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Effects of β-Glucan on the Release of Nitric Oxide by RAW264.7 Cells Stimulated with Escherichia coli Lipopolysaccharide

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Abstract : This research analyzed the effect of β-glucan that is expected to alleviate the production of inflammatory mediator in macrophagocyte, which was processed by the lipopolysaccharide (LPS) of Escherichia, a pathogen related to allergy. The incubated layer was used for nitric oxide (NO) analysis. The DNA-binding activation of the small unit of NF-κB was measured using ELISA-based kit. In RAW264.7 cells that were vitalized by E.coli LPS, β-glucan inhibited both the combatant and rendering phases of inducible NO synthase (iNOS)-derived NO. β-glucan increased the expression of heme oxygenase-1 (HO-1) in the cell that was stimulated by E.coli LPS, and HO-1 activation was inhibited by SnPP. This shows that NO production induced by LPS is related to the inhibition effect of β-glucan. The phosphorylation of JNK and p38 induced by LPS were not influenced by β-glucan, and IκB-α decomposition was not influenced either. Instead, β-glucan remarkably inhibited the phosphorylation of STAT1 that was induced by E.coli LPS. Overall, β-glucan inhibited the production of NO in macrophagocyte that was vitalized by E.coli LPS through HO-1 induction and STAT1 pathways inhibition in this research. As the host inflammation reaction control by β-glucan weakens the progress of allergy, β-glucan can be used as an effective treatment method.

Keywords : β-glucan, lipopolysaccharide (LPS), nitric oxide (NO), RAW264.7 cells, STAT1
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