OMICS Group Conferences
Accelerating Scientific Discovery

3rd World Congress on
Cell Science & Stem Cell Research
November 20-22, 2013 DoubleTree by Hilton Baltimore-BWI Airport, MD, USA

Nicotine-induced toxicity in rat embryonic neural stem cells

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Maternal smoking substantially increases the risk of learning disabilities, behavioral problems, and attention deficit/hyperactivity disorder in offspring. Nicotine is the main pharmacologically active component of tobacco smoke. Prenatal exposure to nicotine is capable of causing fetal brain damage. To evaluate nicotine's effects on the developing nervous system and explore potential mechanisms underlying such toxicity, rat embryonic neural stem cells were used.

Brain cortices were collected from fetal rats (gestational day14, GD14) for neural stem cell isolation and subsequent culture in commercial rat growth medium. On the 8th day *in vitro* (DIV), confluent neural stem cells were exposed to nicotine at concentrations of 0.5, 1.0, 2.0, 5.0 and 10 μM for 24 hours. Neural stem cells were identified using monoclonal anti-nestin antibody. Markers of cellular proliferation (EdU), mitochondrial health (MTT), cell death/damage (LDH) and immunohistochemical staining of activated caspase 3 were monitored to determine the nature of nicotine-induced neurotoxicity.

Nicotine treatment resulted in a substantial dose-dependent reduction in mitochondrial function as evidenced by significant decreases in the metabolism of MTT. No significant effect of nicotine on LDH release was observed. 1 μM nicotine significantly increased the expression of activated caspase 3, suggesting that nicotine induced neural stem cell apoptosis.

These results suggest that nicotine decreased neural stem cell viability, and nicotine-induced cell death is probably apoptotic in nature.

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