The Effect of Nicotine on Embryonic GABA Neurite Morphology

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Nicotine is a highly addictive drug that has been found to disrupt gamma aminobutyric acid (GABA) neuron migration in the developing cerebral cortex. This project investigates if GABA neuron morphology is affected by nicotine exposure. Mouse embryos were dissected on embryonic day 15 (E15). Tail and brain samples were harvested by my researcher. I then performed genotyping with PCR and gel electrophoresis on the tail samples to collect data on each embryo's sex. The embryonic brain explants were exposed to nicotine via cell culture. I then imaged the results, taking images from the rostral region. A box was overlaid onto the image at the beginning of the explant to the edge of the farthest migrated neuron. I collected data on the number of branching points, whether it was multipolar for each neuron, and neurite length. I counted as many neurons as could be clearly seen in each image. I then ran two-way ANOVAS for percent of multipolar neurons, branching points per neuron, and neurite length. All tests yielded no significant findings as the p-values were above 0.05. The nicotine males did have slightly longer primary neurites compared to control males ($p=0.06$), but this did not reach significance after running a Tukey's post hoc comparison. I also ran a 3-way ANOVA for percent of multipolar neurons over distance, which revealed a significant interaction between sex and distance. However, a Tukey's post hoc comparison was conducted and none of the comparisons reached significance. These findings indicate that neuron morphology is not the mechanism of migration that nicotine affects.

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