Figure S1: Neuronal copper deficiency does not affect time to eclosion. Time to eclosion was measured for Elav>\textit{ATP7}\textsuperscript{OE} and control flies on A) copper-limited, B) basal and C) CuSO\textsubscript{4}-supplemented media. Day 1 was taken as the first day any adult flies from either genotype first eclosed from a particular food type; BCS and CuSO\textsubscript{4}-supplementation both slow down development. No change in time to eclosion due to \textit{ATP7}\textsuperscript{OE} over expression was observed under any of the conditions. N = 3 for each genotype.
Figure S2: Pan-neuronal knockdown of *Phm* and / or *ATP7* has no effect on viability or wing expansion. *Elav-Gal4* was used to drive RNAi transgene expression in all neurons. Flies of each genotype were raised in density-controlled conditions on basal, copper-limited and CuSO₄-supplemented media. A) % Survival is shown for each genotype. No genotype / media combination had an appreciable effect on survival. B) % of adults with normal wing morphology >24 hours post-emergence is shown for each genotype / media combination. No genotype / media combination had any effect on wing expansion. (* p-value < 0.05, ** p-value < 0.01, *** p-value < 0.001)
**Figure S3: Severe copper overload in CCAP cells does not affect gross axonal morphology.** Brains of 3rd instar larvae were dissected, fixed and stained with anti-HRP (red), which stains all axons. A and B) $CCAP^+\text{+}$ control brains showing normal number and morphology of CCAP-producing cells. C) Addition of $Ctr1B^{OE}$ construct results in a clear decrease in the number of visible cell bodies (arrows) and a strong reduction in axonal GFP levels (green). A’ and A”) Negative control for anti-HRP staining. $CCAP^+\text{+}$ control brains (B’ HRP-alone and B” HRP + GFP) and $CCAP>Ctr1B^{OE}$ brains (C’ HRP alone and C” HRP + GFP) show similar gross axonal morphology as highlighted by anti-HRP staining.