Volume 12

Washington University
Undergraduate Research Digest

Spring 2017

# The Molecular Mechanisms of Sensory Synapse Formation

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# The Molecular Mechanisms of Sensory Synapse Formation

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Estimated to cost the country over 635 billion dollars per year, in both treatment costs and lost productivity, pain is an extremely exigent issue in society today. Therefore, it is critical to develop a more comprehensive understanding of the mechanism with which these pain signals are processed, potentially opening up new ideas for possible treatments. Studies have shown that two classes of proteins, neurexins and neuroligins, play a large role in the specificity of synaptic connectivity, leading to the hypothesis that the expression patterns of these neuroligins and neurexins create a code for connectivity. With this in mind, we propose the hypothesis: β neurexins in sensory neurons influence interactions with neuroligins, in turn altering aspects of synapse formation. Utilizing a co-culture of sensory neurons, cultured from mouse dorsal root ganglia, and fibroblast COS7 cells transfected with purified neuroligin DNA adhesion molecules and presynaptic terminals were be immunostained and then imaged—allowing for quantitative analysis of synapse formation utilizing the images from the confocal. To isolate the effects of the neurexin gene, a conditional gene knockout technique using viral vectors was implemented in order to knockout the genes coding for β neurexins. Under the premise of our hypothesis—that β Neurexins in sensory neurons influence interactions with neuroligins, there would be distinctly increased synapse formation in sensory neuron cultures containing the Neurexin gene than those with the genes knocked out. This would be visually indicated by the number of stained adhesion molecules and presynaptic terminals in the contrasting confocal images, showing that trans-synaptic interactions of neurexins and neuroligins play a large role in the specification of synaptic connections.