Massachusetts Neurologic Association
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CALL FOR ABSTRACTS
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Title: Removing endogenous Appl enhances neurotoxicity induced by human α-synuclein in *Drosophila*

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Abstract:
Recent studies have shown that α-synuclein accumulates in the brains of many patients with Alzheimer's disease. But it is unclear what exact mechanisms underlie the interactions between α-synuclein and Aβ. Based on the literature, I hypothesize that α-synuclein and Aβ interact in the cytoplasm and mitochondria of neurons to cause dysfunction. To test this hypothesis, human α-synuclein was overexpressed in *Appl* null flies. Motor function was assessed by the climbing test. H&E staining was conducted in brain sections and the number of neurons in the anterior medulla was counted. At the age of 10 days, α-synuclein transgenic flies, but not *Appl* null flies, exhibited significant climbing deficits and neuron loss in the anterior medulla compared to control flies. Flies overexpressing α-synuclein in an *Appl* null background performed significantly worse on the climbing test than flies overexpressing α-synuclein in a wildtype background. Consistently, flies overexpressing α-synuclein in an *Appl* null background had greater neuronal loss in the anterior medulla compared with flies overexpressing α-synuclein in a wildtype background. In conclusion, removing endogenous Appl enhances neurotoxicity induced by human α-synuclein in *Drosophila*. Future directions will include studying whether depleting endogenous Appl increases α-synuclein aggregates in the brain.