

Supplementary Figures for:

α -synuclein expression from a single copy transgene increases sensitivity to stress and accelerates neuronal loss in genetic models of Parkinson's disease

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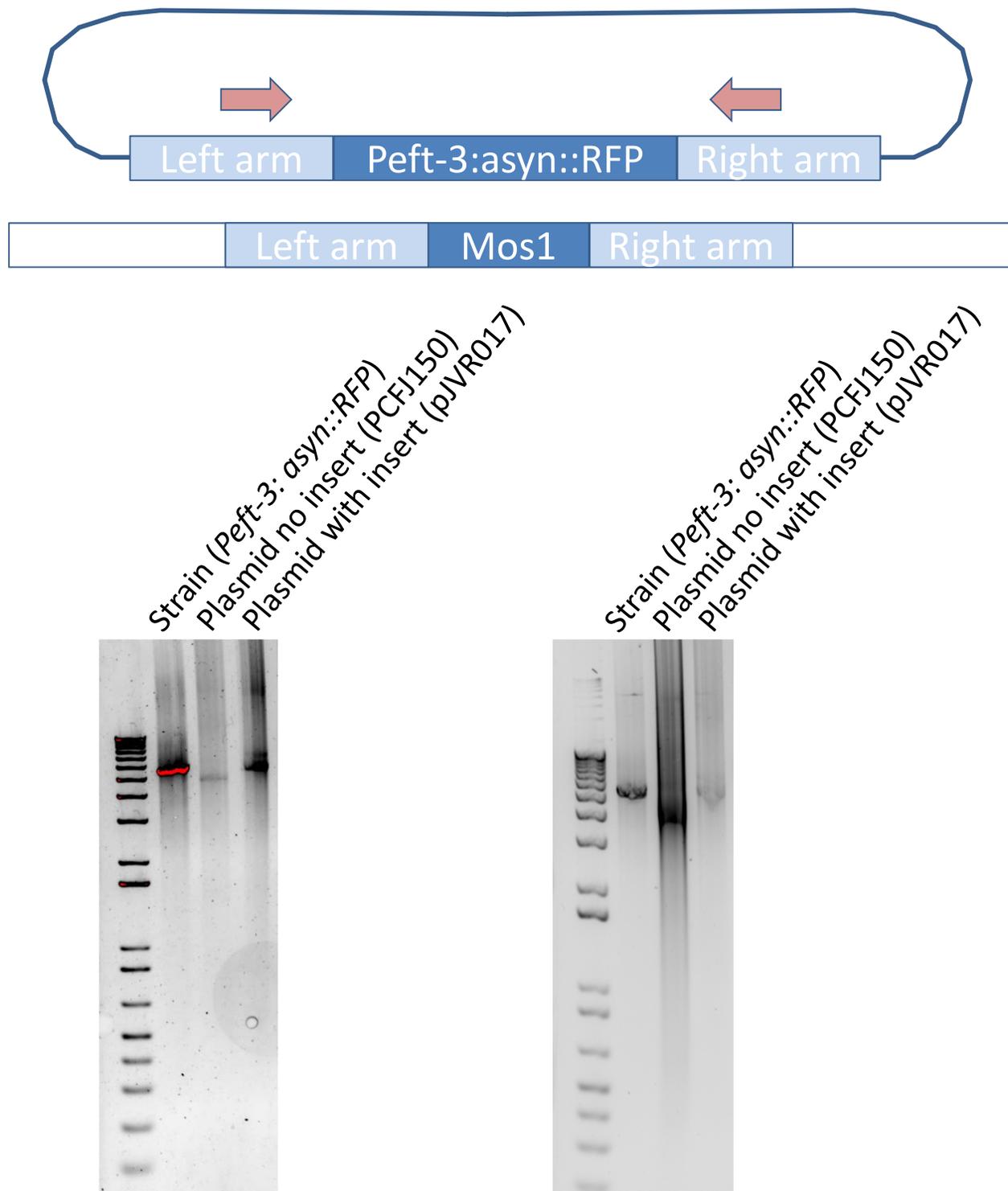


Fig. S1. A single copy of α -synuclein transgene is present in ubiquitously expressing α -synuclein model. A. Diagram of MosSCI (Mos1-mediated single copy insertion) approach. The *mos-1* gene present in the genomic DNA is replaced with the genetic material between the left and right arms, in this case the Peft-3::asyn:RFP transgene (and *unc-119* rescue construct). Red arrows indicate the location of primers. **B.** Amplification of Mos1 region in *Peft-3::asyn:RFP* worm produces a band which is identical to amplification of the original plasmid DNA (pJVR017). The size of this fragment is larger than the plasmid without the Peft-3::asyn:RFP transgene integrated (PCFJ150). This confirms that the asyn:RFP was inserted as a single copy transgene.

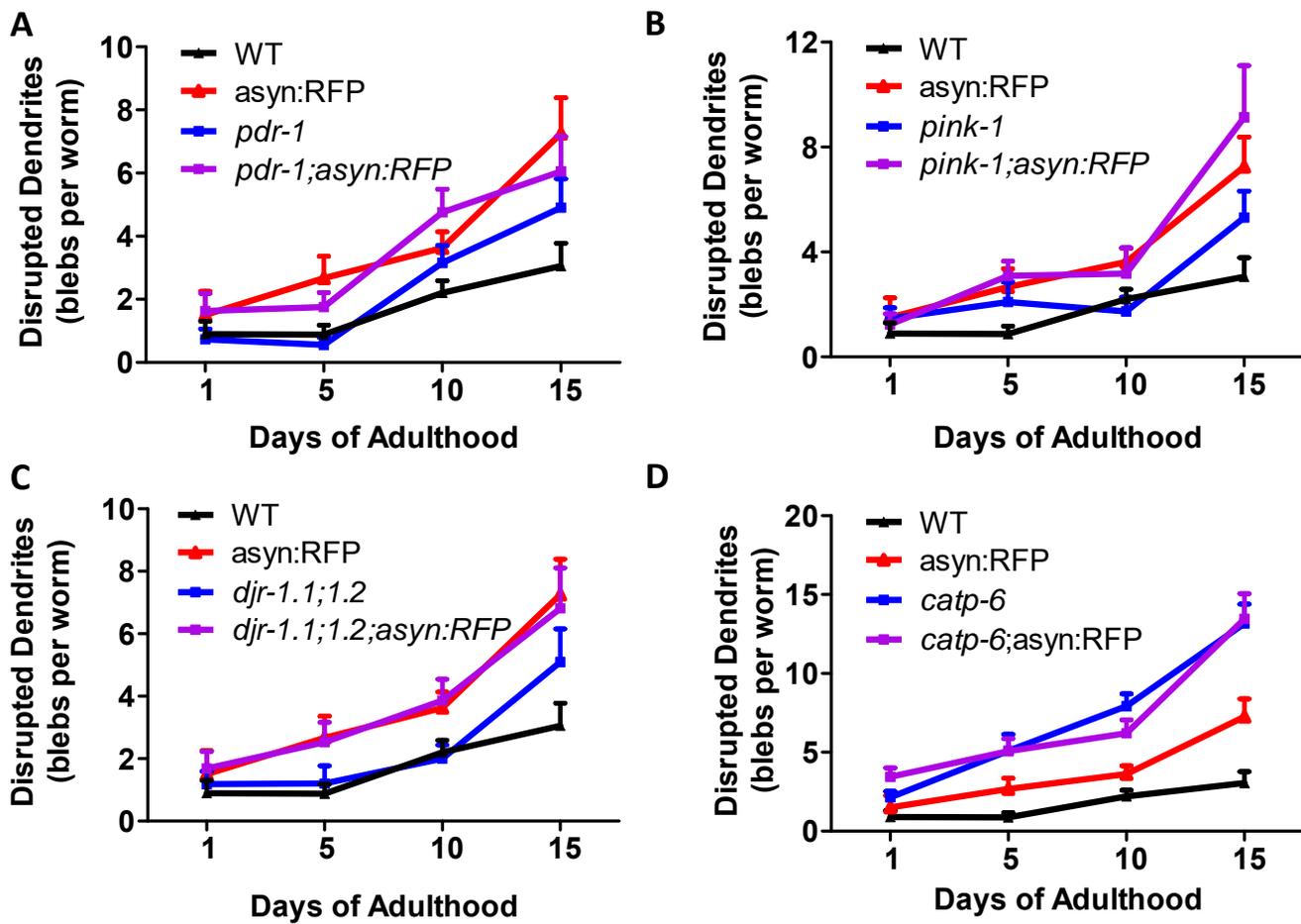


Fig. S2. α -synuclein expression increases neuronal blebbing independent of strain background. The addition of *asyn::RFP* increases dendritic blebbing compared to control in *pdr-1* (A), *pink-1* (B), and *djr-1.1* (C) animals but does not further increase blebbing in *catp-6* worms (D). Error bars indicate SEM.

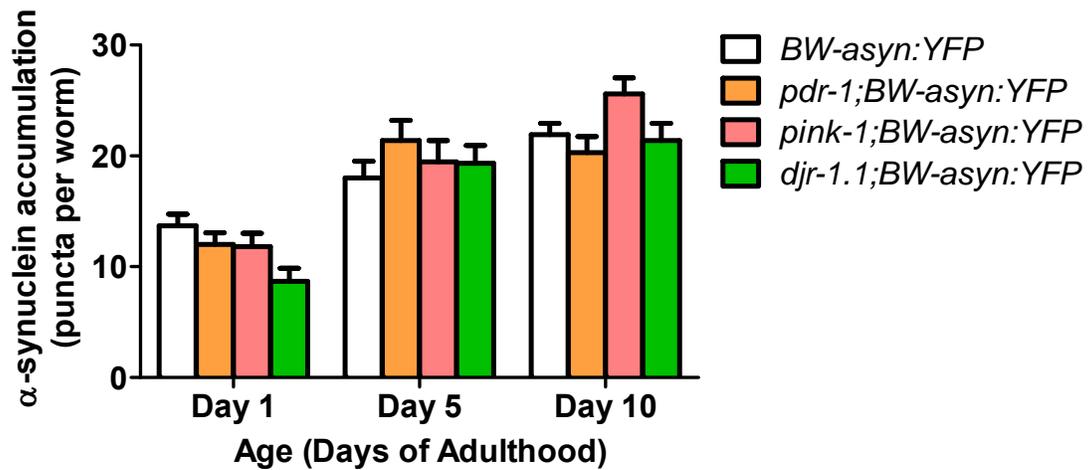


Fig. S3. Mutations in *pdr-1*, *pink-1* and *djr-1.1* do not accelerate aggregation in *BW-amyloid precursor protein:YFP* worms. Fluorescent puncta were quantified using an automated image analysis protocol. *Punc-54::alpha-syn:YFP* worms are indicated as *BW-amyloid precursor protein:YFP*. Error bars indicate SEM. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.