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# Experience-Induced Changes in Taste Identification of Monosodium Glutamate (MSG) Are Reversible

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## Abstract

A few studies have reported experience-inducible changes in human taste and olfactory sensitivities. However, no study thus far has systematically characterized the stability of the enhanced sensitivities. In our previous study, we found increases in taste identification ability for monosodium glutamate (MSG) in subjects who had been briefly exposed to MSG in food for 10 days. Here, we tested the temporal stability of the enhanced taste identification ability. First, we exposed a group of 20 subjects to MSG in food and then compared their sensitivities to MSG with those of a control group. When tested on day 11 or 12, the mean MSG taste identification ability of the MSG-exposed group was significantly higher than the control group. Next, 11 of the subjects who were exposed to MSG in food initially, and then stopped being exposed performed significantly poorer in identifying MSG after 10 days of the nonexposure than they did 10 days before. In contrast, nine subjects who were exposed to MSG initially and continued being exposed maintained their high identification levels. These results support earlier finding of the plasticity in the taste identification of MSG and show that the enhanced identification ability can be reversed rapidly when MSG exposure is not sustained.

**Key words:** human taste induction, plasticity, umami

## Introduction

Is human taste sensitivity for a particular taste compound stable over time? Unlike other sensory systems, the gustatory system has been suggested to exhibit considerable plasticity induced by the environment in peripheral as well as in central structures and functions (Hill and Doi, 2004). Our previous study provided evidence for the rapid plasticity in taste. When tested after 10 days of brief exposures to monosodium glutamate (MSG) in a treatment food, a group of subjects increased their identification ability for MSG taste as compared to the control subjects (Kobayashi and Kennedy, 2002). Both earlier and subsequent studies found experience-induced changes in sensitivity to sweet taste (Eylam and Kennedy, 1998; Peo *et al.*, 2005), and a recent neuroimaging investigation found that people's sensitivity to novel taste stimuli resulted in cortical activations/deactivations (Faurion *et al.*, 2005). Also, olfactory induction studies have demonstrated enhanced sensitivities in androstenone- or glutaraldehyde-anosmic subjects after repeated exposures to androstenone (Wysocki *et al.*, 1989) or to glutaraldehyde (Cain and Schmidt, 2002). But, how long is the enhanced sensitivity maintained after the initial induction? A recent olfactory study indicated that induced sensitivity change to androstenone persists as long as 6 weeks (Wysocki *et al.*, 1989). However, the durations of the induced taste plasticity in human taste are yet to be explored.

The flavor or taste of MSG has been described as ''umami'' (Yamaguchi and Ninomiya, 1998) by some investigators and as glutamate taste by others (Halpern, 2002a). Umami has been considered as a fifth basic taste following ''sweet,'' ''salty,'' ''sour,'' and ''bitter'' by some taste researchers (Faurion, 1987, 1991; Beauchamp *et al.*, 1998; Kurihara and Kashiwayanagi, 2000; Halpern, 2002b). Two earlier studies, including our own, found a significant difference between Japanese and Americans in their MSG taste discriminability (Ishii *et al.*, 1992) and identification ability (Kobayashi and Kennedy, 2002). In our study, we showed that taste identification ability to MSG increased after 10 days of brief exposures to MSG in a treatment food (Kobayashi and Kennedy, 2002). However, we did not evaluate whether or not the experience-induced change

continued in the absence of further exposure to MSG. Thus, the duration of the increased identification ability was unknown.

The present study aimed at determining temporal properties of the experience-induced change; that is, to ask the question: Whether and how quickly will the enhanced taste sensitivity to MSG dissipate in the absence of exposure to MSG? As in our previous work, we compared a group of subjects who had been exposed to MSG in a snack food, with a control group without MSG exposure in their control snack food, for changes in their taste identification ability for MSG after 10 days of exposures to MSG. We predicted that the group of subjects who had been exposed to MSG in food would perform better in a forced-choice task for MSG taste than those who had not been exposed to MSG. To examine the maintenance of the improved taste identification ability, we then divided the MSG-exposed group further into two groups: subjects who continued to eat the treatment snack food for another 10 days and subjects who stopped the snacking. Both groups were tested after an additional 10 days with the same forced-choice test.

## Materials and methods

### Subjects

The protocol of this research was reviewed and approved by the University Committee on Human Subjects at Cornell University. Prior to the experiments, informed consent was obtained from all participants. Most participants were Cornell undergraduate students who participated in the study for extra credit in some Psychology courses. Of the total 40 subjects, 19 were males and 21 were females, with an overall mean age of 24.2 (±7.23 SD) years. Thirty-seven subjects were North Americans and three were Europeans. All subjects were healthy, and none of them had known physiological hypersensitivity to MSG or family members with asthma, including themselves. Subjects were asked about their dietary patterns in a multiple-choice questionnaire shortly before the two test sessions.

### Treatment foods and test solutions

The treatment foods were two snack foods, ''potato-flavored snack'' and ''sweet potato-flavored snack''; Nong Shim, Dokjak-gu, Seoul, Korea; purchased from a local oriental market. The MSG Group treatment food was potato crackers (the potato-flavored snack) containing MSG (list of ingredients: wheat flour, potato powder, palm oil, corn starch, rapeseed oil, sugar, salt, onion powder, MSG). The Control Group treatment food was the sweet potato crackers (list of ingredients: wheat flour, sweet potato, palm oil, sugar, corn starch, salt, black sesame). These snacks were similar in appearance, texture, and flavor. Other compounds that are often described as having ''umami'' taste such as inosine 5′-monophosphate and guanosine 5′-monophosphate were not listed as ingredients by the manufacturer.

Test solutions contained various concentrations of either MSG (Ajinomoto, Chuo-ku, Tokyo, Japan; provided as a gift to Halpern) or sodium chloride (NaCl) (grade = 99.7%; Sigma, St Louis, MO). The MSG test solutions included a low concentration (0.625 mM), which almost no subject was expected to identify as MSG, and a high concentration (2.5 mM), which almost all were expected to identify as MSG, as well as three intermediate concentrations (0.925, 1.25, 1.85 mM) that some but not all subjects were expected to identify as MSG, according to previous data (Yamaguchi, 1991; Ishii *et al.*, 1992; Kobayashi and Kennedy, 2002). Each MSG solution was paired with an NaCl solution of approximately equal taste intensity (0.625, 0.925, 1.25, 1.85, 2.5 mM), as indicated in those previous studies (Yamaguchi, 1991; Ishii *et al.*, 1992; Kobayashi and Kennedy, 2002). All solutions were prepared with purified (polished reverse osmosis) water in our laboratory. Quality (i.e., conductivity and refractive index properties) of the water was checked whenever the new solutions were made [refractive index ($r$) $\leq 1.3330 \times 10^4$and conductivity $\leq 2.5 \times 10^{-16}$ 1/Ω at 20°C]. The test solutions were stored in a refrigerator at 5°C for 2–3 weeks and brought to 25 ± 1°C before tasting sessions.

### Procedures

Twenty subjects (11 males, 9 females) were given a bag of potato crackers (MSG Group), and another 20 subjects (8 males, 12 females) were given a bag of sweet potato crackers (Control Group) (Figure 1). The subjects were assigned to treatment groups randomly. All subjects were instructed to ''eat a few pieces of potato or sweet potato crackers each day for 10 days at home.'' They were instructed not to pay attention to the taste of the treatment food. Subjects were limited in the use of treatment foods (i.e., 1–3 pieces of crackers

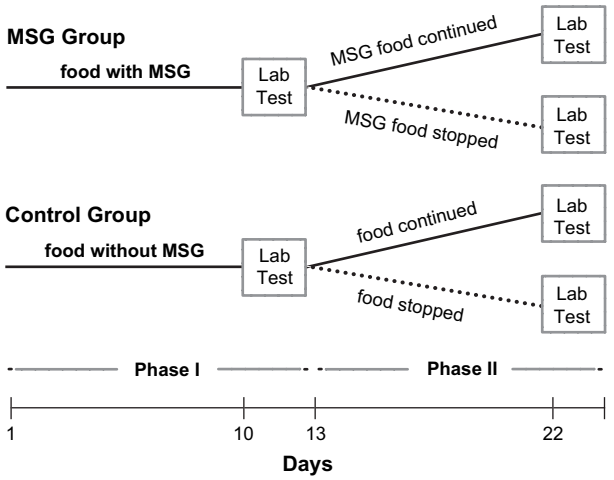


**Figure 1** Schematic plan of experiment.

a day). No specific time interval was given for the amount of time that subjects should eat the treatment foods. On day 11 or 12, after 10 days of exposures to the treatment foods (Phase I), all subjects were tested in the laboratory with aqueous solutions of MSG and NaCl in a forced-choice procedure (see below and Figure 1, Session 1 ''Lab Test''). Then, nine subjects in the MSG Group (the group that received MSG-containing potato crackers for snacking) and 10 subjects in the Control Group (the group that received sweet potato crackers for snacking) were asked to continue eating (MSG-Continued and Control-Continued groups), while 11 subjects in the MSG Group and 10 subjects in the Control Group were asked to stop eating the treatment foods (MSG-Stopped and Control-Stopped groups) (Phase II) (Figure 1). On day 21 or 22 (after another 10 days of exposure/nonexposure to MSG in the treatment foods), all subjects were tested again with the same procedure (Figure 1, Session 2 Lab Test).

Subjects had been instructed not to eat for at least 1 h before the Lab Test tasting sessions. Testing procedures were as follows. First, each subject sipped and spit 10 ml of a high concentration (5 mM) of MSG and was told, ''This is MSG. Choose the MSG solution in each pair using this first solution as a reference. One in each pair is the MSG solution and one is a salt solution.'' They then sipped and spit the five pairs of solutions in order of ascending concentration, with 30-s intervals between each member of a pair, and indicated the MSG solution of each pair on the data sheet. Within each pair, the MSG/NaCl presentation order was random. Subjects rinsed their mouths with a comfortable amount of purified water before sipping each solution. The temporal sequence of the experiment was controlled by the experimenter (C. Kobayashi) seated behind the subjects using a digital stopwatch or wristwatch. The sequence was as follows: 40 s for each solution, including 10 s of tasting, then sipping and rinsing, and 1 min between each pair to minimize the effects of adaptation.

### Statistical analyses

Categories of identification levels were defined as follows: Category 6 for subjects who identified all five test concentrations (0.625, 0.925, 1.25, 1.85, 2.5 mM) of MSG correctly; Category 5 for those who identified the four highest concentrations correctly; Category 4 for those who identified the three highest concentration correctly; Category 3 for those who identified the two highest concentrations correctly; Category 2 for those who identified only the highest concentrations correctly; and Category 1 for those who did not identify even the highest concentration. For Phase I, a comparison of median identification categories (MICs) after 10 days of exposures/nonexposures to MSG between the two groups (MSG Group and Control Group) was made by a one-tailed permutation test (a.k.a., randomization test) for two independent samples (Conover, 1980; Siegel and Castellan, 1988; Darlington, 2005). We also compared the changes in the MICs between Phase I and Phase II for each of the four subgroups (MSG Continued, Control Continued, MSG Stopped, and Control Stopped) by two-tailed permutation tests for paired samples (Conover, 1980; Siegel and Castellan, 1988; Darlington, 2005). In addition, to determine gender effects, we compared between the genders for their MICs in each phase by two-tailed permutation test for two independent samples.

## Results

### Phase I: effects of 10 days of exposures to MSG in food

There was a significant difference between the MSG Group and the Control Group in identifying the MSG solutions. As expected, after Phase I, the MSG identification ability of the MSG Group had a significantly greater identification for MSG taste than the Control Group ($P < 0.05$) (Table 1 and Figure 2). For all concentrations, more subjects in the MSG Group identified the MSG solutions correctly than in the Control Group after the 10-day treatment period of Phase I.

### Phase II: effects of additional 10 days of exposures/nonexposures to MSG in food

As shown in Figure 3, there was a significant decrease in the MIC from Phase I to Phase II only for the MSG-Stopped Group ($P < 0.05$). The changes in the MIC for the other three groups from Phase I to Phase II were all nonsignificant, $P > 0.05$ (Table 1 and Figure 3).

### Effects of gender

There was no difference in the MSG identification ability between the genders for Phase I, $P > 0.1$ [median ± semi-interquartile range, males ($n = 19$) and females ($n = 21$): 3 ± 1] or for Phase II, $P > 0.05$ (males: 4 ± 1; females: 3 ± 2; $P > 0.5$). In addition, there were no changes in the MSG identification ability for male subjects ($P = 1$) or for female subjects ($P > 0.3$) between Phase I and Phase II.

## Discussion

There are two important findings from our results. First, we replicated Kobayashi and Kennedy (2002) by finding

**Table 1** MIC measured after Phase I and Phase II

| Group | MIC after Phase I (semi-interquartile range) | MIC after Phase I | MIC after Phase II |
|---|---|---|---|
| MSG Continued ($n = 9$) | 3.5 (1.125) | 3 (1.5) | 5 (1.5) |
| MSG Stopped ($n = 11$) | | 4 (1) | 3 (1.25) |
| Control Continued ($n = 10$) | 2.5 (1.5) | 1.5 (1.25) | 3.5 (1.5) |
| Control Stopped ($n = 10$) | | 3 (0.75) | 2 (2.125) |

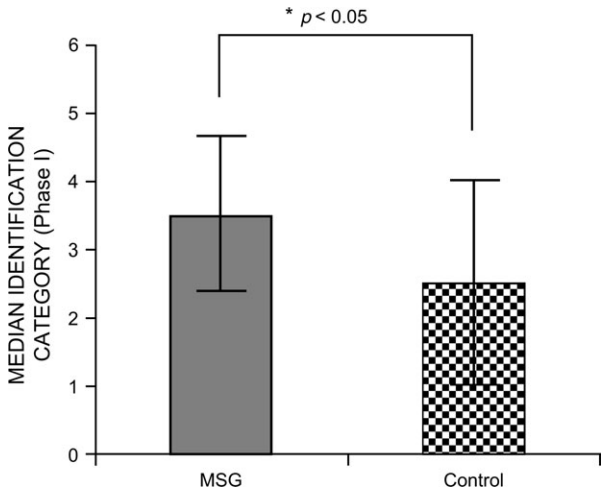


**Figure 2** MICs after 10 days of exposures to MSG. Data for subjects exposed briefly each day for 10 days to potato crackers are given by the solid bar, and data for control subjects exposed to sweet potato crackers by the patterned bar ($P < 0.05$, permutation test) (sticks represent semi-interquartile ranges).

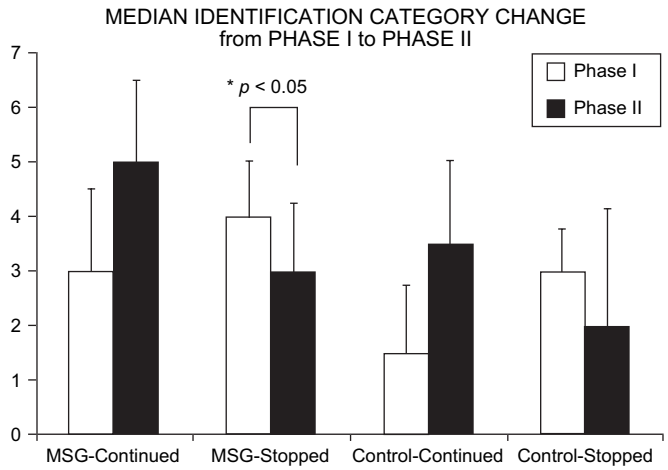


**Figure 3** Changes in the MICs of the four groups during Phase I (white bars) and Phase II (black bars). At the end of the Phase II, there were small changes for the MSG-Continued and the two control groups, but these changes were not statistically significant ($P > 0.05$). The only statistically significant change was in the MSG-Stopped Group, which showed a clear decrease in the MIC ($P < 0.05$) (sticks above the bar represent positive portions of semi-interquartile range).

a significant difference between the MSG Group and the Control Group in MSG identifications after Phase I. This finding adds to the evidence that there is an experience-inducible component in MSG taste identification ability. Second, we found a significant decline of MSG taste identification ability in the MSG-Stopped Group but maintenance of such ability in the MSG-Continued Group after Phase II. These results indicate that without continued exposure to MSG in food, initial enhancements in MSG identification ability can be reversed fairly quickly. Both data sets demonstrate that human taste sensitivity can change within 10 days. Previously, we had designated the increased identification ability for MSG as "induction" (Kobayashi and Kennedy, 2002), but we did not know how transient or experience-dependent this gustatory plasticity might be.

The significance of the present results also is twofold. First, an experience-inducible increase in MSG taste identification ability was replicated using experimental and control treatment foods that were more similar in texture and flavor than in the previous study (Kobayashi and Kennedy, 2002), which had used shrimp crackers with MSG as the experimental treatment food and chocolate candies without MSG as the control treatment food. Thus, we eliminated a possibility that the effects found in the previous work were due to some unknown qualitative differences in the treatment foods unrelated to the MSG component. A second significance was that the enhanced MSG taste identification ability lasted for less than 10 days in the absence of exposure to MSG. This short-lived plasticity may be specific to the taste system because in the olfactory induction the androstenone-induced group remained sensitive for as long as 6 weeks (Wysocki *et al.*, 1989). However, to determine whether or not this type of rapid plasticity is specific to the taste system and/or the compound requires further studies that measure the temporal stability of plastic phenomena in other sensory systems and with other compounds.

A recent olfactory induction study found a significant difference between genders for the susceptibility to olfactory induction: women of reproductive age were induced more easily than men for a variety of odors (Dalton *et al.*, 2002). However, we found no such difference between the genders. Although there was a tendency for men to do better in MSG identification than women (as in Kobayashi and Kennedy, 2002), the difference was not significant. These results may reflect some difference in induction mechanisms between olfactory and taste systems—the olfactory system being more susceptible to gender effects than taste system. They may also reflect some difference in plasticity specific to the compound. Taste plasticity to MSG may be unique and less susceptible for gender effects than plasticity for other odor/taste compounds.

A possible confound in the present study is that we gave two subjects choices between the two treatment foods to ensure that they could continue snacking at least for 10 days. We did so because it is difficult for people to eat a food that they do not like. Nonetheless, for all of our subjects, the treatment foods were entirely new. Thus, it is unlikely that they chose which one to snack based on their absolute preferences (i.e., that they knew they liked the particular snacks). Moreover, even though the two people were both in the Control Group, they did at or better than average in identifying MSG for both phases (one person's MICs were 4 and 3, and the other person's MICs were 4 and 6 for the Phase I and Phase II, respectively).

As in our previous study (Kobayashi and Kennedy, 2002), there were a number of subjects who correctly identified the

lowest MSG concentrations but not the middle and/or the highest concentrations. We regarded these subjects as having guessed randomly when they chose between the pairs of solutions since the forced-choice procedure required that subjects choose one of the two solutions whether or not they were absolutely confident about their choices. However, the proportions of subjects who were correct for the lower concentration but incorrect for the highest and/or middle concentrations did not differ between the Control Group and the MSG Group (50% in each group) for each phase. These distributions suggest that subjects in both groups were equally confident in the choices they made during both testing phases.

It is possible that the dietary patterns of the subjects affected our results. The results of the survey suggest no major difference between the MSG-exposed and Control groups (Table 2). However, this absence of observed relations to diet cannot be conclusive because the questionnaire was neither descriptive nor asked precisely how often or how much food that presumably contained MSG or related compounds each subject consumed. It is known that glutamic acid (a component of MSG) is contained in some popular unprocessed foods in America, such as mushrooms and ripe tomatoes, as well as common cheeses (Yamaguchi, 1998; Halpern, 2000). Also, MSG is present in many commercially prepared and restaurant foods in America. We have no way of knowing how much and how often each subject ate those foods that contain glutamic acid or MSG during the 21- to 22-day period of our experiment. However, since our subjects were assigned to each group randomly (except the few subjects in the Control Group) and since they were all from similar socioeconomic backgrounds in the same area (Ithaca, NY), it seems that any effects from these uncontrolled variables were distributed more or less similarly among the subjects in the MSG Group and the Control Group.

The enhanced MSG identification ability in our present and previous studies may be due to changes in the peripheral taste mechanism. It has been suggested that olfactory induction coincides with the turnover cycle of the olfactory epithelium (Wysocki *et al.*, 1989). In support of a peripheral explanation, Wang *et al.* (2003) found that repeated exposure to androstenone led to an increase in amplitudes of evoked potentials from the nasal area. Alternatively, the enhanced MSG identification ability may be the result of some changes in brain mechanism or interactions between the peripheral and central mechanisms. It has been shown that cortical reorganization occurs after practices in tactile (Recanzone *et al.*, 1992) and auditory (Recanzone *et al.*, 1993) tasks in monkeys. More recently, a brain imaging study has shown that the tonotopic map in auditory cortex reorganizes after short periods of exposures to particular tones (Jäncke *et al.*, 2001). Furthermore, Faurion *et al.* (1998) found changes in activation of the periinsular cortical region that varied as the subjects acquired perceptual familiarity with taste stimuli. It is possible that a cortical rewiring is involved in the taste plasticity observed in our experiments.

In sum, several previous studies have reported enhanced olfactory and taste sensitivities after exposures to particular odors or tastes. However, it was not known whether and when the initial plasticity could be reversed. The present study is the first study to examine the temporal stability of this kind of sensitivity changes in a systematic way. Subjects who had exhibited an increase in their ability to identify MSG after exposure to MSG in food subsequently showed a decrease in their taste identification ability to MSG after 10 days of absence of such exposures to MSG. These results suggest that the taste sensory system can be tuned to the changing environment more rapidly than it had been thought previously. Future psychophysical studies could clarify whether this kind of gustatory plasticity can be generalized to other tastants or can be elicited and reduced over briefer time periods.

**Table 2** Dietary patterns of subjects: frequency of consuming oriental meals (e.g., Japanese, Chinese, or Vietnamese)

| | Number of subjects | | | |
|---|---|---|---|---|
| | Rarely | Once a month | Once a week | Almost everyday |
| Usually | | | | |
| MSG group | 0 | 6 | 4 | 10 |
| Control group | 0 | 5 | 5 | 10 |
| During Phase II | | | | |
| MSG group | 1 | 4 | 5 | 10 |
| Control group | 2 | 1 | 7 | 9 |

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