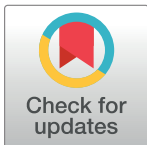


CORRECTION

Correction: Bacterial recognition by PGRP-SA and downstream signalling by Toll/DIF sustain commensal gut bacteria in *Drosophila*

Shivohum Bahuguna, Magda Atilano, Marcus Glittenberg, Dohun Lee, Srishti Arora, Lihui Wang, Jun Zhou, Siamak Redhai, Michael Boutros, Petros Ligoxygakis

[Fig 6](#) is incorrect. The authors have provided a corrected version here.



OPEN ACCESS

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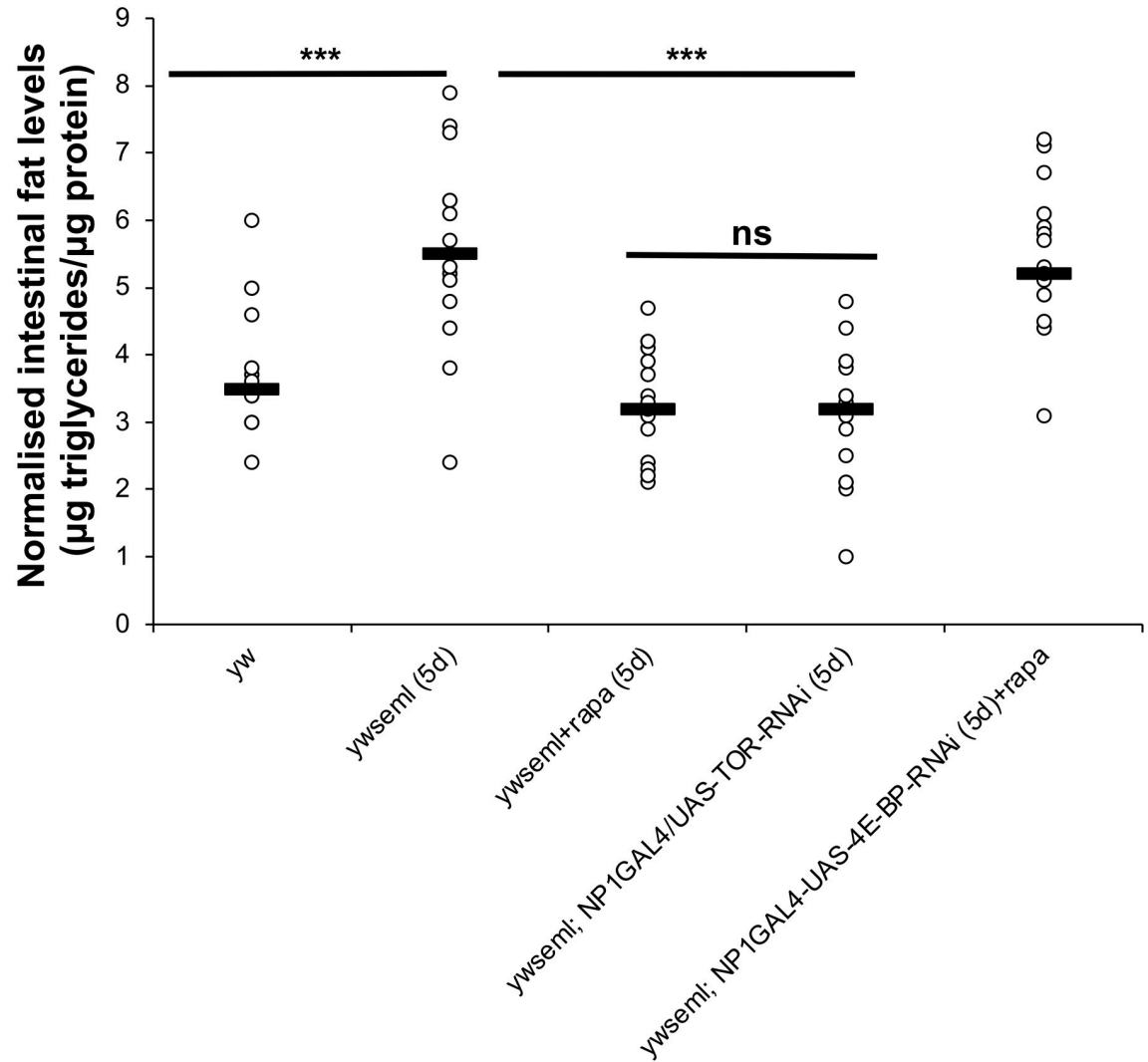


Fig 6. Loss of PGRP-SA increases intestinal fat levels. Loss of PGRP-SA increased intestinal triglyceride levels in 5-day old flies. This phenomenon was suppressed with pharmacological inhibition (rapamycin) or RNAi against TOR in ECs. This was dependent on 4EBP as *yw semi*; *NP1>4E-BP^{RNAi}* treated with rapamycin had fat levels statistically indistinguishable from *yw semi*. N = 15/genotype/treatment a total of three independent experiments (each with n = 5/genotype/treatment). Values of mutants and controls were statistically compared using student's t-test (**p<0.001, all other comparisons non-significant except *yw semi*; *NP1>4E-BP^{RNAi}* treated with rapamycin compared to *yw*, which has a p value<0.001-comparison not shown in the graph).

<https://doi.org/10.1371/journal.pgen.1010082.g001>

Reference

1. Bahuguna S, Atilano M, Glittenberg M, Lee D, Arora S, Wang L, et al. (2022) Bacterial recognition by PGRP-SA and downstream signalling by Toll/DIF sustain commensal gut bacteria in *Drosophila*. PLoS Genet 18(1): e1009992. <https://doi.org/10.1371/journal.pgen.1009992> PMID: 35007276