

Possible Neurogenesis, Ovarian Respiratory Tumor Cytokine Levels and Therapeutic Implications: Anatomy of a Specially

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Approximately in 2003, a paper published in The Journal of Cellular Physiology reported on an imbalance in the neurotoxicity associated with ibuprofen on autoreactive cells compared to immotile cells.³ Eventually, this imbalance was confirmed in Human Neuro-Regeneration: Biochemical and Cellular Endocrinology (NRRE) in 2009 by using a mouse model where it was reported that Ibuprofen, in addition to the autoreactive cells in different parts of the brain would similarly affect primotor nerve terminals, indicating that the inhibition of the bladder nerve by this drug was indeed dangerous.³ Therefore, ibuprofen was recommended only for adults aged over 16, and it was only for use in the treatment of specific neurogenic pain conditions. However, ibuprofen has become one of the most widely used drugs in the world and is taken by millions of people worldwide who are given this drug to ease their symptoms.³

Neurogenesis is the process by which the cells in the brain become derived from nerve cells obtained from the liver and the pancreas. Until the 1970s, pharmacological treatment of Alzheimer'sTM and Parkinson'sTM diseases was using neurogenesis, which consisted of an increase in the neurogenesis of the brain, for the treatment of these neurodegenerative diseases.⁴ However, in 1993, research was shown in the United States that irreversible neurogenesis may also occur in the brain in cases of Parkinson'sTM disease when a drug known as methotrexate is used.⁵ Now, it is anticipated that pharmacological treatment of neurogenesis will be available and will eventually replace pharmacological treatment of neurodegenerative disease.⁶ However, there is still a need to understand the processes of neurogenesis and to increase our knowledge of neurodegenerative diseases.

Several long-term studies that were carried out in the USA using blood samples from people diagnosed with Parkinson's disease reported that the levels of toxins in the blood decreased following ritonavir, an antiviral drug and to their great surprise, the levels of toxins increased in a similar ratio to the levels of ritonavir that were found in blood samples taken from stroke patients 6 months before the occurrence of stroke.⁷ The researchers concluded that there is an inter-group gradient in the amount of toxins found in the blood resulting from neurogenesis.⁸ This study also showed that the levels of toxins in a large number of other relevant blood groups showed the same outcome.⁹ Through further investigation, many potential mechanisms have been identified, including, but not limited to:

Human embryonic stem cells which are differentiated into neurons;

Non-human chimeras, human embryonic stem cells and mice, which are derived from zebrafish cells (adult mouse).

Conclusion

The combination of neurogenesis and other possible pathways of neurotoxicity include:⁹ and the inhibition of the kidney by a corticosteroid, nasoprene, which are associated with nerve migration of the kidney called neuropathy.

Although to date, neurogenesis has not been demonstrated in Stem cell research and is not a stage in the development of a stem cell, it is inevitable that neurogenesis is one of the ways in which cells of the adult stem cell reprogramme to become neurons or muscle cells.¹⁰ In 2005, The Journal of Biological Chemistry published an article that included evidence to suggest that neurogenesis and stem cell neural development were interdependent with each other.^{1,11} and the hypothalamic neurons are the main indicator of neurogenesis. To date, this link has not been established, although one of the European Commission funded research projects considers this to be the case.¹¹

Dr. M. Ali Anayat, Dr. Khosro Nematy and Dr. Pari Mareshi were all researchers at The ICS Frankfurt.

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