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The cholecystokinin-A receptor mediates inhibition of food intake yet is not essential for the maintenance of body weight

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In the final stages of the production process, panel *a* of Figure 2 was mistakenly repeated as panel *b*. The correct display of the figure and accompanying legend is reproduced here. We regret the error and have provided corrected reprints to the corresponding author: Alan S. Kopin, Tupper Research Institute, 750 Washington Street, Box 239, Boston, Massachusetts 02111, USA. Phone: (617) 636-7703; Fax: (617) 636-8692; E-mail: alan.kopin@es.nemc.org

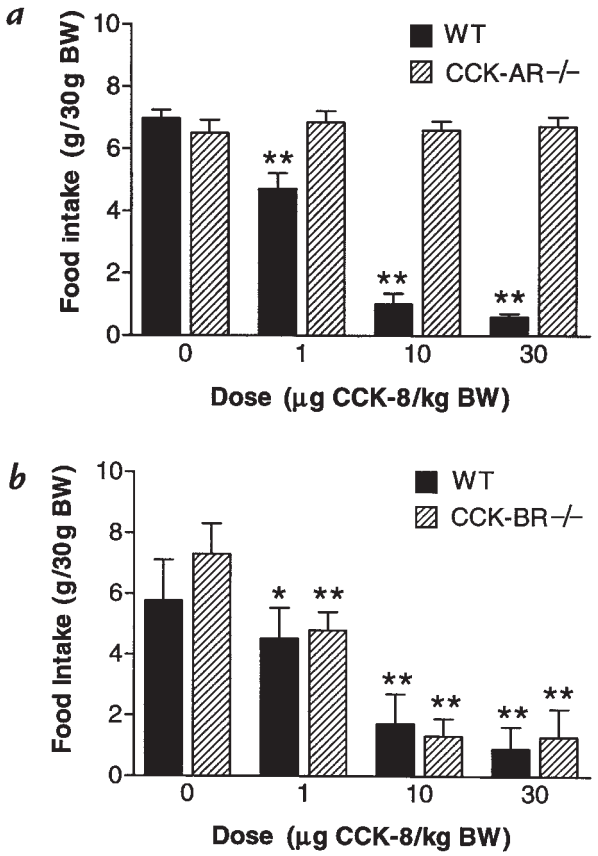

Figure 2
CCK-8 induced inhibition of food intake is mediated through the CCK-A receptor. After an overnight fast, animals were injected with either saline or CCK-8 and provided access to chocolate-flavored Ensure as described in Methods. Cumulative intake (mean ± SEM) over a 15-min period after injection is shown. Significance vs. intake after saline injection (0 μg CCK-8/kg body weight [*BW*]): $*P < 0.05$, $**P < 0.01$. (***a***) CCK-8 induced, dose-dependent inhibition of food intake is observed in wild-type (*WT*), but not in CCK-AR$^{-/-}$ mice. Food consumption by 10 wild-type and 10 CCK-AR$^{-/-}$ animals was compared. ANOVA parameters were [$F(3,39) = 89.23$, $P < 0.0001$] and [$F(3,39) = 0.16$, $P = 0.92$] for comparisons among wild-type and CCK-AR$^{-/-}$ animals, respectively. (***b***). CCK-8 induced, dose-dependent inhibition of food intake is observed in both wild-type and CCK-BR$^{-/-}$ mice. Food consumption by 9 wild-type and 10 CCK-BR$^{-/-}$ animals was compared. ANOVA parameters were [$F(3,35) = 44.40$, $P < 0.0001$] and [$F(3,39) = 135.21$, $P < 0.0001$] for comparisons among wild-type and CCK-BR$^{-/-}$ animals, respectively. *BW*, body weight.

Deletion of the fibrinogen alpha-chain gene (*FGA*) causes congenital afibrogenemia

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In the editing process, the abbreviation for FGA was incorrectly spelled out. The correct spelling appears above.