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Heterogeneous LPS of *Porphyromonas gingivalis* differentially modulate the innate immune response of human gingiva

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Objective

Porphyromonas gingivalis lipopolysaccharide (PgLPS) is a crucial virulence factor strongly involved in chronic periodontitis. PgLPS is known to contain both tetra- (Pg LPS1435) and penta-acylated (PgLPS 1690) lipid A structures with opposing effects. Present study aimed to examine the effect of two Pg LPS isoforms on human gingival epithelium.

Methods

Reconstituted human gingival epithelia (RHGE) were challenged with two isoforms of PgLPS together with *E. coli* LPS as the positive control. mRNA and proteins were harvested from tissues and culture supernatants were collected. Expression of pro-inflammatory and anti-inflammatory cytokines was evaluated by Q-PCR and ELISA. Involvement of pattern recognition receptors and signaling pathways were also analyzed by Q-PCR and western blot. Next, RHGE was blocked for CD14, TLR2, and TLR4 and followed by stimulation of PgLPS isoforms and effect was evaluated at cytokine level by Q-PCR and ELISA. Furthermore, we used "tissue proteomics" approach to study the differential proteomic expression profiles of gingival epithelium upon Pg LPS stimulation.

Results

It was shown that penta-acylated PgLPS1690 significantly upregulated the secretion of pro-inflammatory cytokines IL-1β, IL-6, IL-8 and TNF-α in RHGE compared to tetra-acylated PgLPS1435. It seemed that regulation of pro-inflammatory cytokine by PgLPS1690 is mediated through both TLR2 and 4 and CD14/NF-kB axis for most of the cytokines. Proteomic studies indicated a differential protein profiles of RHGE induced with two isoforms.

Conclusion

P. gingivalis LPS heterogeneity differentially modulates the host innate immune response in human gingival epithelium, which may explain the niche-specific pathogenic mechanism of this periodontal pathogen.

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