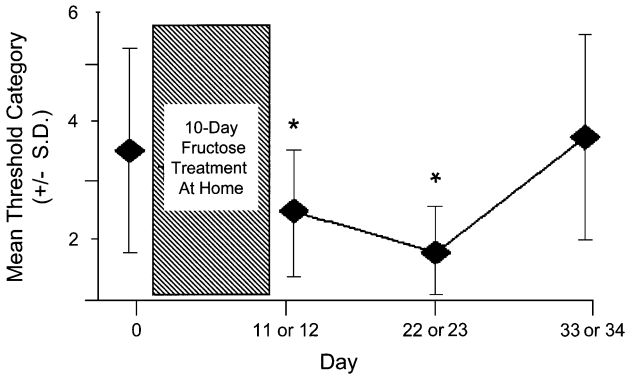

Figure 5 Time course of changes in glucose discrimination ability for the 14 glucose pretest, fructose-treated participants in Experiment II. The changes over time were significant ($P < 0.04$, repeated measures MANOVA). On Day 11 or 12, the mean threshold category was significantly lower than pretest levels ($P = 0.02$). On Day 22 or 23, 2 participants had returned to their own pretreatment sensitivity levels, but the overall mean threshold category for the group remained significantly lower than pretreatment levels ($P = 0.004$). On Day 33 or 34, the group had returned to the pretreatment sensitivity level: the mean threshold category for glucose discrimination on Day 33 or 34 was not significantly different from the mean threshold category before treatment ($P = 0.65$) (paired *t*-tests).

23, whereas 39% (8) of participants did not show pretreatment levels until the test at Day 33 or 34. Two participants showed no change over the entire experiment and 2 continued to increase in glucose discrimination ability throughout the experiment.

Conclusions and discussion

Participants treated with fructose discriminated glucose as sugar at significantly lower concentrations than participants treated with water. Participants treated with fructose also discriminated glucose as sugar at significantly lower concentrations than they did before fructose treatment. When treatment was ceased, the induction effect reversed, that is, participants returned to their pretreatment glucose threshold categories. These results confirm the experience-induced increase in glucose taste discrimination first reported by Eylam and Kennedy (1998a), and show that the phenomenon is reliable, by both across-groups and longitudinal experimental designs. Experience-induced increases in responsiveness to novel stimuli are known, and we now have confirmed, with robust results, that such plasticity also occurs with a familiar stimulus—sugar.

When participants received no pretest, those treated with fructose discriminated glucose at significantly lower concentrations than those treated with water. But when participants received either a glucose pretest or a sham pretest, the small differences between fructose-treated and water-treated participants were not significant (Figure 3). Moreover, glucose pretest or sham pretest participants treated with water discriminated glucose at significantly lower concentrations than those who had received no pretest before water treatment. These results show first that a glucose pretest, consisting of 5 brief tastings of glucose in a 6-min period, is sufficient to affect discrimination ability when tested 11 or 12 days later. The effects of the sham pretest suggest a practice or learning effect, presumably involving a cognitive, central nervous system (CNS) process.

As predicted by statistical theory, there was no difference in discrimination ability before treatment between the 2 glucose pretest groups in Experiment I and among the 3 glucose pretest groups of the 2 experiments. This result confirms empirically that there is no difference in pretreatment discrimination abilities between groups when the participants are randomly assigned from a population (the Clark University community) to treatment groups. Given that a pretest is sufficient to affect discrimination ability at 11 or 12 days and that there was an effect, presumably cognitive, of a pretest when tested after treatment, an experimental design consisting of random assignment of participants to treatment groups, and no pretest, is an appropriate design for further study of the induction.

Because participants were treated with fructose and then tested with glucose, the results suggest that the experience inducible mechanism is one that is common or interactive for the 2 sugars. Some psychophysical data suggest that mechanisms for fructose and glucose are different (Kennedy et al. 1997; Armstrong et al. 1998; Eylam and Kennedy 1998a; Savant and McDaniel 2004; Heath et al. 2006), whereas others (e.g., Breslin et al. 1996) suggest a common mechanism. Presumably, the different mechanisms occur peripherally, perhaps in the several binding sites of the single known heterodimeric sweet taste receptor, T1R2/T1R3 (see Nelson et al. 2001; Li et al. 2002). The common or interactive mechanism may occur later in the sugar taste–processing pathway, perhaps in the whole sweet receptor molecule, in the cellular transduction pathways, or in neural pathways.

Here, we found that treatment with fructose increased glucose discrimination ability. But, we have found elsewhere that while treatment with the high-intensity sweetener Na-cyclamate increased glucose discrimination, treatment with acesulfame-K did not (Hassan et al. 2006). Likewise, Dalton et al. (2002) found that treatment with Na-saccharin did not decrease taste thresholds for Na-saccharin. It appears that acesulfame-K and Na-saccharin are inhibitory for sweet taste. Galindo-Cuspinera et al. (2006) showed that when participants tasted either Na-saccharin or acesulfame-K for the first time, the stimulus was perceived as sweet. Upon a second tasting, at high concentrations, both sweeteners act as antagonists, inhibiting the sweet taste of sucrose as well as of the acesulfame-K and Na-saccharin. The inhibition of sweetness was proposed to be the result of a conformational change of the allosteric binding site on the sweet taste receptor, hTAS1R2/hTAS1R3 (Galindo-Cuspinera et al. 2006). A similar inhibition by acesulfame-K and Na-saccharin could be responsible for the lack of

induction in our Hassan et al. (2006) and the Dalton et al. (2002) studies.

Although 2 participants in Experiment II had returned to their individual pretreatment glucose threshold categories by Day 22 or 23, the mean and median threshold categories for the group remained low at Day 22 or 23 and did not return to pretreatment levels until the test on Day 33 or 34. In contrast, Kobayashi et al. (2006) found a significant reversal of increases in taste identification of MSG at 22 or 23 days. Possibly, the use of a complex treatment stimulus (MSG in potato crackers) by Kobayashi et al. (2006) as compared with our use of a simple fructose solution resulted in a weaker induction that reversed more rapidly. In contrast to gustation, increased olfactory sensitivity to adrostenone remained 6 weeks after termination of exposure (Wysocki et al. 1989). Likewise, the olfactory system of rats maintained increased sensitivity to androstenone 45–50 days after bilateral olfactory nerve transaction, and the sensitivity was reversed 121–203 days after surgery (Yee and Wysocki 2001). This difference may involve inherent differences in the gustatory and olfactory systems.

There were no significant differences in data from males and females. Similarly, the 2 MSG induction studies (Kobayashi and Kennedy 2002; Kobayashi et al. 2006) and the Wang et al. (2003) olfactory induction study found no male/female differences in their data, but the lack of a difference in Wang et al. (2003) may have been due to the small number of participants (Wang et al. 2003). In contrast, Dalton et al. (2002) found that olfactory sensitivity increased an average of 5 orders of magnitude in women of reproductive age, but not in men, for a variety of odors. The mean age of the 99 females and 68 males in our study was 24.49 (±11.25 SD) years, a reproductive age, but we did not screen for reproductive ability of our participants. These different results may reflect some difference in induction mechanisms between olfactory and gustatory systems, with the olfactory system more susceptible to gender effects than the gustatory system.

Investigation of the biological mechanisms underlying the induction requires first, determination of the locus of the induction. Some animal data suggest that experience-induced changes in chemosensory sensitivity take place in the peripheral nervous system (PNS). In hamsters, the magnitudes of chorda tympani responses increased after repeated stimulation with novel taste stimuli (Berteretche et al. 2005). Also, inspection of fly taste receptor cell data reveal that firing rates increased after repeated stimulation with sucrose (see Figure 3 in Kennedy and Halpern 1980). Thus, the locus of plasticity may be in or before the taste receptor cell. Similarly, olfactory data implicate the olfactory epithelium as the place where the change in androstenone sensitivity takes place: in normosmic mice, sensitivity to androstenone increased following exposure to androstenone during a time that the epithelium and olfactory bulbs were disconnected (Yee and Wysocki 2001).

However, the mechanisms in animal models could differ from those in humans. Also, peripheral mechanisms do not rule out the possibility of mechanisms in the CNS as well. Corresponding central changes have been shown in humans: psychophysical and fMRI responses in participants repeatedly exposed to novel taste stimuli showed "familiarization," that is, pixel activations and estimations of stimulus isointensities and magnitudes that increased concurrently over several weeks of exposure to the stimuli (Faurion et al. 1998; Faurion et al. 2002). Yet, those CNS changes could have resulted from changes that had occurred in the periphery. Wang et al. (2003) addressed the PNS versus CNS question by recording human electro-olfactogram (EOG) and brain olfactory event-related potentials (OERP) simultaneously, while the participants were repeatedly exposed to, and were developing a reduced psychophysical threshold for, androstenone. Regression analysis showed a significant relationship between the psychophysical threshold and EOG with a simple exponential relationship. The same order of magnitude changes in the EOG and OERP supported the idea that the plasticity takes place in the PNS (Wang et al. 2003). Further work on the locus of the taste induction is needed.

In summary, experience with fructose increases discrimination ability for the familiar taste of a sugar, glucose. There are no significant differences in sugar discrimination between groups of randomly assigned participants before treatments. A single session of 5 brief tastings of glucose has an effect on discrimination when tested 11 or 12 days later. Without continued treatment, discrimination abilities return to pretreatment levels within 33 or 34 days. Currently, we are investigating whether the mechanisms for experience-induced changes reside in taste receptor cells and what the specific biological mechanisms might be.

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