Effect of P. betle on Cell-Surface Hydrophobicity of Oral Candida.

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Introduction: Cell surface hydrophobicity (CSH) is a key attribute of virulence among infectious microorganisms. An understanding of this property would help to explain the high colonization affinity of Candida on denture materials.

Objectives: To determine the CSH of seven strains of oral Candida. The influence of aqueous P. betle extract on the CSH was also assessed.

Methods: Seven Candida ATCC strains were used and hexadecane represented the hydrophobic compound in the experiment. Following rigorous agitation of each candidal suspension with hexadecane, the optical absorbance (OD) of each suspension was read. The percentage of adsorption of each strain to hexadecane was then calculated. To assess the effect of P. betle extract on the CSH, the Candida cells were first treated with extract at concentrations of 0-15mg/ml. 0.12% w/v chlorhexidine (CHX)-containing mouthrinse and sterile distilled water were used as positive and negative controls, respectively.

Results: C. krusei, C. parapsilosis and C. tropicalis showed the highest adsorption capacity to hexadecane of about 40% while the others were within the range of 12-17%. The CSH of all Candida strains were significantly reduced following treatment with the extract (p<0.05). At 2mg/ml, P. betle was able to reduce the CSH of C. lusitaniae (94.09%), C. parapsilosis (91.60%), C. albicans (78.16%), C. dubliniensis (73.33%), C. krusei (35.66%), C. tropicalis (32.36%) and C. glabrata (24.43%).

Conclusion: The aqueous extract of P. betle tended to reduce the hydrophobic cell-surfaces of all the Candida tested. This suggested its potential to be used in candidal control especially for denture wearers.

Calcium Signal Facilitates the Immediate Induction of HIF-1α by Lipopolysaccharide.

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Objectives: hypoxia-inducible factor 1 alpha (HIF-1α) is an oxygen-sensitive nuclear transcription factor, which responds to hypoxia or non-hypoxia stimuli by transcribing dozens of downstream genes and participates profoundly in cellular physiology. The regulation of HIF-1α is typically through oxygen-sensitive prolyl hydroxylases (PHDs), which hydroxylize HIF-1α to facilitate its degradation. Other mechanisms include the regulation of the transcription and translation of HIF-1α. We previously reported that lipopolysaccharide (LPS) induces HIF-1α in human primary gingival fibroblasts (HGF). The present study aimed to explore the underlying mechanism via looking into Ca2+/Calmodulin-dependent protein kinase II (CaMKII) and calcineurin pathway.

Methods: Human gingival biopsies were harvested from teeth scheduled for extraction because of orthodontic reasons. HGFs were cultured and subjected to LPS treatment only or in combination with actinomycin D (transcription inhibitor), dimethylamilglycine (DMOG, PHDs inhibitor), KN-93 (CaMKII inhibitor) or cyclosporin A (calcineurin inhibitor). Realtime RT-PCR, immunoprecipitation and western blotting were performed to detect the transcript and peptide of HIF-1α.

Results: Although HIF-1α accumulation could be detected as early as 3 hours after LPS challenge, the transcription level of HIF-1α gene did not increase until after 6 hours. Moreover, when transcription was prohibited by actinomycin D, LPS still induced HIF-1α. Similarly, LPS enhanced the HIF-1α accumulation in DMOG-treated cells, indicating that the LPS effect on HIF-1α may not be through actions of PHDs. However, cyclosporin A and KN-93 dramatically attenuated the LPS-induced HIF-1α accumulation.

Conclusions: Ca2+/CaMKII-calcineurin pathway may be essential to the immediate accumulation of HIF-1α in HGFs as a response to LPS challenge.