How I know what I know about the obesity epidemic

In 1969 Dr. John Olney demonstrated that the glutamic acid (GLU) component of monosodium glutamate (MSG) administered in high doses to mice caused brain damage in various parts of the brain including the arcuate nucleus of the hypothalamus which was followed as adults by stunted skeletal development, marked obesity, and female sterility. In the decade that followed, Olney’s work was replicated, and challenges to his findings were dismissed by all except those employed by the glutamate industry.

MSG is a flavor-enhancing food additive added to processed food that some claim only causes transient adverse reactions in a small set of people sensitive to the substance. Others maintain that it causes adverse reactions ranging from simple skin rash to migraine headache, heart irregularities, seizures and anaphylactic shock. There is no question, however, that ingestion of MSG causes adverse reactions.

Although known to cause both brain damage and adverse reactions, there is no restriction imposed by the FDA on the amount of either MSG or GLU that a single food ingredient may contain.

Although I knew a great deal about the toxic effects of MSG, it was only as people began reporting adverse reactions following ingestion of food that did not contain MSG, that I realized it was the GLU in MSG -- and in flavor enhancers other than MSG -- that was causing what consumers were calling “MSG reactions.”

Sometime around 2016, I began thinking about obesity and reading about the adverse effects of ultra-processed foods. I realized that because ultra-processed foods were made of cheap food and chemicals, ultra-processed foods would contain flavor-enhancers to compensate for lack of flavor, with all of those flavor-enhancers containing excitotoxic GLU.

And I began to think about the unexplained obesity epidemic. Knowing that glutamate fed in large amounts to animals with immature brains causes brain damage followed by gross obesity, and that the brains of fetuses and neonates are vulnerable to glutamate insult, I realized that if glutamate in large amounts could be “fed” to human fetuses and neonates, brain damage would follow. It had occurred to me that if a pregnant woman
consumed free glutamate in excess of what she needed for normal body function, the excess, which would be excitotoxic, would be passed to her fetus through the placenta and/or to her infant while nursing.

Glutamate is a Jekyll and Hyde amino acid. When present in protein or released from protein in a regulated fashion (through routine digestion) glutamate is vital for normal body function. It is the principal neurotransmitter in humans, carrying nerve impulses from glutamate stimuli to glutamate receptors throughout the body. It becomes toxic only when present in greater quantity than a healthy human needs for normal body function. Then, as an excitotoxic neurotransmitter, it fires repeatedly damaging targeted glutamate-receptors and/or causing neuronal and non-neuronal death by over exciting those glutamate receptors until their host cells die.

I also realized that with the proliferation of processed and ultra-processed foods, there is sufficient free glutamate in food to provide the “excess” glutamate needed to cause the glutamate ingested by pregnant women to become excitotoxic – brain damaging -- if more than one glutamate-containing ingredient is consumed during the course of a day.

From my research I knew that there is nothing to prevent ingested glutamate from entering the brains of immature beings. The blood-brain-barrier (BBB) is not fully developed in either the fetus or the newborn. But more to the point, the arcuate nucleus, which is the area of the brain where brain damage fosters obesity, is a circumventricular organ. Circumventricular organs lie outside the BBB. They are not impervious to glutamate-induced brain damage.

And I knew two other things. I had read the industry-sponsored studies done in the 1970s that were said to be failed replications of studies demonstrating glutamate-induced brain damage -- studies in which methods and materials were varied from the originals enough to guarantee that no traces of glutamate-induced brain damage would be found.

I also knew that in 1957 the manufacture of MSG and GLU was changed from a slow and costly method calling for extraction of GLU from a protein source to a method called bacterial fermentation that allowed virtually unlimited amounts of MSG and GLU to be produced. The obesity epidemic
only happen after essentially unlimited amounts of GLU could be found in processed food.

There are five pieces to the puzzle of the obesity epidemic: the concept of excitotoxicity with glutamate being the principal excitotoxin; the fact of glutamate-induced brain damage followed by obesity; the abundance of excitotoxic glutamate in processed and ultra-process food; the fact that pregnant females can pass excitotoxic glutamate to their fetuses; and the correlation between the time that virtually unlimited amounts of free glutamate became available in food and the beginning of the obesity epidemic. I accounted for them all.

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