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**Title:** Effect of epigallocatechin-3-gallate on inflammatory mediators release in LPS-induced Parkinson's disease in rats

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**Abstract:** Degeneration of dopamine (DA)-containing neurons in the substantia nigra of the midbrain causes Parkinson's disease (PD). Although neuroinflammatory response of the brain has long been speculated to play a role in the pathogenesis of this neurological disorder, the mechanism is still poorly understood. The aim of the present study was to examine the effect of epigallocatechin-3-gallate (EGCG) in prevention of inflammatory mediators release and protection of dopaminergic neurons from lipopolysaccharide (LPS)-induced neurotoxicity. A single intraperitoneal injection of LPS (15 mg/kg) in male Sprague Dawley rats resulted in an increase of midbrain content of TNF-alpha, NO and a decrease of DA level at 4, 24 h, 3 and 7 days compared to the control. In addition, LPS reduced the number and the density of tyrosine hydroxylase-immunoreactive (TH-ir) neurons in the midbrain at 7 days. Pretreatment with EGCG (10 mg/kg) 24 h before LPS for 7 days decreased TNF-alpha and NO compared to LPS-treated rats. Moreover, it increased DA level and preserved the number and the density of TH-ir neurons compared to LPS group. In conclusion, EGCG was found to have a potential therapeutic effect against LPS-induced neurotoxicity via reducing TNF-alpha and NO inflammatory mediators and preserving DA level in midbrain.

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