## Abstract template

## (Title) Activity-dependent retrograde laminin A signaling regulates synapse growth at *Drosophila* neuromuscular junctions

## (Presenter indicates by bold face and underline)

**<u>Pei-I Tsai<sup>1,2</sup></u>**, Manyu Wang<sup>1,2</sup>, Hsiu-Hua Kao<sup>1</sup>, Ying-Ju Cheng<sup>1</sup> Yu-Jing Lin<sup>1</sup>, Ruey-Hwa Chen<sup>2,3</sup>, and Cheng-Ting Chien<sup>1,2</sup>

<sup>1</sup>Institute of Molecular Biology, Academia Sinica, Taipei 115, Taiwan

<sup>2</sup>Institute of Molecular Medicine, National Taiwan University, Taipei 106, Taiwan

<sup>3</sup>Institute of Biological Chemistry, Academia Sinica, Taipei 115, Taiwan

Retrograde signals induced by synaptic activities are derived from postsynaptic cells to potentiate presynaptic properties, such as cytoskeletal dynamics, gene expression, and synaptic growth. However, it is not known whether activity-dependent retrograde signals can also depotentiate synaptic properties. Here we report that laminin A (LanA) functions as a retrograde signal to suppress synapse growth at *Drosophila* neuromuscular junctions (NMJs). The presynaptic integrin pathway consists of the integrin subunit  $\beta v$  and focal adhesion kinase 56 (Fak56), both of which are required to suppress crawling activity-dependent NMJ growth. LanA protein is localized in the synaptic cleft and only muscle-derived LanA is functional in modulating NMJ growth. The LanA level at NMJs is inversely correlated with NMJ size and regulated by larval crawling activity, synapse excitability, postsynaptic response, and anterograde Wnt/ Wingless signaling, all of which modulate NMJ growth through LanA and  $\beta v$ . Our data indicate that synaptic activities own-regulate levels of the retrograde signal LanA to promote NMJ growth.