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L-phenylalanine releases cholecystokinin (CCK) and is associated with reduced food intake in humans: evidence for a physiological role of CCK in control of eating.

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Exogenous administration of cholecystokinin (CCK) reduces food intake in humans; however, it is not clear if endogenous CCK is a true satiety hormone. The aim of this experiment was to manipulate endogenous release of CCK using L-phenylalanine (L-PA), a potent releaser of CCK, and to measure subsequent food intake. On separate occasions, six normal-weight fasted subjects (four men, two women) were administered 10 g of L-PA, D-PA, or placebo 20 minutes before being presented with a standard meal of known calorie content. Preliminary experiments had shown that peak plasma concentrations of CCK were obtained 20 minutes after administering L-PA. The test meal was given to coincide with this peak. One hundred-millimeter visual analog scales (VAS) to assess hunger, desire to eat, and fullness were completed premeal, postmeal, and at intervals thereafter. Blood was taken before administering PA/placebo immediately premeal and postmeal and stored for measurement of CCK levels by bioassay. Subjects consumed 1,089 +/- 86 kcal after L-PA (P = .03) compared with 1,587 +/- 174 kcal after placebo and 1,492 +/- 126 kcal after D-PA. The reduction in calorie intake after L-PA was associated with a significantly greater sensation of fullness. Basal levels of CCK were 1.10 +/- 0.12 pmol/L; 20 minutes after L-PA, CCK levels increased to 5.49 +/- 0.83 pmol/L. There was no increase in CCK following D-PA or placebo. Release of CCK by L-PA is associated with a reduction in subsequent food intake, and this suggests that CCK is an important satiety hormone in humans.

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