

Control of Lipid Metabolism by Tachykinin in *Drosophila*

Wei Song,* Jan A. Veenstra, and Norbert Perrimon*

*Correspondence: rsong@genetics.med.harvard.edu (W.S.), perrimon@receptor.med.harvard.edu (N.P.)
<https://doi.org/10.1016/j.celrep.2020.02.011>

(Cell Reports 9, 40–47; October 9, 2014)

In the originally published version of this article, the Tk-g-Gal4 line that was used does not solely express in Tk EEs, as was originally stated. It does show some expression in the brain that we estimate to be ~50 neurons among the >900 Tk neurons in wild-type adult. This residual expression does not appear to affect our conclusion that gut-derived Tk regulates intestinal lipid metabolism, because (1) Tk-g-Gal4 induced Tk deficiency (UAS-Rpr/+; Tk-g-Gal4/+; or Tk-g-Gal4/UAS-Tk-i-JF01818) fails to change Tk mRNA expression in the brain, but diminishes ~90% Tk mRNA levels in the gut (Figure 2 in the manuscript); (2) Tk-g-Gal4 induced Tk deficiency does not affect Tk neuronal functions as measured by locomotor activity, olfactory response, and/or weight change (Figure S2 in the manuscript); and (3) TkR99D signaling in the gut plays similar roles of lipid metabolism as Tk-g-Gal4-manipulated Tk expression (Figure 4 in the manuscript).

The authors regret this error and thank Dr. Ilona Grunwald Kadow for indicating to us the expression of Tk-g-Gal4 in some neurons.

