

# 0898 *P. gingivalis* LPS Modulates Antimicrobial Peptide Expression in Gingival Epithelia

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**Objectives:** This study aimed to determine whether *P. gingivalis* LPS could modulate the gingival epithelial expression of human  $\beta$ -defensins (hBDs) through the pattern recognition receptors. **Methods:** Reconstituted human gingival epithelia (RHGE) were incubated with 10  $\mu$ g/ml of *P. gingivalis* LPS or 10 ng/ml of *E. coli* LPS for 2, 6, 12, 18, 24, 36, 48, 72 and 96 h. In the blocking assay, the RHGE were pre-incubated with TLR-2 and -4 and CD14 antibodies. The expression of hBDs 1-3, CD14, LBP, TLR-2 and -4, MD-2 and MyD88 mRNAs in RHGE was detected by RT-PCR, while hBDs peptides were detected by immunohistochemistry and ELISA. **Results:** The expression of hBD-2 mRNA was significantly enhanced by *P. gingivalis* LPS in a time-dependant manner within 36 h and remained up to 96 h, which was approximately 3 times higher than that of hBD-1 and -3. Whereas *E. coli* LPS induced a prompt expression of hBD-2 and -3 messages which decreased to basal levels within 36 h. The hBDs peptides were detected in RHGE stimulated with *E. coli* LPS but not in those challenged by *P. gingivalis* LPS. The basal expression of CD14, MD-2 and MyD88 mRNAs was not affected by *P. gingivalis* or *E. coli* LPS. Blockage of CD14, TLR-2 and -4 functions significantly inhibited *E. coli* LPS-induced upregulation of hBD-2 mRNA and peptide expression. However, although combined blockage of TLR-2 and -4 or plus CD14 functions markedly inhibited *P. gingivalis* LPS-induced upregulation of hBD-2 mRNA, it resulted in an increased expression of hBD-2 peptide. **Conclusions:** *P. gingivalis* LPS could enhance the *in vitro* expression of hBDs mRNAs in gingival epithelia likely with the involvement of TLR-2 and -4. While it might evade innate defense surveillance by interference of hBD-2 peptide induction. Supported by Hong Kong Research Grants Council (RGC HKU 7310/00M & 7518/05M).

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