The 2nd International Neuroinflammation Congress and 2nd Student Festival of Neurosience

Shefa Neuroscience Research Center, Tehran, Iran, 17-19 April, 2018

The Neuroscience Journal of Shefaye Khatam

Volume 6, No. 2, Suppl 1

Poster Presentation

LPS Preconditioning Declines Oncogenic Factors and Inflammatory Responses in PC12 Cells

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Published: *17 April, 2018*

Abstract

The release of molecules from injured tissue leads to produce inflammatory response that can result in apoptosis and cell death. Preconditioning (PC) can decrease the inflammatory response, increase neuroprotective mechanism on different levels. So, we investigated the role of PC as a suitable preventative approach in neurodegenerative disease in and inflammatory oncogenic factors in PC12 cells. So, we treated differentiated PC12 cells with ultra-low and high doses LPS 3µg/ml and 750µg/ml respectively. Our results showed that C-myc and IL-1β, were significantly increased in high dose LPS respect to the control. In addition, C-myc was enhanced despite the inhibition of apoptosis even if cells were treated by high dose LPS. But results have shown that C-myc level was markedly reduced in presence of PC induction respect to the high dose LPS group. C-myc in the PC group in compare with the control has shown no significant difference. Despite of the apoptosis inhibition in the PC group, C-myc level was not significantly increased. Further evidences have shown that IL-1β in the preconditioned cells were significantly decreased in compared with high dose LPS group and PhosphoSer46P53/P53 significantly decreased in PC group in presence of the apoptosis inhibitor compared with PC group. It has concluded that PC could be effectively reduced the level of inflammatory responses and oncogenic factors. Some PC agent like ultra-low dose LPS causes gene reprogramming which can induce neuroprotection and decrease proinflammatory responses.

Keywords: Preconditioning, LPS, Inflammatory Responses

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