Modulation of LPS-Induced Nitric Oxide Production by a Ca2+ Channel Blocker

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Background & Objectives: Lipopolysaccharide (LPS) as an endotoxin is a strong stimulator of nitric oxide (NO) production in monocytes/macrophages. NO has been shown to be an important inducer of inflammation. Verapamil, a calcium channel blocker, has been widely used in treatment of cardiovascular diseases. Moreover the anti-inflammatory properties of verapamil have been demonstrated. In this study the effect of verapamil on LPS-induced NO production in a human monocytic cell line has been investigated in vitro.

Methods: The human monocytic THP1 cells were cultured in complete RPMI medium. The cells at logarhytmic growth phase were stimulated with LPS at optimum concentration and then incubated with different concentrations of verapamil (0.001-1000 µg/ml). Afterward the NO production in cell culture supernates was measured by the Griss assay.

Results: Verapamil significantly decreased the NO production in LPS-stimulated THP1 cells in a dose-dependent manner.

Conclusion: The results of the present study showed that verapamil down-regulates the NO production in human monocytic THP-1 cells. So the anti-inflammatory properties of verapamil may be partly due to its inhibitory effects on NO production. Verapamil might have potential implication in planning of therapeutic approaches for NO-mediated inflammatory disorders.

Keywords: Verapamil; Lypopolysaccharide; Nitric Oxide; Monocytes