The 2nd International Neurotrauma Congress & the 4th International Roads Safety Congress

Shefa Neuroscience Research Center, Tehran, Iran, 18-20 February, 2015

The Neuroscience Journal of Shefaye Khatam

Volume 2, No. 4, Suppl. 3

Poster Presentation

Traumatic Brain Injury and Genes

Hadi Aligholi, Fatemeh Attari, Sayed Mostafa Modarres Mousavi, Babak Khodaie*

Shefa Neuroscience Research Center, Khatam Alanbia Hospital, Tehran, Iran.

Published: 18 February, 2015

Abstract

ثفار فتم

Traumatic brain injury (TBI) is one of the serious neurodisorders commonly caused by car accidents and sports. Preventive measures are highly recommended to reduce the risk and number of TBI cases. After TBI, the primary injury to the brain initiates a secondary injury process that spreads via multiple molecular mechanisms in the pathogenesis of TBI. Many studies in animals using cDNA microarray hybridization technique have shown differential regulation of 86 genes (mainly transcription factors, signal transduction genes and inflammatory proteins) which take part in the physiological and pathological response to TBI. Genetic polymorphisms which involve interleukin-6 (such as-174G>C and -572G>C) and haemoxygenase -1 may influence the inflammatory effects seen after TBI. In addition, genes regulating the vascular responses including the hypoxia-inducible factor-1 and 2 (HIF1&2) genes activate fallowing cerebral ischemia. Moreover, Apolipoprotein epsilon and P53 genes regulate the neuronal response to TBI. Also, there are three isoforms of the enzyme catechol-o-methyltransferase (COMT) encoded by 3 genetic polymorphisms (COMT Val/Val, COMT Val/Met, and COMT Met/Met) which regulate catecholamines activity fallowing TBI. A greater understanding of the genetics could aid in the prediction of outcomes and could be targeted for treatment strategies.

Keywords: Catecholamines, Car Accident, Ischemia, Traumatic Brain Injury.

*Corresponding Author: Babak Khodaie

E-mail: vet_babak@yahoo.com