Glucocorticoid-induced Stress Mechanism in Mouse Primary Neurons

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Introduction: Depression is a disease that effects an estimated 5% of adults globally, and is the leading cause of disability worldwide. This study aimed to examine whether inducing a depression-like state caused any changes in gene expression or axon length in cultured neurons.

Methods: To accomplish this primary E18 cortex neurons were cultured for a total of 11 days in Poly-D-Lysine coated six well plates. The cells were grown in NbActiv1 for the duration of the experiment. The media was changed on day four. After 8 days, the neurons were treated with either Dexamethasone, RU 486 + Dexamethasone, or received no treatment. RU 486 is a progestin antagonist that was used to block the effects of Dexamethasone. Treatments were reapplied on day 10 and cells were harvested for RNA extraction 24 hours later. One plate of cortex neurons was grown in an IncuCyte incubator to allow axon length to be continuously measured by NueroTrack software.

Results: Controls had an average of 69.36 mm/mm2 and a variance of .71. Dexamethasone had an average length of 68.87 mm/mm2 with a variance of 2.05. RU 486 and Dexamethasone had an average of 70.27 with a variance of .97. A p value of .00115 was calculated for the data.

Conclusion: It was concluded that dexamethasone does reduce the axon lengths of neurons, and that it can be used to induce a depression like state in neuron culture. Next, gene expression profiling of these neuron cultures will be characterized by RNA sequencing.

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