[PA1-41] [10/18/2002 (Fri) 09:30 - 12:30 / Hall C]

Immunomodulatory activity of a polysaccharide isolated from cell culture of Acantopanax senticosus

Ahn HyoJung°, Han SangBae, Yoon YeoDae, Lee HaengSoon, Moon JaeSun, Lee ChangWoo, Lee KiHoon, Park SongKyu, Kim HyungChin, Kim HwanMook

Biopotency Evaluation Laboratory, Korea Research Institute of Bioscience Biotechnology (KRIBB)

Many polysaccharides isolated from plants are considered to be biological response modifiers and have been shown to enhance various immune responses in vivo and in vitro. Here we demonstrate that a polysaccharide isolated from cell culture of Acanthopanax senticosus (AS) has a unique mode of immunostimulation with regard to its cell-type specificity. AS was found to markedly increase polyclonal IgM antibody production and the proliferation of B cells, and to activate iNOS transcription and NO production in macrophages. However, AS did not affect the proliferation of T cells, the IL-2 expression of Th1 cells, or the IL-4 expression of Th2 cells. Although AS and lipopolysaccharide (LPS) had a similar mode of action in B cells and macrophages, they were differentiated by the fact that AS-induced cellular activation was not inhibited by polymyxin B, a specific inhibitor of LPS. AS activity in B cells from C3H/HeJ, known to have a defective TLR4, was decreased in comparison with that in control B cells from C3H/HeN mice. Anti-TLR2, anti-TLR4, anti-CD19 and anti-CD79b, but not anti-CD38, antibodies blocked B cell proliferation, indicating the possible cellular binding sites of AK. Our results demonstrate that AS is a specific activator of B cells and macrophages but not of T cells, and suggest that AS is quite distinct from other well-known immunostimulants, such as lentinan and schizophyllan, which mainly act upon macrophages and T cells.

[PA1-42] [10/18/2002 (Fri) 09:30 - 12:30 / Hall C]

PLATYCODIN D, NOT D3, CAUSES VASODILATATION IN THE RAT

DONG-YOON LIM, YEONG-WOO KEE, EUN-BANG LEE

Department of Pharmacology, College of Medicine, Chosun University, Gwangju 501-759, Korea; Natural Products Research Institute, Seoul National University, Seoul 110-460, Korea

The purpose of the present study was to determine the effects of platycodin D and D3 on the contractile force of the isolated rat aorta and blood pressure of the anesthetized rat, and also to establish the mechanism of action.The phenylephrine (10 μM)-induced contractile responses were greatly inhibited in the presence of platycodin D (4 ~ 24 μg/ml) in a dose-dependent fashion. Also, high potassium (56 Mm)-induced contractile responses were depressed in the presence of platycodin D (24 μg/ml), but not affected in low concentration of platycodin D (4 ~ 8 μg/ml). However, Platycodin D3 (8 ~32 μg/ml) did not affect the contractile responses evoked by phenylephrine and high K+. Interestingly, the infusion of a moderate dose of Platycodin D (1.0 mg/kg/30 min) made a significant reduction in pressor responses induced by intravenous norepinephrine. However, Platycodin D3 (1.0 mg/kg/30 min) did not affect them. Collectively, these results demonstrate that intravenous Platycodin D dose-dependently depresses norepinephrine-induced pressor responses in the anesthetized rat at least partly through the blockade of adrenergic α-receptors. Platycodin D, given intravenously, elicited rapid and temporary hypotensive responses in a dose-dependent fashion. Platycodin D also causes vascular relaxation in the isolated aortic strips of the rat via the blockade of adrenergic α-receptors, in addition to the unknown direct vasorelaxation. However, Platycodin D3 did not affect both norepinephrine-induced pressor responses, and also the isolated rat aortic contractile responses evoked by phenylephrine and high potassium. Based on these results, it seems likely that there is much difference in mode of action between Platycodin D and Platycodin D3.