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The Influence of *Tas1r3* Polymorphism on Short-term Sweetener Consumption in 6 Mouse Strains

Some mouse strains consume significantly greater amounts of 0.3M sucrose over a 24-hour period than do other mouse strains. This variation in sweetener consumption has been explained by allelic variations in the *Tas1r3* gene, which encodes for T1R3, a key protein of a G-protein-coupled, sweet-taste receptor called T1R2/T1R3. The current model for how *Tas1r3* could mediate strain differences in long-term sweetener consumption is as follows: alternate alleles of *Tas1r3* encode two forms of the receptor protein; taster strains express a relatively high-affinity receptor, while nontaster strains express a low-affinity receptor. According to this model, tasters should produce a stronger peripheral nerve response to and, consequently, consume more sweeteners. If the taster/nontaster difference in long-term consumption is mediated by differences in peripheral taste responsiveness, then we predicted that taster strains should lick more vigorously from sweeteners in a short-term licking test. To evaluate this prediction, I used a 1-minute preference test to examine licking responsiveness to two sweeteners (sucrose and SC45647, a non-caloric sweetener) in 3 taster (SWR, B6 and FVB) and 3 nontaster (DBA/2, 129/B3 and C3H) strains. I found that, even though sensitivity to sweeteners appear to assort with taster/nontaster status, licking responsiveness to sweeteners did not. For example, one of the strains that licked most vigorously was DBA, a nontaster, and one of the strains that licked least vigorously was B6, a taster. My results indicate that peripheral taste responsiveness to sweeteners alone cannot account for strain differences in long-term consumption of sucrose. A model providing a more comprehensive explanation of how additional ingestive mechanisms mediate long-term sweetener consumption is needed.