

## **Abstract**

Apoptosis in neurons is induced to eliminate excess or damaged cells during the development and maturation of the nervous system. The apoptotic pathway in neurons is commonly studied using a sympathetic neuron model of NGF (nerve growth factor) deprivation. Recent studies from our lab have shown that the apoptotic events in neurons triggered with NGF deprivation are activated as a transient pulse rather than a persistent signal. These transient events allow for the reversal of apoptosis, which has been analyzed through a NGF deprivation and restoration model. In this study, we analyzed how other apoptotic insults impact the reversibility of apoptosis in neurons, as well as if previous exposure to an apoptotic insult confers increased sensitivity or resistance to a second apoptotic insult. Here we show that inducing apoptosis through ER (Endoplasmic Reticulum) stress using Tunicamycin leads to similar levels of neuron survival post-restoration compared to NGF deprivation, and that a 2-hit experiment of the NGF deprivation model led to increased neuron survival compared to neurons subjected to only one round of deprivation. Together, these results indicate that neurons have the ability to reverse from multiple apoptotic triggers. Additionally, we found that neurons that survive an apoptotic insult are better protected from a subsequent insult.