

# **2327 Expression of LBP, CD14 and TLR-2/4 in Human Chronic Periodontitis**

**L.J. JIN**<sup>1</sup>, L. REN<sup>1</sup>, W.K. LEUNG<sup>2</sup>, and R.P. DARVEAU<sup>3</sup>, <sup>1</sup>University of Hong Kong, Hong Kong, <sup>2</sup>University of Hong Kong, Hong Kong SAR, China, <sup>3</sup>University of Washington, Seattle, USA

**Objectives:** This study aimed to investigate the interrelationship of LBP and CD14 expression in human gingiva as well as the co-expression of TLR-2 and -4 in association with periodontal health and disease. **Methods:** Gingival biopsies were collected from 43 subjects with chronic periodontitis, including periodontal pocket tissues (PoTs) and clinically healthy tissues (HT-Ps), and from 15 periodontally healthy subjects as controls (HT-Cs). The protein expression of LBP, mCD14, TLR-2 and -4 was detected by immunohistochemistry, while the LBP and CD14 mRNAs were detected by RT-PCR. CD68 and CD1a were co-detected with mCD14, respectively. **Results:** LBP and mCD14 peptides were simultaneously detected in 91% of PoTs and 85% of HT-Ps, and in 100% of HT-Cs. LBP and mCD14 mRNAs were co-detected in 55% of PoTs, 55% of HT-Ps and 75% of HT-Cs. The expression of LBP was mainly confined to the gingival epithelium, while mCD14 was observed around the epithelium-connective tissue interface. The expression levels of both LBP and mCD14 in HT-Cs were significantly higher than those in PoTs ( $p < 0.05$ ). A positive correlation existed between LBP and mCD14 ( $r = 0.304$ ,  $p < 0.05$ ). In PoTs, TLR-2 was detected in both pocket epithelia and the macrophage-like cells in connective tissues; while TLR-4 was predominantly detected in connective tissues. In HT-Ps and HT-Cs, only a weak expression of TLR-2 could be found in gingival epithelia and no TLR-4 expression was detected. In PoTs, mCD14 was co-detected on CD68-labelled macrophages in the connective tissues beneath pocket epithelium as well as on CD1a-labelled dendritic cells in the epithelium and connective tissues interface. No similar expression was detected in HT-Ps and HT-Cs. **Conclusions:** The present study implies that inappropriate expression of LBP and mCD14, coupled with altered expression profiles of TLR-2 and -4, may be related to periodontal pathogenesis. Supported by Hong Kong Research Grants Council (RGC HKU 7310/00M).

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