

POSTER PRESENTATION

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

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From Institut Pasteur International Network Annual Scientific Meeting  
Hong Kong. 22-23 November 2010

## 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-1 $\beta$ , IL-6, IL-8 and TNF- $\alpha$  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.

## Acknowledgements

Supported by grants GRF 7518/05M and GRF HKU766909M to LJJ.

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Published: 10 January 2011

doi:10.1186/1753-6561-5-S1-P86

**Cite this article as:** Herath et al.: Heterogeneous LPS of *Porphyromonas gingivalis* differentially modulate the innate immune response of human gingiva. *BMC Proceedings* 2011 5(Suppl 1):P86.

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