FIGURE S4.—Lactose-induced RNAi feeding against canonical Rac signaling pathway members results in synaptic vesicle misaccumulations, but not architectural breaks, in GABAergic D-type motor neurons of dorsal nerve cords. The percentage of axonal GFP gaps (percent young adult worms with gaps per total sample size; n=30 for each of three to five independent experiments) in GABAergic D-type motor neurons of dorsal nerve cords (DNCs) of various RNAi treatments is depicted. Soluble GFP expression showed no architectural breaks in the DNC axons of young adult oxIs12 (Punc-47::GFP) worms with mock (α-synuclein) RNAi or RNAi against Rac signaling pathway members (light gray bars). Yet, RNAi against most Rac signaling pathway members resulted in misaccumulated synaptic vesicles, as revealed by gaps in GABAergic neuron-specific expression of a synaptobrevin-1 (SNB-1) and GFP translational fusion protein (dark gray bars). Despite not significantly disrupting SNB-1::GFP localization in ventral nerve cords (VNCs) (see Figure 5) of young adults, mig-15(RNAi) yielded SNB-1::GFP misaccumulations in young adult DNCs. Conversely, pes-7(RNAi) did not significantly disrupt SNB-1::GFP localization in DNCs, despite affecting VNCs (see Figure 5). Combinatorial RNAi was used against two of three triply redundant Racs, ced-10 and mab-2. Results from mock RNAi against oxIs12 worms were used to standardize other results with oxIs12 worms. Likewise, results from mock RNAi against juIs1 (Punc-25::SNB-1::GFP) worms were used to standardize other results with juIs1 worms. Each data point represents mean ± mean of standard deviations. *p < 0.05; Fisher’s Exact Test.