SUPPLEMENTARY INFORMATION

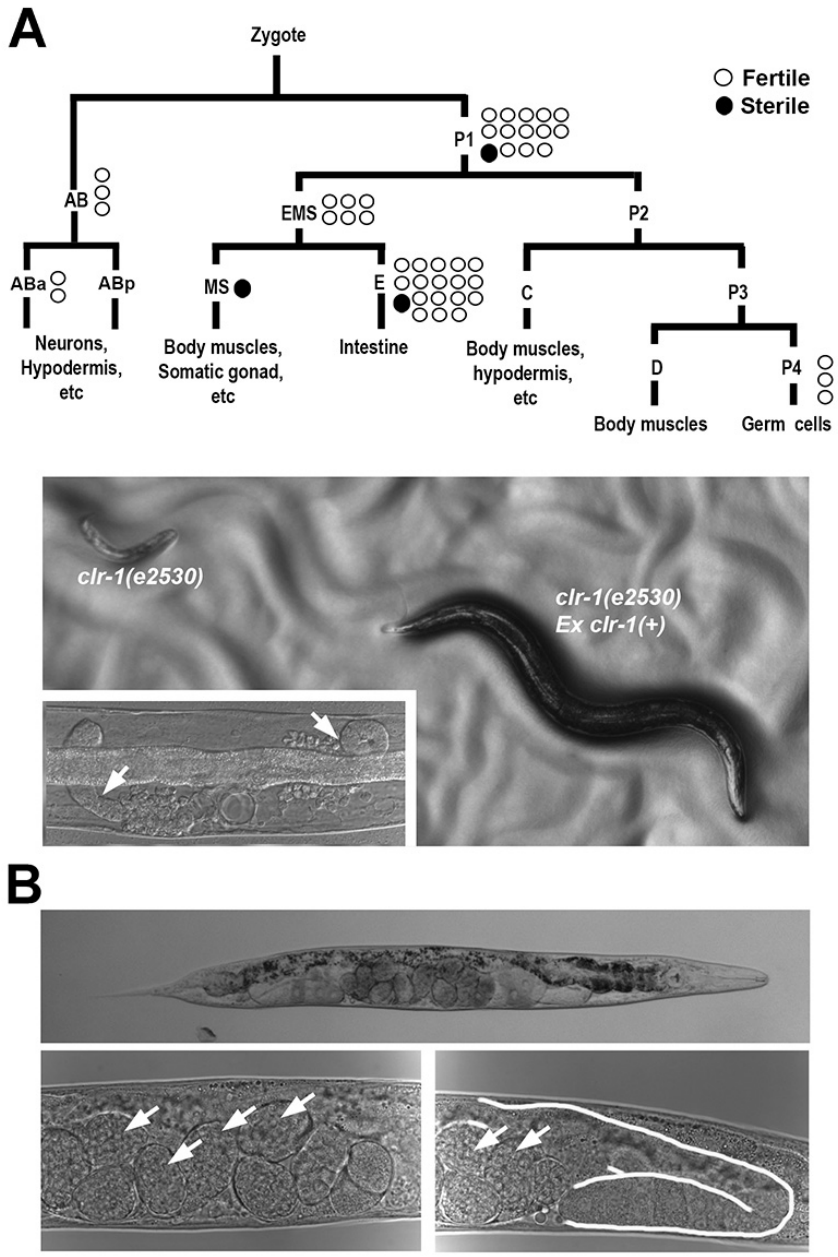

Figure S1. CLR-1 expression in the hypodermis promotes survival and prevents germ cell degeneration. (A) Genetic mosaic analysis showing cell lineages of major tissues. Each circle indicates one genetic mosaic worm. Points at which the genomic copy of *clr-1(+)* was lost and the resulting phenotype are indicated. Below are representative DIC images of *clr-1(e2530)* mosaic worms with and without *clr-1* expression. *Ex clr-1(+)* indicates expression of the *clr-1* genomic locus via an extrachromosomal array. Inset shows a close-up image of a *clr-1* mutant degenerating gonad (arrows). The hypodermis is syncytial and derived from multiple lineages. Thus, hypodermal *clr-1* loss occurs only when the *Ex clr-1(+)* transgene is lost from both AB and P1. Note that *clr-1* loss from the germ line (P4) or somatic gonad lineages (EMS) does not cause gonad

degeneration or other obvious defects. (B) Driving *clr-1* expression in the hypodermis using the *dpy-7* promoter is sufficient to partially rescue the *clr-1(e1745)* fluid accumulation and gonad defects. Gonad is outlined in white. Arrows indicate fertilized eggs.

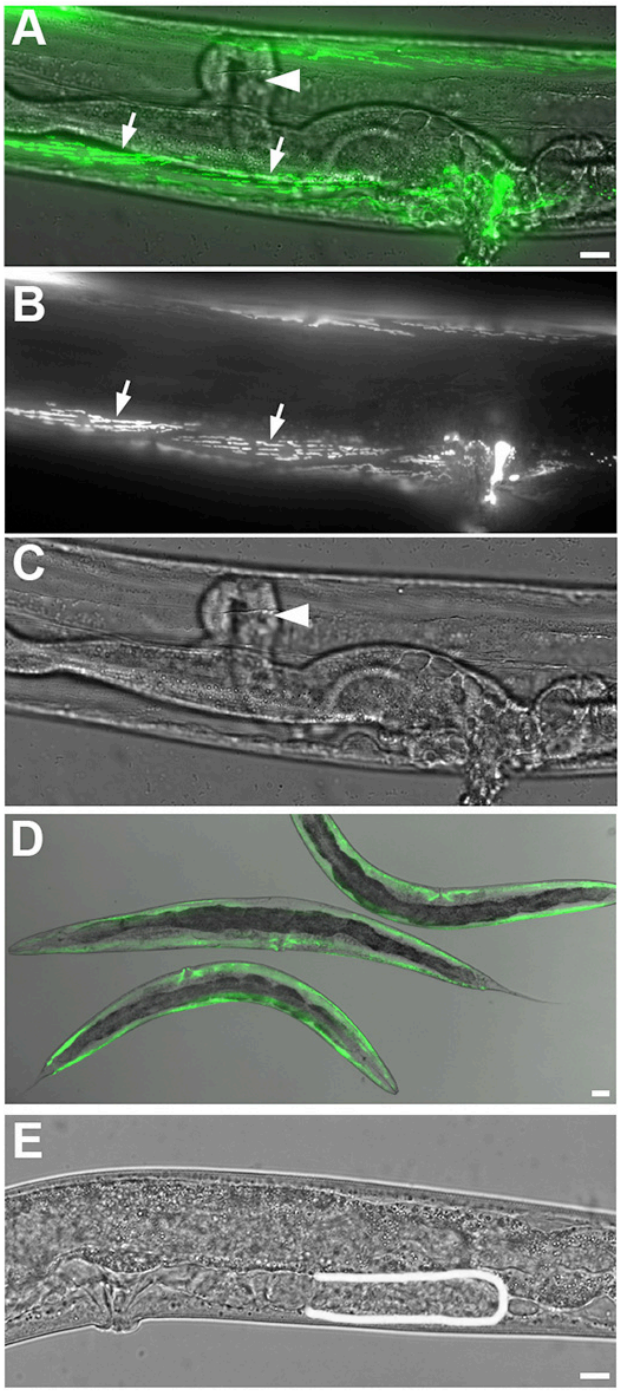

Figure S2. Effects of *clr-1* or *vab-1* MSPd receptor loss on *vpr-1* mutant phenotypes. (A-D) CLR-1 receptor loss suppresses the *vpr-1(tm1411)* body wall muscle

mitochondrial localization defect, but not the gonadogenesis defect. Merged DIC and GFP (A), GFP only (B), and DIC only (C) images of a *vpr-1 M- Z-* mutant exposed to *clr-1* RNAi throughout larval development. The *myo-3p::mitoGFP* transgene was used to visualize body wall muscle mitochondria. Arrows indicate muscle mitochondrial networks showing linear arrays seen in the wild type (not shown), but not in *vpr-1 M- Z-* mutants expressing *clr-1*. See Han et al. (2012 and 2013) for muscle phenotypes. Arrowhead indicates an arrested *vpr-1 M- Z-* gonad. (D) *vpr-1 (tm1411); clr-1(e1745)* transgenic mutants expressing the *rol-6p::clr-1* and *myo-3p::mitoGFP* transgenes. A merged DIC and GFP image is shown. Worms were grown at 25°C. Notice that the early larval lethality defect (Figure S1A) is suppressed by the *rol-6p::clr-1* transgene, but the worms are still sterile. (E) VAB-1 MSPd receptor loss does not suppress the *vpr-1(tm1411)* gonad defect. DIC image of a *vpr-1 (tm1411); vab-1(dx31)* hermaphrodite showing arrested gonadogenesis (E) that is similar to *vpr-1(tm1411)* single mutants (Figure 1). Gonad is outlined in white. Bars, 10 μm.

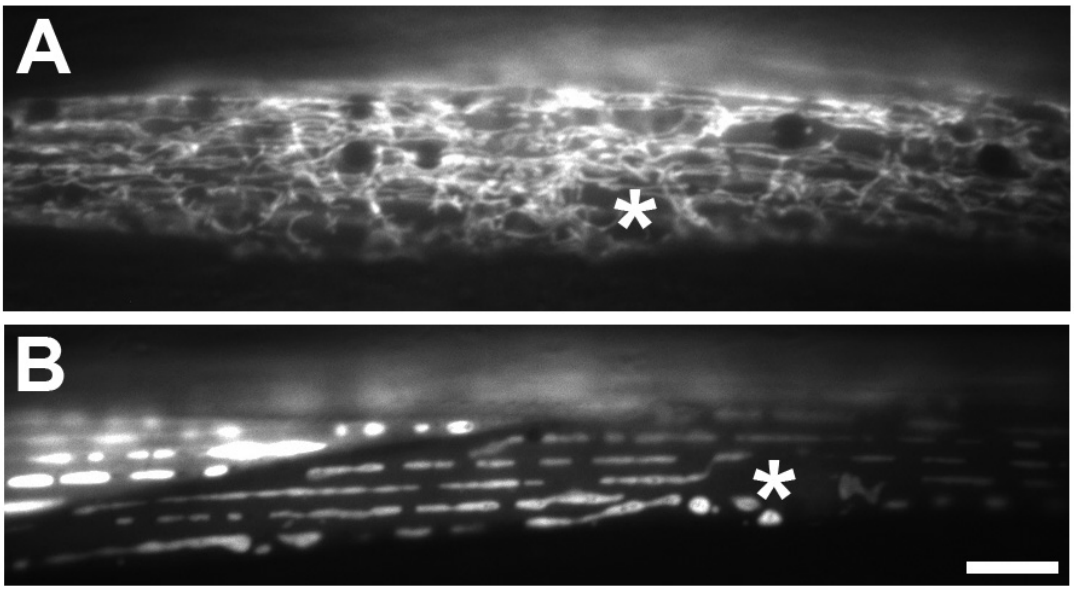

Figure S3. Neuronal *vpr-1* expression using the Q system is sufficient to rescue the *vpr-1(tm1411)* body wall muscle mitochondrial defect. *vpr-1* expression was driven in head interneurons under the *glr-5* promoter. The *myo-3p::mitoGFP* transgene was used to visualize body wall muscle mitochondria. (A, B) GFP images of transgenic *vpr-1* mutant muscles grown in the absence (A) and presence of QA (B), which induces *vpr-1* expression. Asterisks indicate muscle nuclei. Wild-type controls (not shown) are similar to mitochondrial arrays observed in panel B (Han et al., 2012 and 2013). Bar, 10 μm.