

## Letter to the Editor

# Monosodium glutamate is not associated with obesity or a greater prevalence of weight gain over 5 years: findings from the Jiangsu Nutrition Study of Chinese adults – comments by Samuels

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The recent study by Shi *et al.*<sup>(1)</sup> has serious logical and methodological flaws.

In 1969, Olney<sup>(2)</sup> demonstrated that monosodium glutamate (MSG) administered to neonatal mice produced brain lesions in the area of the hypothalamus. Those brain lesions were followed by obesity and other endocrine disorders that manifested as treated animals approached maturity<sup>(2)</sup>. Subsequent feeding studies wherein MSG was fed to laboratory animals were shown to have the same effects<sup>(3,4)</sup>.

The fact of MSG toxicity, its role as an endocrine disrupter, and its role as a trigger for adverse reactions such as migraine headache, asthma and heart irregularities have been hotly contested by the glutamate industry which points to a collection of badly flawed studies, each with negative results<sup>(5,6)</sup>, as substantiation for the claim that MSG poses no substantial risk to humans. In the most recent example of research with conclusions that cast doubt on the relationship between MSG and adverse reactions or, as in this case, between MSG intake and obesity<sup>(1)</sup>, Shi *et al.*<sup>(1)</sup> concluded that their findings 'indicate that when other food items or dietary patterns are accounted for, no association exists between [monosodium glutamate] intake and weight gain'.

The intent of the Shi *et al.* study<sup>(1)</sup>, as stated in the abstract, was '...to investigate a possible association between MSG intake and obesity, and determine whether a greater MSG intake is associated with a clinically significant weight gain over 5 years'. However, the subject of association between MSG intake and weight or obesity was entirely ignored. All statistical analyses reported were for MSG intake in 2002 and weight change between 2002 and 2007. There were no analyses reported for an association between MSG intake and weight in 2002, and it would appear that there were no data collected on MSG intake in 2007.

The question of the relationship between MSG intake and weight gain (defined as a change in weight over a 5-year period  $\geq 5\%$ ) is interesting, but says nothing about MSG intake causing or not causing obesity. Considering change in weight (as defined as a change in weight over a 5-year period  $\geq 5\%$ ) without considering and controlling for change in MSG intake is inappropriate as is using 2002 MSG intake figures without using 2007 MSG intake figures. Thus, drawing conclusions about the relationship between MSG intake and weight gain is unacceptable.

Finally, it must be pointed out that the design of the Shi *et al.* study<sup>(1)</sup> facilitated the production of negative results. The authors did not look at the association between MSG

intake and weight, but chose instead to look at weight gain, and only weight gain that was clinically significant ( $\geq 5\%$ ). The authors used non-parametric statistics as well as weight change calculations adjusted for variables of height, weight, dietary intake, dietary patterns and lifestyle factors such as use of motorised transportation *v.* walking or bicycling, daily leisure-time physical activity, and daily time spent on sedentary activities, which would have diminished the power of the statistical analyses used as compared with use of parametric statistics and unadjusted (or less adjusted) weight change. It would also appear to be relevant to the production of negative results that their regression analyses were not adjusted for urban/rural differences which had a significant negative relationship with MSG intake in 2002, while the regression analyses were adjusted for leisure time, physical activity, and alcohol intake which were not significantly related to MSG intake (Table 1 of Shi *et al.*<sup>(1)</sup>).

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