Research Article

Oral Dysbiosis Exacerbates *Candida parapsilosis* Sensu Stricto Biofilm Production via Up-Regulation of the CPH2 Biofilm Master Gene

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**ABSTRACT**

**Introduction:** *Candida parapsilosis* sensu stricto is the second to third most frequent cause of candidemia. Studies place this yeast as a frequent colonizer of niches of the oral cavity, predominantly in pathological conditions. We hypothesize that a buccal environment in dysbiosis enhances the virulence of *C. parapsilosis* sensu stricto.

**Objective:** To evaluate the phenotype and molecular level of the production of biofilm in oral isolates of *Candida parapsilosis* sensu stricto and correlate the results with the clinical origin (dysbiosis versus eubiosis).

**Materials and Methods:** The biofilm-forming ability was compared in 50 oral isolates of *Candida parapsilosis* sensu stricto obtained from patients with and without oral dysbiosis; by quantification of metabolic activity. The results were corroborated by confocal fluorescence microscopy, and correlated with the transcriptional activity of *CPH2*, by RT-qPCR. The data were analysed by Excel 2010, and InfoStat 2018, with a 95% confidence interval.

**Results:** The metabolic activity in biofilm was significantly higher in oral dysbiosis relative to control (p = 0.0025). Basal expression of *CPH2* increased 2.8 times more in oral dysbiosis related to the control condition and showed no significant differences with pathogenic isolates of this same yeast, derived from onychomycosis lesions.

**Conclusion:** The oral cavity in dysbiosis increases the virulence of *C. parapsilosis* sensu stricto due to possible changes in epigenetic marks. This finding suggests that the oral cavity in dysbiosis may be an alternative route to the skin in the epidemiology of nosocomial candidemia.

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