Supporting Information

Chinchore et al. 10.1073/pnas.1110666109

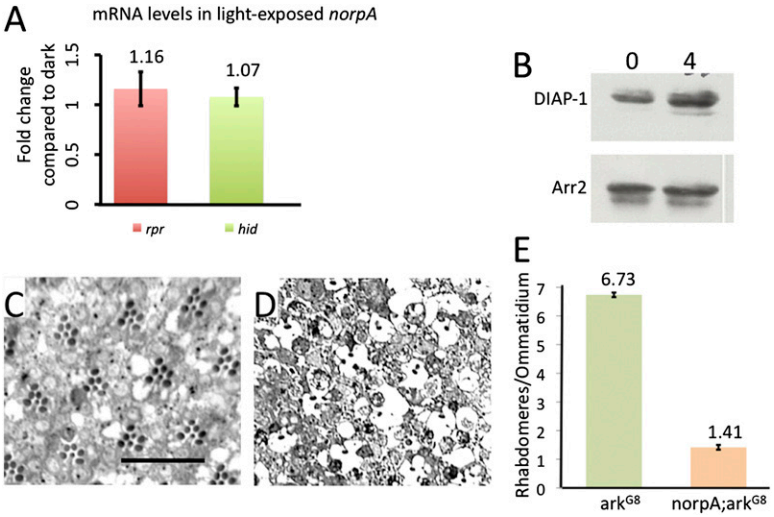

Fig. S1. Regulators of developmental PCD are not engaged during *norpA* degeneration. (*A*) Real-time quantitative RT-PCR data depicting *rpr* and *hid* transcript levels in 2-d light-exposed *norpA* flies compared with flies raised in darkness. (*B*) Immunoblot analysis to assess the steady-state level of DIAP-1 protein in dark-raised (0) and 4-d light-exposed (4) *norpA* retina. Photoreceptor-specific protein Arrestin-2 (Arr-2) serves as a loading control. Cross-sections (0.5 μm) of retinas from $norpA^{+}; ark^{G8}$ (*C*) or $norpA; ark^{G8}$ (*D*) flies treated with 7 d of light. The w^{-} area of the retina corresponding to the ark^{G8} clones is depicted. (Scale bar, 20 μm.) Genotypes are $w\ norpA^{EE5}eyFLP/w;\ FRT42D\ ark^{G8}/FRT42\ w^{+}\ cl\text{-}R11$ (*C*) and $w\ norpA^{EE5}eyFLP/Y;\ FRT42D\ ark^{G8}/FRT42D\ w^{+}\ cl\text{-}R11$ (*D*). (*E*) Ratio of the total number of discernable rhabdomeres to the total number of ommatidia in $norpA^{+}\ ark^{G8}$ and $norpA\ ark^{G8}$ flies. The w^{-} clones corresponding to ark^{G8} photoreceptors were counted. The ratio for $norpA^{+}\ ark^{G8}$ was 6.73 ± 0.08 (55 ommatidia, $n = 4$ flies), and the ratio for $norpA\ ark^{G8}$ was 1.41 ± 0.08 (101 ommatidia, $n = 5$ flies). Data are the mean ± SEM.

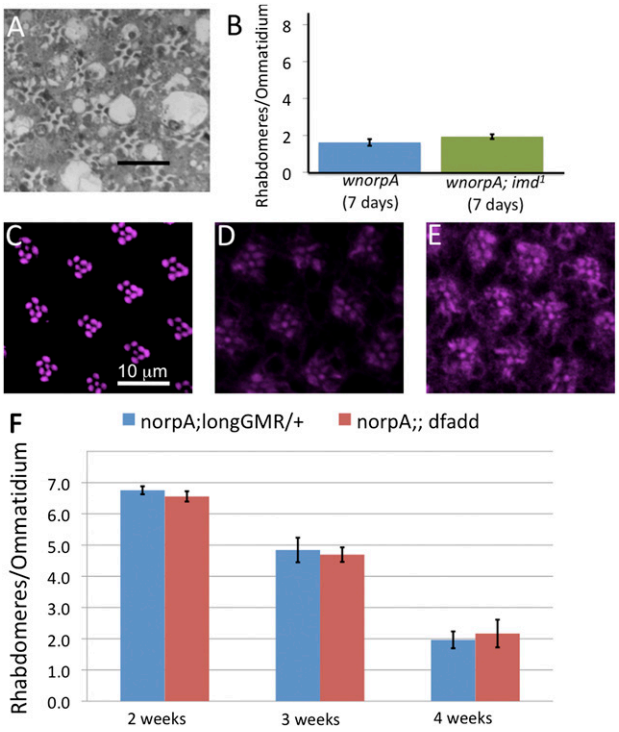

Fig. S2. Photoreceptor cell death in *norpA* is independent of known upstream activators of Dredd. (*A*) Cross-sections of retinas from *norpA; imd*^{1} flies exposed to 7 d of light. (Scale bar, 10 μm.) (*B*) Quantitation of rhabdomeres/ommatidium of *norpA imd*^{1} flies at 7 d of light exposure and compared with *norpA* controls exposed for a similar time. The ratio for *norpA; imd* was 2.17 ± 0.12 (183 ommatidia, 4 flies). The *norpA;; dfadd* ^{f02804} flies were exposed to light for 2 wk (*C*), 3 wk (*D*), and 4 wk (*E*), and retinas were isolated for whole-mount staining for Actin. (*F*) Quantitation of rhabdomeres/ommatidium for *norpA dfadd* double mutants at the indicated time points. *norpA; LGMR-GAL4/+* flies are used as a control because they are similarly pigmented. The ratios for *norpA; LGMR/+* (2 wk), 6.8 ± 0.1 (76 ommatidia, 3 flies); for *norpA; LGMR/+* (3 wk), 4.8 ± 0.4 (73 ommatidia, 4 flies); for *norpA; LGMR/+* (4 wk), 2.0 ± 0.3 (106 ommatidia, 4 flies); for *norpA;; dfadd* (2 wk), 6.6 ± 0.2 (46 ommatidia, 2 flies), for *norpA;; dfadd* (3 wk), 4.7 ± 0.2 (62 ommatidia, 3 flies), and for *norpA;; dfadd* (4 wk), 2.2 ± 0.4 (106 ommatidia, 5 flies) are shown. Data are the mean ± SEM.

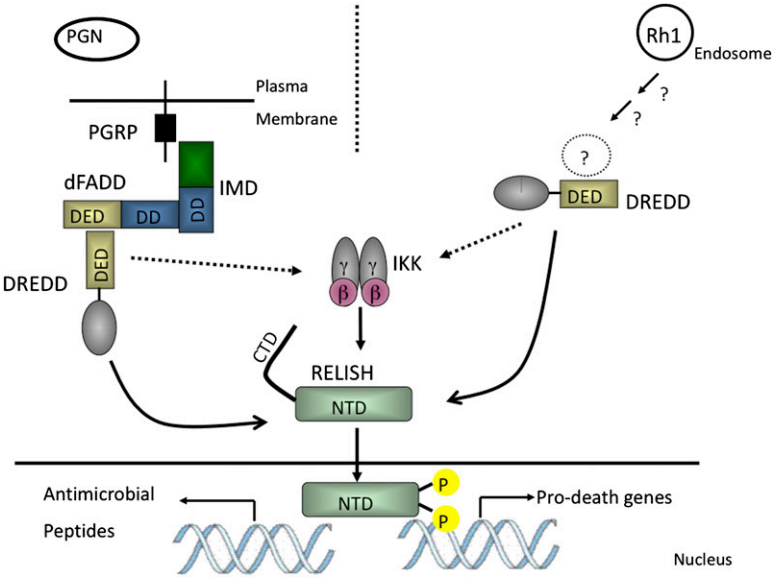

Fig. S3. Relish activation in photoreceptor cell death and the immune response. During the immune response, Dredd activates Relish. Dredd activation depends on Imd and dFadd proteins. In *norpA* photoreceptors, Rh1 accumulation in endosomes activates Dredd by an unknown mechanism. This leads to activation of Relish, which mediates transcription of genes that trigger death in photoreceptors. DD, death domain; DED, death effector domain; PGN, peptidoglycan; PGRP, peptidoglycan receptor protein.

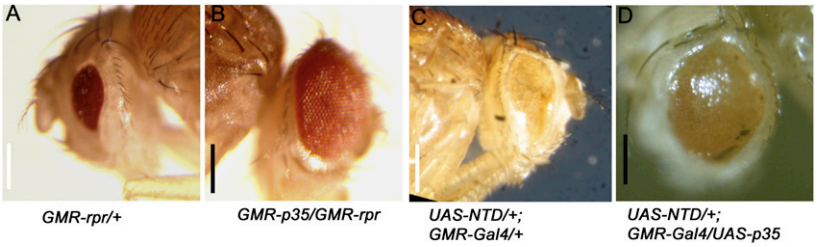

Fig. S4. (*A*) Eye ablation induced after ectopic expression of *rpr* from one copy of *GMR-rpr* transgene. (*B*) The *rpr*-induced eye ablation is rescued by expression of p35 from one copy of *GMR-p35* transgene. (*C*) Eye phenotype of a fly expressing Relish NTD from one copy of the transgene. Reduced eye curvature, glazed appearance, aberrant pigmentation is apparent. (*D*) Overexpression of p35 from a UAS transgene does not rescue the phenotype induced by Relish NTD. (Scale bars, 150 μm.)