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Acetylcorynoline Impairs the Maturation of Mouse Bone Marrow-Derived Dendritic Cells

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Abstract

Background: Dendritic cells (DCs) are major modulators in the immune system. One active field of research is the manipulation of DCs as pharmacological targets to screen novel biological modifiers for the treatment of inflammatory and autoimmune disorders. Acetylcorynoline (ACL) is the major alkaloid component derived from Corydalis bungeana herbs. We assessed the capability of ACL to regulate lipopolysaccharide (LPS)-stimulated activation of mouse bone marrow-derived DCs. Methodology/Principal Findings: Our experimental data showed that treatment with up to 20 M ACL does not cause cytotoxicity in cells. ACL significantly inhibited the secretion of tumor necrosis factor-α, interleukin-6, and interleukin-12p70 by LPS-stimulated DCs. The expression of LPS-induced major histocompatibility complex class II, CD40, and CD86 on DCs was also decreased by ACL, and the endocytic capacity of LPS-stimulated DCs was restored by ACL. In addition, LPS-stimulated DC-elicited allogeneic T-cell proliferation was blocked by ACL, and the migratory ability of LPS-stimulated DCs was reduced by ACL. Moreover, our results confirmed that ACL impairs the responses of LPS-stimulated activation of DCs through suppression of IκB kinase and mitogen-activated protein kinase activities. Coadministration of ACL with 2,4-dinitro-1-fluorobenzene prevented 2,4-dinitro-1-fluorobenzene-induced delayed-type hypersensitivity. Conclusions/Significance: ACL may be a new potent immunosuppressive agent and could be used in the prevention and treatment of inflammation, and autoimmunity through the blockage of DC maturation and function.