Activation of The Innate Immune System

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Heat-killed *Enterococcus faecalis* TH10 (hk-TH10) stimulates macrophage cells for immunoregulation.

Objective

We examined the immunomodulatory effects of heat-killed *Enterococcus faecalis* TH10 (hk-TH10) on murine macrophage RAW264 cells.

Methods

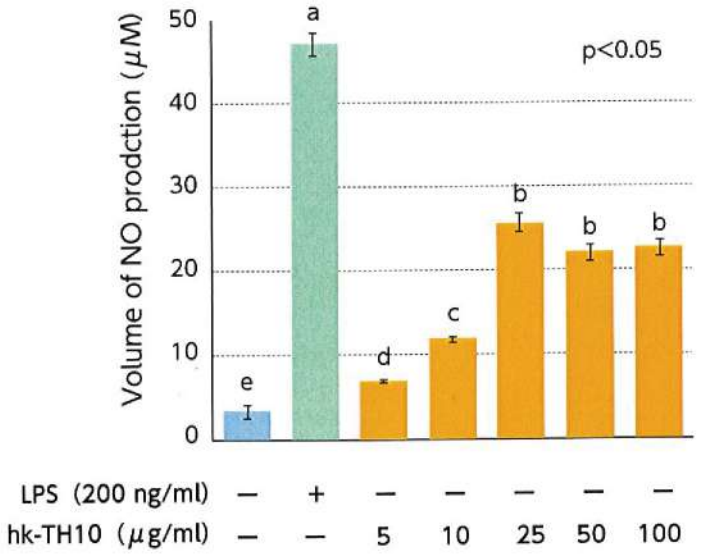
We measured the amount of nitric oxide (NO) produced from a cell in order to examine the macrophage activity which is an index of activation of the innate immune system. We utilized Toll-like receptors (TLRs) knocked down by small interfering RNA (siRNA) in order to determine the pathway in cells where hk-TH10 stimulates macrophage activity.

Results

When we set lipopolysaccharide (LPS) that is known as an enhancer of NO production in cells as a positive control, hk-TH10 stimulated NO production up to a level that is almost half of what LPS does. LPS is a strong enhancer. Therefore, we consider that hk-TH10 has a moderate macrophage activation potency.

According to the result from the experiment using siRNA, macrophage RAW264 cells recognized the passage of hk-TH10 through TLR-2 and TLR-6 pathways, and hk-TH10 stimulated NO production via the activation of NF-kB.

Nitric oxide (NO)-producing ability of heat-killed *E. faecalis* TH10



These results revealed that hk-TH10 stimulates NO production from the macrophage cells and adjusted the innate immune system.