Understanding the basis for individual variation in sweetener intake has become increasingly pertinent as health problems associated with the overconsumption of foods rise. Our research addresses individual variation in sweetener intake among humans by focusing on a parallel phenomenon among inbred mouse strains. Two groups of inbred mouse strains, tasters and nontasters, which possess different alleles of the sweet taste receptor protein gene, *Tas1r3*, have been found to vary in long-term sweetener intake. When given a choice between water and a sweetener over 48 hrs, nontasters consume significantly less sweetener than tasters. The current explanation for this taster/nontaster difference is that due to their *Tas1r3* allele, nontasters have a dysfunctional sweet taste receptor that diminishes their peripheral gustatory response to sweeteners. As a result, nontasters consume sweeteners less avidly than tasters do. To evaluate this explanation, I tested the following hypothesis: if the strength of the peripheral gustatory response determines sweetener intake, then taster strains should lick more vigorously for sweeteners than nontaster strains. Using a 1 min preference test, I evaluated the short-term licking responses of 3 taster strains (FVB, SWR, B6), and 3 nontaster strains (129, C3H, DBA) to a range of sucrose and SC45647 (an artificial sweetener) concentrations. I found large variation in licking responses within the taster and nontaster strains and furthermore, that 2 nontaster strains (DBA, 129) licked more vigorously than the taster strain, B6, for the majority of concentrations for both sucrose and SC45647. That taster/nontaster status did not assort with short-term licking responsiveness suggests that differences in sweetener intake cannot be explained by allelic variation in *Tas1r3* alone.