Abstract

Lipopolysaccharide (LPS) is an endotoxin found in gram-negative bacteria such as *Escherichia coli* (*E. coli*). LPS protects the bacterial cell membrane; however, if the bacteria dies, LPS is released into the tissue where it activates the innate immune system and triggers an inflammatory response. Macrophages are large phagocytes that engulf pathogens, activate leukocytes and release inflammatory mediators such as nitrite (NO$_2^-$) and cytokines. The purpose of this study was to measure the macrophage inflammatory response against LPS *in vitro*. We hypothesized that culturing macrophages with LPS would increase the production of nitrite (NO$_2^-$) and the pro-inflammatory cytokine, tumor necrosis factor alpha (TNF-α). In these experiments, murine RAW 264.7 cells were grown in Dulbecco’s modified eagle high glucose medium with 10% fetal bovine serum at 37°C under 5% CO$_2$. Cells were cultured for one hour to allow cells to acclimate. After one hour, LPS was added (500ng/well) and the cells were cultured for 24 hours. Cell-free supernatants were assayed for nitrite (NO$_2^-$) using the Griess reagent. The concentration of TNF-α was measured using an ELISA. LPS increased the release of inflammatory mediators from RAW 264.7 macrophages. Specifically, LPS increased the production of NO$_2^-$ approximately 7-fold and TNF-α approximately 17-fold. In future studies, *in vitro* experiments will be used to test the inhibitory effect of anti-inflammatory therapeutics on macrophage activity.

*Keywords*: macrophages, lipopolysaccharide, inflammation