

Supporting Information

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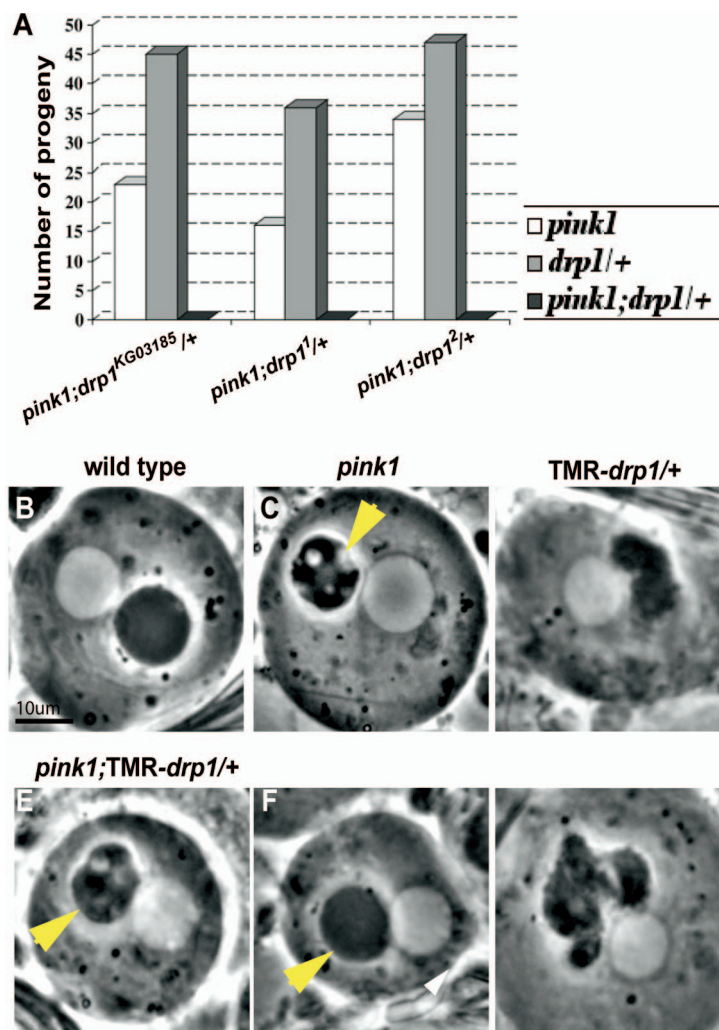


Fig. S1. Genetic interactions between *pink1* and *drp1*. (A) Synthetic lethality in *pink1* mutants carrying a heterozygous mutation in *drp1*. Three different sets of experiments using three independent alleles of *drp1* (*drp1¹*, *drp1²* and *drp1^{KG03185}*) are shown. (B–G) Phase-contrast images of onion-staged spermatids with various genotypes. Yellow arrows point to the nebenkerns. Testes-specific overexpression of *drp1* (TMR-*drp1*) results in abnormal nebenkerns reminiscent of those seen in *fzo* mutants in a portion of flies (D). *drp1* overexpression in the *pink1*-mutant background (E–G) results in a range of phenotypes. Some nebenkerns are *pink1* mutant-like (E), some appear normal (F), and others show bizarre shapes (G). In any event, the partial suppression of the *pink1* phenotypes suggests an interaction between *pink1* and *drp1*. The variable phenotypes associated with *drp1* overexpression are likely caused by the late expression timing of the promoter (TMR, i.e., β 2-tubulin, which is expressed just before the onion stage). (Scale bars: 10 μ m.)

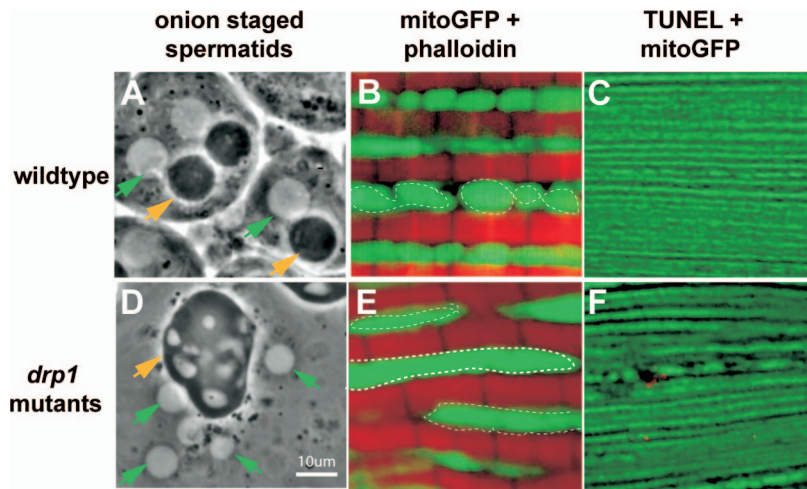


Fig. S2. *drp1* mutants show testes and muscle phenotypes distinct from *pink1* mutants. *drp1²* mutants are largely lethal. However, rare adult escaper males show aberrant nebenkerns. Compared with WT specimens (A), mitochondria in *drp1* mutant spermatids exhibit large, bizarrely shaped blobs (orange arrow). They also contain irregularly shaped phase-light materials distinct from the phase-light nucleus (green arrow) (D). The borders of some mitochondria are marked with white dashed lines. Note that the cytoplasmic bridges connecting spermatids are disrupted during sample preparation. Our results are consistent with a previous study in which lack of *drp1* function in germline clones displayed defects in distribution of mitochondria to spermatids, which results in mitochondria being stuck in ring canals linking nearby spermatids [Aldridge AC *et al.* (2007) Roles for Drp1, a dynamin-related protein, and Milton, a kinesin-associated protein, in mitochondrial segregation, unfurling and elongation during *Drosophila* spermatogenesis. *Fly* 1:38–46]. Compared with wildtype (B), muscles from *drp1* mutant flies show elongated mitochondria (E) but no clumps (F), which is different from *pink1* mutant phenotypes (Fig. 3 B and G). In addition, *drp1* mutant flies do not show TUNEL positive signals (F), which is also different from *pink1* mutants (Fig. 5E). (Scale bars: 10 µm in A and D.)