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Plasma Glutamate Concentrations in 1-Year-Old Infants and Adults Ingesting Monosodium L-Glutamate in Consommé[1]

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ABSTRACT. This study tested the hypothesis that infants metabolize glutamate more slowly than adults. Eight 1-yr-old infants ingested 160 ml of a beef consommé providing monosodium L-glutamate at 0, 25, and 50 mg/kg body weight. Plasma glutamate and aspartate concentrations were measured sequentially for the next 2 h. The results were compared to values noted in nine adult subjects ingesting equivalent doses of monosodium L-glutamate in consommé. In adults, mean (± SD) peak plasma glutamate concentrations were 5.59 ± 1.56, 10.2 ± 2.08, and 17.0 ± 8.06 μmol/dl, respectively; the area under the plasma glutamate concentration time curves were 96 ± 42, 257 ± 80, and 442 ± 303 μmol/dl × min, respectively. In infants, the mean (±SD) peak plasma glutamate concentrations were 6.94 ± 1.43, 10.6 ± 2.36, and 12.0 ± 1.16 μmol/dl, respectively; the plasma glutamate area under the curve values were 47 ± 28, 191 ± 85, and 358 ± 105 μmol/dl × min, respectively. The data indicate that the plasma glutamate concentration response in 1-yr-old infants ingesting MSG at these glutamate doses is no higher than values observed in adult subjects. (*Pediatr Res* 20: 53–58, 1986)

Abbreviations

AUC, area under the curve
MSG, monosodium L-glutamate
ANOVA, analysis of variance

The ability of human infants to metabolize MSG has been one area of concern in considering glutamate's safety as a food additive. Olney (1–3) postulated that human infants metabolized dicarboxylic amino acids more slowly than adults, and projected theoretical plasma glutamate concentrations for infants at much higher values than those for adults following glutamate ingestion.

The present study directly tested this hypothesis by measuring the plasma glutamate and aspartate response in 1-yr-old infants fed a beef consommé containing graded doses of MSG. The results of these studies were compared with previously reported (4) plasma glutamate and aspartate values in normal adults administered equivalent doses of MSG in the same consommé.

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MATERIALS AND METHODS

Eight infants (four male, four female) were studied. The proposed study was explained to at least one of the parents and informed, written consent was obtained. The protocol of the study was reviewed and approved by the Committee on Research Involving Human Subjects of the University of Iowa.

Each child ingested 160 ml of beef consommé providing 0, 25, and 50 mg/kg body weight of added MSG in a balanced crossover design. Each infant was randomly assigned to receive consommé providing either 0 or 25 mg MSG/kg body weight, followed by the other dose at least 1 wk later. After these two tests had been completed and the results evaluated, each infant ingested consommé providing MSG at 50 mg/kg body weight. All tests were begun at 0800 h following an overnight fast.

The monohydrate of monosodium L-glutamate was purchased from a local grocery store (Accent International, Pet Incorporated, St. Louis MO). The soup was prepared from a special beef consommé base supplied by the Ajinomoto Company (Tokyo, Japan). The composition of the soup base is shown in Table 1. As noted in Table 1, 160 ml of reconstituted soup contained 37 mg of free glutamate, for a mean dose of 4.1 mg/kg body weight. Thus, the true final doses of MSG studied in these infants were 4.1, 29.1 and 54.1 mg/kg body weight.

Each infant was assigned by random allocation into one of two equal groups that differed with respect to time of blood sampling. Since only four blood samples were obtained from each infant after dosing, two different sampling schedules were utilized so as to obtain samples during the entire absorptive period. Blood samples at each dose level were obtained from four infants at 0, 30, 60, and 120 min After ingestion of the consommé. Blood samples from the other four infants were obtained at 0, 15, 45, and 90 min.

Blood samples were obtained by heel skin puncture using the precautions described by Stegink *et al.* (5) for appropriate collection of capillary blood. These samples were processed and analyzed as previously described (4).

Values for infants were compared with values previously obtained for normal adult subjects (4). In the latter study, nine adult subjects (five male, four female) were studied in a Latin square design (three subjects/cell) using three different servings of the same beef consommè. The consommè was administered at a level of 4.2 ml/kg body weight and provided added MSG at 0, 25, and 50 mg/kg body weight. Blood samples for amino acid analyses were collected at 0, 15, 30, 45, 60, 90, 120, 150, 180, and 240 min after ingestion of the consommé.

Statistical analyses were carried out using the paired t test and ANOVA (6, 7). Results were assessed for statistical significance both before and after application of the Bonferroni correction (8). The Bonferroni procedure is a simple method for controlling type I error (differences due to chance alone) during multiple

Table 1. *Composition of beef consommé soup used**

Component	% dry wt
Crude protein	6.4
Fat	11.3
Carbohydrate	22.5
Ash	56.0†
Water	1.6
Free glutamate	1.2

* Soup base reconstituted at a level of 1.94 g base/100 ml hot water. Soup reconstituted at this level provides 4.1 mg MSG/kg body weight and 77 mg carbohydrate/kg body weight when 160 ml is fed to a 9.1 kg infant. Adult subjects ingested 4.3 ml of reconstituted soup per kg body weight. This level provides approximately 1 mg MSG/kg body weight and 18.8 mg carbohydrate/kg body weight.

† 54.4% of this sodium chloride.

application of the *t* test to a set of data (8). Plasma amino acid values at each time point following the loading dose were compared to zero time values. The area under the plasma time-concentration curve for each amino acid was calculated using the summation of the trapezoidal areas between plasma values at successive time points. The base of each trapezoid was set as the normal fasting level for that amino acid. The AUC is expressed as μmol/dl × min.

RESULTS

Table 2 lists infant weight, age, and the time required for the infants to ingest the soup. The age and weight of infants ingesting MSG at 0 and 25 mg/kg body weight were similar, as expected. Because the 50 mg/kg body weight dose of MSG was studied last, the infants were older and larger.

The mean time of soup ingestion by infants was 18 min and did not vary with the dose of MSG. This was slower than the ingestion rate observed in adults, where only 7 min were needed to drink the soup.

Plasma glutamate and aspartate concentrations in infants and adults ingesting soup without added MSG are shown in Table 3. Baseline values for both glutamate and aspartate were significantly lower ($p < 0.005$ ANOVA, Bonferroni correction applied) in adults than in infants. The reason for this difference is unknown, but it has been observed in previous studies with other nitrogen sources (9, 10). The ingestion of consommè without added MSG increased slightly, but not significantly, mean plasma glutamate concentrations over baseline values in both infants and adults. The peak increase over baseline plasma glutamate concentration was slightly, but not significantly, larger in adults (+1.9 μmol/dl) than in infants (+0.7 μmol/dl). Similarly, the area under the 2-h plasma glutamate concentration-time curve (AUC) in adults was slightly, but not significantly, higher than the 2-h AUC value in infants. No significant change in plasma aspartate concentration was noted after ingestion of soup without added MSG in either infants or adults. The peak increase over the baseline plasma aspartate concentration was slightly higher in infants (+0.29 μmol/dl) than adults (+0.04 μmol/dl) as was the AUC value; neither difference was statistically significant.

Plasma glutamate and aspartate concentrations in infants and adults ingesting consommé providing added MSG at 25 mg/kg body weight are shown in Table 4. Glutamate ingestion significantly increased mean (± SD) plasma glutamate concentration in adults from a baseline value of 3.93 ± 1.10 μmol/dl to a peak value of 10.2 ± 2.08 μmol/dl 30 min after dosing. A similar response was noted in infants, where the mean plasma glutamate concentration increased from a baseline value of 7.22 ± 1.19 μmol/dl to a peak value of 10.6 ± 2.37 μmol/dl 15 min after ingestion. The increase in infants was significant using paired t-test analysis ($p < 0.05$), but was not significant when the data were analyzed by ANOVA. The increase over the baseline plasma

Table 2. *Age, wt, and time required to ingest soup of participating infants (mean ± SD)*

Group (mg/kg)	Age (days)	Wt (kg)	Time required to drink soup (min)
0	305 ± 55	9.10 ± 1.18	18.5 ± 6.8
25	299 ± 54	9.08 ± 1.17	16.6 ± 4.2
50	317 ± 61	9.28 ± 1.29	19.0 ± 7.1

Table 3. *Plasma glutamate and aspartate concentrations in young infants and adults ingesting soup with no added MSG*

Time (h)	Plasma glutamate		Plasma aspartate	
	Infants	Adults*	Infants	Adults*
0	6.24 ± 1.52†	3.69 ± 1.08‡	1.25 ± 0.36†	0.70 ± 0.50‡
0.25	6.54 ± 0.71	5.59 ± 1.56	1.37 ± 0.34	0.74 ± 0.62
0.5	6.94 ± 1.43	5.44 ± 1.15	1.53 ± 0.41	0.71 ± 0.45
0.75	6.11 ± 0.82	5.36 ± 1.57	1.54 ± 0.32	0.69 ± 0.60
1.0	6.52 ± 1.85	4.86 ± 1.49	1.18 ± 0.52	0.65 ± 0.44
1.5	6.15 ± 1.10	4.26 ± 1.28	1.29 ± 0.43	0.68 ± 0.50
2.0	5.53 ± 1.50	4.13 ± 1.77	1.32 ± 0.49	0.63 ± 0.51
AUC§	48.6 ± 28.3	94.5 ± 41.5	6.83 ± 0.30	−2.18 ± 1.35

* Data in adults taken from Reference 3.

† Data expressed as mean ± SD in μmol/dl; $n = 8$ at zero time and $n = 4$ at all other time points.

‡ Data expressed as mean ± SD in μmol/dl; $n = 9$ at all time points.

§ Expressed as mean ± SD in μmol/dl × min.

glutamate value was slightly higher in adults (+6.27 μmol/dl) than in infants (+3.38 μmol/dl) but the difference was not statistically significant. Similarly, the plasma glutamate AUC value in adults (257 ± 79.6 μmol/dl × min) did not differ significantly from the value in infants (191 ± 84.7 μmol/dl × min). Plasma aspartate concentration increased slightly, but not significantly, in both infants and adults. In adults, ingestion of consommé providing 25 mg/kg body weight of added MSG increased mean plasma aspartate concentrations significantly (ANOVA) from a baseline value of 0.66 ± 0.53 μmol/dl to a peak value of 1.12 ± 0.70 μmol/dl 30 min after ingestion. Mean plasma aspartate concentration also increased in infants, but the change was not statistically significant. The peak increases in plasma aspartate concentration over baseline values in adults (+0.46 μmol/dl) and infants (+0.61 μmol/dl) were not significantly different nor were the plasma AUC values.

Plasma glutamate and aspartate concentrations in infants and adults ingesting soup providing 50 mg/kg body weight added MSG are shown in Table 5. In adults, mean (± SD) plasma glutamate concentration increased significantly from a baseline value of 3.77 ± 0.80 μmol/dl to a peak value of 17.0 ± 8.06 μmol/dl 30 min after soup ingestion. In infants the mean plasma glutamate concentration increased significantly from a base line value of 6.71 ± 1.14 μmol/dl to a peak value of 12.0 ± 1.16 μmol/dl 15 min after soup ingestion. Although the mean peak increase over baseline concentration in adults (+13.2 μmol/dl) was significantly greater ($p < 0.005$, ANOVA, Bonferroni correction applied) than in infants (+5.29 μmol/dl), plasma glutamate AUC values did not differ significantly. Ingestion of consommé providing 50 mg/kg body weight MSG produced only small changes in plasma aspartate concentration. In adults, mean (± SD) plasma aspartate concentration increased significantly ($p < 0.005$, ANOVA, Bonferroni correction applied) from a baseline value of 0.43 ± 0.19 μmol/dl to a peak value of 1.40 ± 0.71 μmol/dl 30 min after dosing. In infants, the increase in the mean plasma aspartate concentration was smaller and not statistically significant. The increase over the mean baseline value was similar in adults (+0.97 μmol/dl) and infants (+0.70 μmol/dl), as were

Table 4. *Plasma glutamate and aspartate concentrations in infants and adults ingesting soup with 25 mg/kg body wt added MSG*

Time (h)	Plasma glutamate		Plasma aspartate	
	Infants	Adults*	Infants	Adults*
0	7.22 ± 1.19†	3.93 ± 1.10‡	1.50 ± 0.35†	0.66 ± 0.53‡
0.25	10.6 ± 2.37§	7.37 ± 2.24§,‖,¶,**	1.85 ± 0.62	0.74 ± 0.41
0.5	10.2 ± 2.36§	10.2 ± 2.08§,‖,¶,**	1.53 ± 0.48	1.12 ± 0.70§,‖,¶,**
0.75	9.90 ± 2.81	6.77 ± 1.88§,‖	2.11 ± 0.62	0.89 ± 0.55
1.0	6.83 ± 1.31	5.73 ± 1.65	1.70 ± 0.78	0.71 ± 0.50
1.5	7.68 ± 1.97	4.37 ± 1.43	1.69 ± 0.19	0.78 ± 0.50
2.0	6.11 ± 1.21	4.91 ± 2.02	1.33 ± 0.15	0.73 ± 0.49
AUC††	191 ± 84.7	257 ± 79.6	32.7 ± 23.9	24.1 ± 1.12

* Data in adults taken from Reference 3.
† Data expressed as mean ± SD in μmol/dl; $n = 8$ at zero time and $n = 4$ at all other time points.
‡ Data expressed as mean ± SD in μmol/dl; $n = 9$ at all time points.
§ Values differed significantly ($p < 0.05$, paired t test) from baseline values.
‖ Values differ significantly ($p < 0.005$, ANOVA, Bonferroni correction applied) from baseline values.
¶ Values differ significantly ($p < 0.05$, paired t test) from values when MSG is not added.
** Values differ significantly ($p < 0.005$, ANOVA, Bonferroni correction applied) from values when MSG is not added.
†† Expressed as mean ± SD in μmol/dl × min.

Table 5. *Plasma glutamate and aspartate concentrations in infants and adults ingesting soup with 50 mg/kg body wt added MSG*

Time (h)	Plasma glutamate		Plasma aspartate	
	Infants	Adults*	Infants	Adults*
0	6.71 ± 1.14†	3.77 ± 0.80‡	1.59 ± 0.78†	0.43 ± 0.19‡
0.25	12.0 ± 1.16§	8.09 ± 4.26§,‖,¶,**	2.05 ± 0.57	0.75 ± 0.41§
0.5	11.5 ± 2.17§	17.0 ± 8.06§,‖,¶	1.57 ± 0.49	1.40 ± 0.71§,‖,¶,**
0.75	11.3 ± 2.05§	9.88 ± 4.03§,‖,¶	2.29 ± 0.67	0.70 ± 0.40§
1.0	9.23 ± 2.93	6.78 ± 2.76§,‖	1.91 ± 0.80	0.73 ± 0.50
1.5	8.10 ± 1.93	4.39 ± 1.87	1.93 ± 0.66	0.50 ± 0.37
2.0	5.83 ± 0.89	3.88 ± 1.94	1.20 ± 0.24	0.36 ± 0.19
AUC††	358 ± 105	442 ± 303	52.1 ± 12.0	31.2 ± 26.6

* Data in adults taken from Reference 3.
† Data in infants expressed as mean ± SD in μmol/dl; $n = 8$ at zero time and $n = 4$ at all other time points.
‡ Data in adults expressed as mean ± SD in μmol/dl; $n = 9$ at all time points.
§ Values differ significantly from baseline values, $p < 0.05$, paired t test.
‖ Values differ significantly ($p < 0.005$, ANOVA, Bonferroni correction applied) from baseline values.
¶ Values differ significantly ($p < 0.005$, ANOVA, Bonferroni correction applied) from values when MSG was not added.
** Values differ significantly ($p < 0.005$, ANOVA, Bonferroni correction applied) from values when MSG is ingested at 25 mg/kg body wt.
†† Expressed as mean ± SD in μmol/dl × min.

the AUC values. Neither value differed significantly between infants and adults.

Erythrocyte glutamate and aspartate concentrations were also measured in these infants. No significant increase in erythrocyte glutamate or aspartate concentration over baseline value was noted after any glutamate dose. Thus, despite elevations in plasma glutamate concentrations after MSG loading, erythrocyte levels of glutamate and aspartate were unchanged.

DISCUSSION

Our data demonstrate similar plasma glutamate and aspartate concentration responses in infants and adults ingesting equal doses of glutamate on a mg/kg body weight basis in a beef consommè. The mean plasma glutamate concentration response to glutamate loading at equivalent doses appears to be slightly less in infants than adults. At each glutamate dose, mean infant plasma AUC values for glutamate are lower than mean adult values. Similarly, at each dose of MSG, the mean peak increase over baseline for plasma glutamate is higher in adults than in infants.

The difference in plasma glutamate response to glutamate ingestion between adults and infants may reflect the more rapid rate of soup ingestion in adults, rather than an age-related phenomenon. The average time required for adults to ingest the soup was 7 min, while the value in infants was 18 min.

The more rapid clearance of glutamate from the blood by infants than by adults is consistent with other data we have obtained. Normal 1-yr-old infants appear to handle the methionine content of both N-acetyl-L-methionine and L-methionine more rapidly than adults administered identical doses on a mg/kg body weight basis (11). Similarly, infants administered aspartame (L-aspartyl-L-phenylalanine methyl ester) at 100 mg/kg body weight clear aspartame's phenylalanine content from the blood somewhat more rapidly than adults (10).

The results of the present study are also consistent with our earlier data indicating good utilization of glutamate by term and premature infants (9, 12–14). However, in all of these earlier studies glutamate was administered in food that also contained large amounts of carbohydrate. We have recently shown that metabolizable carbohydrate appears to facilitate glutamate metabolism in both adult humans (15–17) and infant pigs (18). The net effect of simultaneous carbohydrate ingestion is a marked reduction in the plasma glutamate concentration increase noted

following a fixed dose of MSG. Byun and Kim (19) also presented data consistent with the hypothesis that carbohydrate affects plasma glutamate concentration after glutamate ingestion.

Tung and Tung (20) reported data that could be interpreted to suggest that infants handle glutamate less well than adults. They administered MSG at 150 mg/kg body weight to premature and term infants (dissolved in infant formula) and adult subjects (porridge meal), reporting mean (± SD) peak plasma glutamate levels of 24 ± 5 μmol/dl in infants and 12.5 ± 4 μmol/dl in adults. However, since the carbohydrate content of the food administered with glutamate varied between groups (formula for infants and porridge for adults), the plasma glutamate levels are not directly comparable. Further, the plasma glutamate levels observed by Tung and Tung (20) in infants and adults were significantly lower than values we observed in normal adults administered 150 mg/kg body weight of MSG in water, where peak plasma glutamate levels reached 59.4 ± 46.5 μmol/dl (16). Thus, the data of Tung and Tung (20) may be explained by the effect of carbohydrate on glutamate metabolism.

In the present study, MSG was ingested in a consommè providing very little carbohydrate. Infants received approximately 700 mg of carbohydrate when ingesting 160 ml of the soup, or about 77 mg/kg body weight for a 9.1 kg infant. Because of the difference in volume administered, adult subjects ingested less carbohydrate (approximately 19 mg of carbohydrate per kg body weight). This small amount of carbohydrate is unlikely to have a detectable effect on the plasma glutamate response in either infants or adults (17).

The quantities of MSG administered in this study represent a reasonable test of the expected intake of MSG from foods likely to be ingested by the young child. According to the Committee on GRAS List Survey—Phase III (21) the 99th percentile of expected daily intake of MSG by the 6–11-month-old infant is 36 mg/kg body weight, while that for the 11- to 23-month-old child is 43 mg/kg body weight. The 99.9th percentiles for these two groups are 46 and 61 mg/kg body weight, respectively. Thus, the 50 mg/kg body weight dose of MSG studied approximated a value between the 99 and 99.9th percentile of projected daily intake. Olney (1–3) had predicted a marked difference in the plasma glutamate response between infants and adults at an MSG dose of 50 mg/kg body weight.

Since we wished to avoid the confounding variable of carbohydrate's effect on glutamate metabolism, we chose a soup that contained as little carbohydrate as possible. Such consommè when sold commercially usually contains added MSG at levels varying from 0.15 to 0.30 g/100 ml as eaten. The consommè used in this study provided MSG at these levels. A 9.1 kg infant received a 0.165% solution of MSG when ingesting MSG at 25mg/kg body weight dose and received a 0.308% solution when ingesting MSG at 50 mg/kg body weight dose.

However, it is possible for infants to receive larger amounts of MSG from soups than the doses investigated in this study. A Consumer Reports review (22) indicates that the MSG content of an average serving of soup reconstituted from commercially available dry soup bases is 735 mg, while soup with the largest amount of MSG provides 1300 mg per serving. A 9.1 kg 1-yr-old ingesting an entire serving of soup providing an average amount of MSG would receive 81 mg/kg body weight, while an infant ingesting an entire serving of soup providing the largest amount of MSG would receive 143 mg/kg body weight. Infants ingesting restaurant soups may also receive higher doses of MSG. Olney (1–3) has suggested (without presenting supporting data) that restaurant soups may contain as much as 5 g of glutamate per serving. Conacher *et al.* (23) measured the MSG content of wonton soup from five restaurants, reporting a mean (± SD) value of 1092 ± 221 mg/100 ml. A 9.1 kg infant ingesting 6 oz on average of wonton soup would receive 216 mg MSG/kg body weight.

The use of glutamate as a food additive has been controversial. Like other dicarboxylic amino acids, glutamate exerts toxic effects when administered at very high doses, although species and age susceptibility vary considerably. Neonatal rodents (24–28) and cats (29, 30) administered large doses of glutamate develop hypothalamic neuronal necrosis. There is disagreement over the ability of glutamate to produce neuronal necrosis in infant nonhuman primates. Olney and colleagues (31, 32) originally reported that large doses of glutamate given to neonatal nonhuman primates produced hypothalamic neuronal necrosis. However, four other laboratories were unable to find lesions in infant monkeys given equally large doses of MSG (33–43). The failure of these groups to observe lesions in neonatal nonhuman primates did not reflect a failure of the glutamate load to produce elevated plasma glutamate concentrations (37, 42). Furthermore, the latter investigators had no difficulty in producing the glutamate-induced lesion in infant rodents.

The critical question, however, is how these data relate to humans. The neonatal human probably resembles the neonatal monkey to a greater extent than the infant mouse. Thus, the negative findings noted in monkeys after administration of MSG by most investigators support its safety as a food additive. However, the controversy over the sensitivity of the infant nonhuman primate to MSG led us to explore additional ways of evaluating the potential neurotoxicity of MSG.

Although the neonatal mouse is sensitive to large doses of glutamate and aspartate, it tolerates substantial doses of these amino acids without developing neuronal necrosis. Doses of aspartate or glutamate that produce plasma glutamate plus aspartate concentrations lower than 60 μmol/dl (6 to 10 times normal) do not produce lesions in the infant rodent (44–49). Lesions are noted only when very large doses of glutamate or aspartate are administered and plasma glutamate plus aspartate concentrations exceed 60 to 100 μmol/dl. Thus, plasma glutamate plus aspartate levels that are not harmful to infant mice should not be harmful to humans.

When MSG is given in water, the lowest dose producing lesions in infant mice is 500 mg/kg body weight (24–26). However, MSG doses of 500 mg/kg body weight or larger do not produce neuronal lesions when MSG is ingested with food (50, 51), a finding that is probably related to the effect of dietary carbohydrate on plasma glutamate concentration.

Infants ingesting 50 mg MSG/kg body weight in consommè had a mean peak plasma glutamate plus aspartate concentration of 14.05 μmol/dl, a level that would not be considered potentially harmful to even the neonatal mouse. It is not clear what the plasma glutamate concentration response would be in infants ingesting soup providing larger quantities of MSG. If infants and adults metabolize glutamate equally well at higher doses of MSG than those studied in this report, data obtained in adults can be used to predict values in infants. Such regression curves based on adult data (4) indicate that an MSG dose of 150 mg/kg body weight, ingested in consommè, bouillon, or tomato juice, would result in a mean peak plasma glutamate concentration of 50 to 60 μmol/dl. This projected plasma glutamate plus aspartate concentration (50 to 60 μmol/dl) approaches the threshold value associated with toxicity in the infant mouse, but is still far below the no effect level of 450 μmol/dl observed in the infant nonhuman primate (37, 42). If, on the other hand, infants metabolize larger doses of MSG more slowly than adults, the plasma glutamate concentration response will be larger in infants than in adults. However, the precise response is difficult to predict since no human studies suggest such an effect. Studies in infant and adult nonhuman primates administered 500 mg MSG/kg body weight indicate that the mean peak plasma glutamate concentration in infant monkeys is twice as high as the value in adults (45). If this factor is applied to humans, such projections indicate that infants ingesting 150 mg MSG/kg body weight might have a mean peak plasma glutamate concentration of 100 to 120 μmol/dl. This putative plasma glutamate concentration would

exceed the threshold value associated with toxicity in the infant mouse, but would still be within the no effect plasma glutamate range in the infant nonhuman primate (37, 42).

Attempts to predict the peak plasma glutamate plus aspartate concentration produced by ingestion of commercial soups containing large amounts of glutamate are complicated by the fact that such soups contain significant amounts of carbohydrate. Thus, mean peak plasma glutamate concentration should be lower than 50 to 60 μmol/dl when humans ingest soup that provides 150 mg/kg body weight MSG and contains carbohydrate [see for example the data of Tung and Tung (20)]. However, the soup must contain significant amounts of carbohydrate to produce a lower plasma glutamate concentration. Our data in adults suggest that the carbohydrate effect on glutamate metabolism is only noted when the carbohydrate dose exceeds 0.25 g/kg body weight (17).

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ANNOUNCEMENTS

First I.U.I.S. Conference on Clinical Immunology

A 2-day conference on clinical immunology will be held in conjunction with the VIth International Congress of Immunology, in Toronto, Canada, from July 5–6, 1986. These dates immediately precede the VIth International Congress. The conference will encompass plenary sessions, minisymposia, and a poster session. Among the major topics: receptor-anti-receptor mediated diseases; new trends in the management of disorders of autoimmunity, immunodeficiency, allergy, immunodiagnostic techniques, and transplantation; and a session concerned with human diseases caused by lymphotropic retroviruses.

Registration and abstract forms may be obtained from: Mr. K. Charbonneau, National Research Council of Canada, Ottawa, Ontario K1A OR6, Canada.

10th International Convocation on Immunology
Buffalo, NY, July 14–17, 1986
Vaccines: New Concepts and Developments

The Ernest Witebsky Center for Immunology will present this symposium in its regular biennial series at the Hyatt Regency Buffalo Hotel following the VIth International Congress of Immunology in Toronto, Canada, which is only 100 miles distant. Closed plenary sessions will focus on the topics: Conceptual Basis of Antigens; Antigen Identification and Purification; Host Response; Production of Vaccines by Recombinant DNA Techniques: Idiotype Vaccines; and Human and Veterinary Vaccines. Open poster sessions for free contributions on the theme will be offered.

For further information contact: Dr. James F, Mohn, Director, The Ernest Witebsky Center for Immunology, 210 Sherman Hall, State University of New York at Buffalo, Buffalo, NY 14214 (716-831-2848).