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## Scavenger Receptor A Activation Enhances Peptidoglycan-Stimulated Interleukin-8 Release In Bronchial Epithelium

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**INTRODUCTION:** The immune response to certain pathogens or cellular stressors is initiated and regulated by pattern recognition receptors of the innate immune system. Pattern recognition receptors are effective by utilizing either endocytic (Scavenger Receptors) or signaling receptors (Toll-like Receptors). It has been previously demonstrated that mouse dendritic cells deficient in scavenger receptor A (SRA) negatively regulated the immune response produced by Toll-like receptors when exposed to modified lipid proteins. Previously, we have shown that peptidoglycan contained in organic dust from swine barns stimulates pro-inflammatory cytokine release in bronchial epithelium. We hypothesized bronchial epithelial SRA would also have a regulatory role in the cytokine response produced by toll-like receptors.

**METHODS/MATERIALS:** Primary and immortalized (Beas-2B) human bronchial epithelial cells were grown in serum-free media to 70% confluency. Cells were incubated with 100 µg/ml fucoidan (a known SRA ligand), 100 µg/ml fetuin (a negative control for SRA), and 100 µg/ml chondroitin sulfate (a known SRB ligand) for 24 hours. Each sample was then incubated with 10 µg/ml of peptidoglycan or media for 24 and 48 hours. The media was then collected and assayed for IL-8 using a standard sandwich ELISA.

**RESULTS:** IL-8 production was significantly increased in cells pre-treated with fucoidan followed by peptidoglycan as compared to either media control (3-fold increase) or peptidoglycan only (2-fold increase). Cells treated with fucoidan, fetuin, or chondroitin sulfate demonstrated no significant increase in IL-8 production vs. media control. Cells pre-treated with fetuin or chondroitin sulfate followed by peptidoglycan produced no enhanced increase in IL-8 production when compared to peptidoglycan alone.

**CONCLUSIONS:** These data show that peptidoglycan can stimulate IL-8 production maximally at 24 hours. When human bronchial epithelium is first treated with a SRA ligand, fucoidan, IL-8 release is increased. Increased production of IL-8 by bronchial epithelium under these conditions suggests that SRA has a co-stimulatory relationship with TLR-2. These results suggest that receptor crosstalk may have a role in the airway inflammatory response to inhaled Gram-positive bacteria.

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