

## **0575 Interrelated Expression of Lipopolysaccharide-binding Protein and CD14 in Subjects with Chronic Periodontitis**

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Bacterial lipopolysaccharide (LPS) triggers host cells to synthesize and release a cascade of inflammatory mediators through a pattern recognition system. Lipopolysaccharide-binding protein (LBP) and CD14 as two key effector molecules of the system, their *in vivo* expression levels and clinical implications are unknown. Objectives: To investigate the expression of LBP and CD14 in gingival tissues and to assess their interrelationship in periodontally healthy and diseased conditions. Methods: Gingival biopsies were collected from 12 subjects with chronic periodontitis during periodontal surgery, consisting of 12 pairs of periodontal pocket tissues (PoT, probing depth 6-10 mm) and the adjacent healthy gingival tissues (HT-P, probing depth 2-3 mm). Seven gingival biopsies were obtained from 7 periodontally healthy controls (HT-C). LBP and CD14 were detected by immunohistochemistry and quantitatively analyzed with a computerized image processing system. Results: LBP and CD14 were co-detected in 11 of 12 PoTs, 10 of 12 HT-Ps and all HT-Cs. LBP expression was mainly confined to the gingival epithelium, while CD14 expression was predominant in the epithelium-connective tissue interface. The expression levels of both molecules were significantly higher in HT-C than in PoT ( $p < 0.05$ ) or HT-P ( $p < 0.05$ ). Overall, CD14 expression level was correlated with probing depth ( $r = -0.610$ ,  $p = 0.0001$ ). The CD14 expression in HT-Ps was higher than in the adjacent PoTs ( $p < 0.05$ ), while CD14 expression in these paired samples was significantly inter-correlated ( $r = 0.638$ ,  $p = 0.0257$ ). Within the HT-Ps, CD14 expression was significantly correlated to LBP ( $r = -0.703$ ,  $p = 0.0108$ ), while no similar finding was noted in PoTs. Conclusions: The local expression levels of LBP and CD14 may be related to periodontal conditions. Their interrelated expression may be crucial in local host responses to bacterial challenge. Supported by the Hong Kong Research Grants Council (RGC No. HKU 7310/00M).

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