

## Administration of Exendin-4 but not CCK alters lick responses and trial initiation to sucrose and intralipid during brief-access tests

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### Abstract

Administration of cholecystokinin (CCK) or the glucagon-like peptide 1 (GLP-1) receptor agonist Exendin-4 (Ex-4) reduces food intake. Findings in the literature suggest CCK reduces intake primarily as a satiety signal whereas GLP-1 may play a role in both satiety and reward-related feeding signals. Compounds that humans describe as *sweet* and *fatty* are palatable yet are signaled via separate transduction pathways. Here, unconditioned lick responses to sucrose and intralipid were measured in a brief-access lick procedure in food-restricted male rats in response to i.p. administration of Ex-4 (3 h before test), CCK (30 min before test), or a combination of both. The current experimental design measures lick responses to water and varying concentrations of both sucrose (0.03, 0.1, and 0.5 M) and intralipid (0.2%, 2%, and 20%) during 10-s trials across a 30-min single test session. This design minimized postingestive influences. Compared with saline-injected controls, CCK (1.0, 3.0, or 6.0  $\mu\text{g}/\text{kg}$ ) did not change lick responses to sucrose or intralipid. Number of trials initiated and lick responses to both sucrose and intralipid were reduced in rats injected with 3.0  $\mu\text{g}/\text{kg}$ , but not 1.0  $\mu\text{g}/\text{kg}$  Ex-4. The supplement of CCK did not alter lick responses or trials initiated compared with Ex-4 administration alone. These findings support a role for GLP-1 but not CCK in the oral responsiveness to palatable stimuli. Furthermore, Ex-4-induced reductions were observed for both sucrose and intralipid, compounds representing *sweet* and *fat*, respectively.

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### Biography

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