Andrographolide downregulates pro-inflammatory cytokines and free radical productions to prevent dopaminergic neuro-degeneration induced by lipopolysaccharide

Abstract

Oxidative stress and inflammation triggers the production of free radicals and toxic pro-inflammatory cytokines leading to neurodegeneration. Down-regulation of oxidative stress and inflammatory mediators offers defence against development and progression of the disease. Andrographolide is the most abundant diterpene lactone isolated from the leaves of Andrographis paniculata. Despite the increasing literature studies on the anti-inflammatory effect of andrographolide, there is still paucity regarding its neuroprotective role as can be ascertained from the search of literature. The present study investigated the potential therapeutic role of andrographolide as a neuroprotective agent via in vitro model of lipopolysaccharide (LPS)-induced brain injury using microglial (BV2) and dopaminergic (N27) cells. Pre-treatment of andrographolide at non-cytotoxic concentration range (0.25-2.0 μM) of BV2 cells followed by LPS dose-dependently suppressed nitric oxide production, pro-inflammatory cytokines (TNF-α, IL-6) as well as attenuating intracellular reactive oxygen species (ROS) and thiobarbituric acid reactive substance (TBARS) compared to cells without andrographolide pretreatment. Further, conditioned media (CM) of the pretreatment had diminished toxicity towards dopaminergic neurons as compared to CM without pretreatment. Keywords: Andrographolide; Proinflammatory cytokines; oxidative stress; microglial; dopaminergic neurodegeneration.

Keyword: Andrographolide; Proinflammatory cytokines; Oxidative stress; Microglial; Dopaminergic neurodegeneration