The growing prevalence of obesity in the United States is among the major concerns of public health practitioners. It is a problem that reflects the over-consumption of fats and sugars in the population. While it is well known that oral effects of sugars (e.g. taste, odor and texture) stimulate intake, recent evidence has shown that post-oral effects of sugars (e.g. nutritional feedback) stimulate intake as well. I examined the interaction between oral and post-oral effects of sweets on intake in an animal model system of sugar intake: C57bL/6J mice. My overall strategy was to compare ingestive responses of mice to a variety of caloric and non-caloric sweet solutions during short and long-term tests. The underlying assumption is that intake during short-term tests is driven solely by oral factors while intake during long-term tests is driven by both oral and post-oral factors. Any discrepancy between the responses to each type of test therefore reflected the contribution of post-oral factors. I offered mice several solutions: water, saccharin (38 mM), glucose (170 mM, 250 mM and 330 mM) and mixtures of saccharin and glucose. I found that the ingestive response to water, saccharin, 170 mM glucose, and saccharin+glucose (170mM) was similar during the short-term and long-term tests. This indicates that the long-term intake of these solutions is primarily controlled by oral factors. The ingestive response to the more concentrated glucose solution differed between the short-term and long-term tests; the mice consumed disproportionately greater volumes of glucose during the long-term tests. This suggests that post-oral factors greatly contributed to mice sugar intake in the long-term tests. These findings are clear evidence of the contributions of post-oral factors to sweetener intake and expand our current understanding of the obesity problem as an epidemic not merely driven by oral stimulation, but post-oral stimulation as well.