JCI The Journal of Clinical Investigation

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

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J Clin Invest. 1999;103(5):759-759. https://doi.org/10.1172/JCI4901E1.

Erratum

J. Clin. Invest.103:383–391 (1999) 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 2CCK-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 […]

March 1999
Volume 103 | Number 5

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J. Clin. Invest. **103**:383–391 (1999)

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

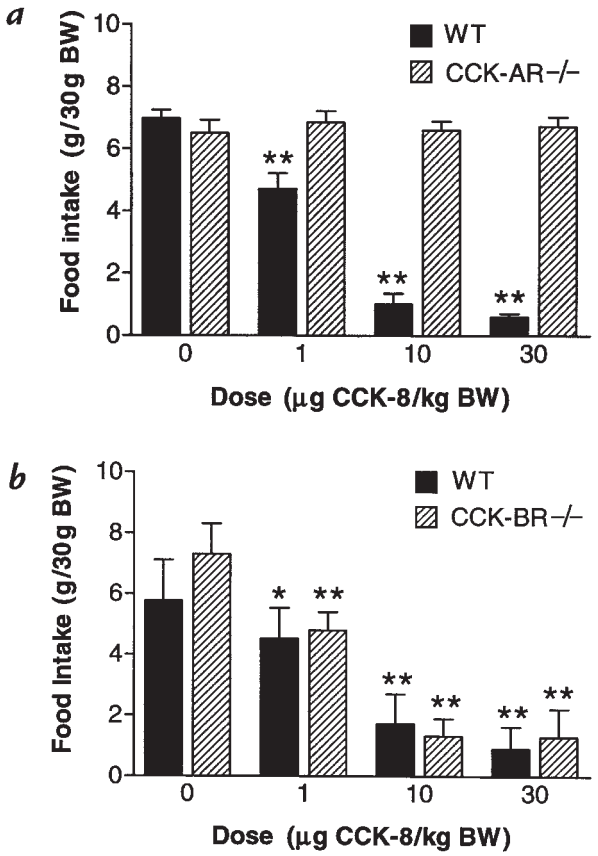

Figure 2
CCK-8 induced inhibition of food intake is mediated through the CCK-A receptor. After an overnight fast, animals were injected with either saline or CCK-8 and provided access to chocolate-flavored Ensure as described in Methods. Cumulative intake (mean ± SEM) over a 15-min period after injection 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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J. Clin. Invest. **103**:215–218 (1999)

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