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Exhaustive exercise enhances immune response to flagellin via adrenaline-mediated up-regulation of TLR5 expression

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ABSTRACT

Objective: We already reported that lipopolysacchride (LPS)-induced tumor necrosis factor (TNF)-α production as a bacterial infection model to induce immune response, was inhibited by exhaustive exercise. However, it remains unclear whether or not the immune response to flagellin (FG), which binds to toll like receptor 5(TLR5) and induces pro-inflammatory cytokine production, is also inhibited by this severe exercise. The aim of this study was to determine whether or not exhaustive exercise affects TNF-α productions after FG injection in mice. Methods: Both exhaustive-exercised (EX; n=12) and non-exercised (N-EX; n=12) male C3H/HeN mice were injected with FG (1 mg/kg, i.v), and blood samples were collected. In addition, to clarify the effect of catecholamine on immune response macrophage and intestinal cells after FG stimulation, RAW264 cells and Caco2 cells were cultured 30min after propranolol (Prop; β-adrenergic receptors blocker) or Ly294002 (Ly; PI3K inhibitor) treatments, and were then stimulated with adrenaline (AD; 1 μM) and FG (5 μg/ml). Moreover, the effect of Prop (10 mg/kg, n=12) on FG-induced TNF-α production in EX mice was also examined. Results: TNF-α in EX group was significantly higher than that in N-EX group after FG injection. In epithelium cells, more intensity of TLR5 localization was observed on the plasma membrane area than in the cytosol area in EX mice, but not N-EX mice. Caco2 cells, but not RAW264 cells, significantly increased the FG-induced TNF-α production using AD treatment. Moreover, Prop treatment attenuated the AD-induced TNF-α production in response to FG in Caco2 cells. Although TLR5 expression on RAW264 cells was significantly decreased after AD treatment, the expression on Caco2 cells was rapidly increased. In fact, we observed the AD-dependent TLR5 translocation from cytoplasm to cell membrane in Caco2 cells, and the membrane translocation was inhibited by Prop and Ly treatment. Moreover, the pretreatment with Prop attenuated the exercise-induced plasma TNF-α response to FG in vivo. Conclusion: Our results suggest that immune response to FG via TLR5 might be enhanced by exhaustive exercise in mice.