**Figure S1.**

**a.** Map of the repair template plasmid with *gfp11*x7 insert. The 5’ and 3’ homology arms can be replaced with ~500bp of regions flanking each side of the target sequence. The *sqt-1*(dominant roller gene) and hygromycin resistance gene allow high throughput positive selection and then can be removed with heat shock-induced Cre expression.

**b.** Map of the GFP1-10 reporter plasmid. Tissue-specific or heat-shock promoters (labeled in red) for driving GFP1-10 expression can be inserted at a multiple cloning site (blue). Adapted from (Dickinson and Goldstein 2016).

**Figure S2. The GFP11x7 knock-in does not disrupt OIG-1 function**.

**a.** Top: Cartoon structure of knock-in at *oig-1* locus after CRISPR injection but before excision of the self-excising cassette (SEC) and the stop codon thus resulting in detectable TagRFP expression from the native *oig-1* promoter. Bottom: Representative images showing expression of TagRFP::SEC::OIG-1::3XFLAG as a single copy knock-in transcriptional reporter before heat-shock induced removal of the SEC (Dickinson and Goldstein 2016). Note the bright TagRFP signal in ventral cord VD neuron soma at the L4 stage. Asterisks mark autofluorescent granules in the gut. Scale bar is 20 µm.

**b.** ACR-12::GFP puncta in VD motor neurons are mislocalized to the ventral nerve cord in *oig-1* mutants (He *et al.* 2015).

**c.** Postsynaptic ACR-12::GFP puncta in GABAergic neurons in dorsal and ventral nerve cords (insets) in the wild type and in the *gfp11x7::oig-1* knock-in line in L4 stage larvae. Scale bars are 10 µm. Asterisks mark autofluorescent granules in the gut.

**d.** The *gfp11x7::oig-1* knock-in retains wild-type OIG-1 function. Quantification showing that ventral ACR-12::GFP fluorescence intensity in the *gfp11x7::oig-1* knock-in is not significantly different from wild type. In contrast, ACR-12::GFP labeled clusters are ectopically relocated to the ventral cord in *oig-1* loss-of-function mutant alleles (He *et al.* 2015) (Howell *et al.* 2015). ns, not significant. N=15, Student’s T-test.

**e.** Representative image of *gfp11x7::oig-1* L4 larva. Note the absence of detectable GFP fluorescence in either dorsal or ventral cords. Asterisk marks gut autofluorescence. Scale bar is 10 µm.

**f.** Representative image of strain expressing *Prab-3*::GFP1-10 as an extrachromosomal array. Note absence of detectable GFP fluorescence in either the dorsal or ventral cords and weak GFP fluorescence in ventral cord cell soma indicative of low background signal from GFP1-10 fragment (arrowhead) (Feng *et al.* 2017) in L4 larva. Asterisks mark gut autofluorescence. Scale bar is 10 µm.

**Figure S3. OIG-1 NATF-GFP fluorescence signal is less punctate than that of mCherry::OIG-1 when over-expressed from an extrachromosomal array.**

**a-b**. Line scans of (**a**) NATF-GFP-labeled OIG-1 in VD GABAergic motor neurons (OIG-1::GFP11x7; *Pttr-39*::GFP1-10) (see Figure 2m) and (**b**) mCherry::OIG-1 over-expressed from transgenic array (*Punc-25::mCherry::oig-1*) (see Figure 2b) in the ventral cord. Dashed line marks 0.75 of normalized fluorescence intensity for each scan. **c**. Quantitative comparison of peaks (> 0.75 normalized fluorescence intensity) shows that the OIG-1 NATF-GFP signal is less punctate than that of mCherry::OIG-1 expressed from an extrachromosomal array. N = 7. Student’s T-test, \*p = 0.0151.

**Figure S4. Secreted GFP1-10 is functional but does not detect extracellular OIG-1.**

**a.** The *gfp11x7::oig-1* knock-in was crossed with the *Prab-3::ssgfp1-10* transgenic line, which drives GFP1-10 secretion from all neurons. No GFP signal was detected in these L4 stage larvae. Asterisks denote gut autofluorescence.

**b-c.** Confocal image **(b)** and inset **(c)** showing robust GFP signal at the nerve cord (arrowheads) and nerve ring (arrow) when the *Prab-3::ssgfp1-10* transgenic line is crossed with the GRASP marker *Pflp-18::gfp11::nlg*. Asterisk denotes gut autofluorescence.

**d.** *Pflp-18::gfp11::nlg* drives expression of Neuroligin (NLG) labeled with GFP11 at its extracellular N-terminus in neurons in the head and in command interneurons (Feinberg *et al.* 2008) that extend processes from the nerve ring into the ventral nerve cord thus resulting in a bright GFP signal arising from complementation in these locations with GFP1-10secreted from *Prab-3::ss::gfp1-10*.

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