

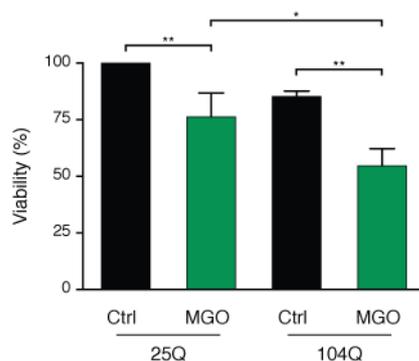
## TITLE

# Glycation potentiates neurodegeneration in models of Huntington's disease

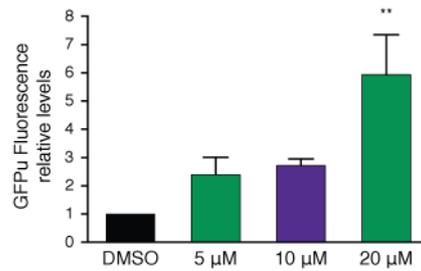
## AUTHORS

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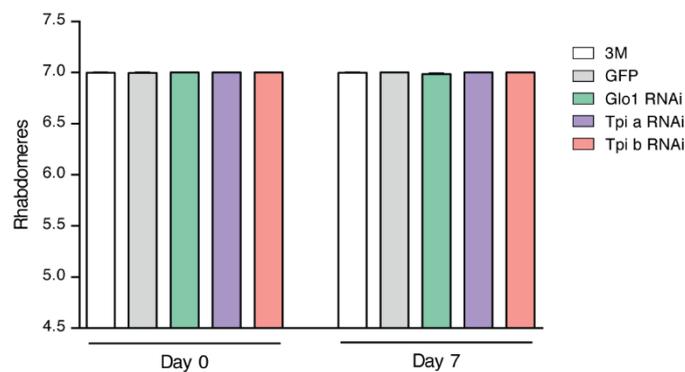
## SUPPLEMENTAL FIGURES



**Fig. S1. MGO treatment reduces the viability of H4 cells expressing HTT.** H4 cells expressing HTT 25Q or 104Q fused with GFP were treated with vehicle (Ctrl) or MGO (0.5 mM) for 16h. Viability was measured by MTT assay (n=3) and normalized to 25Q. Data in all panels are average  $\pm$  SD, \*  $p < 0.05$ , \*\*  $p < 0.01$ , one-way ANOVA followed by Tukey's multiple comparisons test.



**Fig. S2. Proteasome blockade increases GFPu levels.** H4 cells expressing GFPu were treated with vehicle (DMSO) or increasing concentrations of MG132 (5-20 μM). Cells were imaged in vivo and the average fluorescence levels normalized to the total number of GFPu expressing cells. Data normalized to DMSO is presented as average ± SD. \*\* p < 0.01, one-way ANOVA followed by Tukey's multiple comparisons test.



**Fig. S3. Knockdown of *Glo1* or *Tpi* does not induce neurotoxicity in WT flies.** Number of rhabdomeres per ommatidium in WT flies with pan-neuronal knockdown of *Glo1* or *Tpi* is presented at day 0 or 7 post-eclosion, with no neurodegeneration observed. 3M and GFP expressing control flies also do not exhibit degeneration of rhabdomeres. Data in all panels are mean ± SEM, one-way ANOVA with Newman-Keuls post-hoc test.