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POST ANTIFUNGAL EFFECT, PHOSPHOLIPASE AND ASPARTYL PROTEINASE PRODUCTION OF *CANDIDA ALBICANS* ORAL ISOLATES FOLLOWING EXPOSURE TO CHLORHEXIDINE

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An important virulent factor of *Candida albicans*, the major aetiological agent of oral candidosis is its ability to produce extracellular enzymes such as phospholipases and aspartyl proteinases. Post-antifungal effect (PAFE) is the suppression of fungal growth following brief exposure to antifungal agents. Chlorhexidine gluconate (CG) is an antimicrobial mouth wash with antifungal properties used in dentistry. Its concentration in the mouth reaches sub-therapeutic levels during dosage intervals due to the diluent effect of saliva and the cleansing effect of the oral musculature. Therefore, *Candida* undergo only a brief exposure to CG during treatment, similar to that of the PAFE phenomenon. There is no information on the impact of PAFE on the phospholipase and aspartyl proteinase production of oral *C. albicans* isolates following brief exposure to sub-therapeutic concentrations of CG. Hence, the objective was to determine the PAFE, phospholipase and aspartyl proteinase production of oral *C. albicans* isolates following brief exposure to sub-therapeutic concentrations of CG.

Fifty oral *C. albicans* isolates (10 isolates each from smokers, diabetics, asthmatics using steroid inhalers, partial denture wearers and healthy individuals) were exposed to three sub-therapeutic concentrations of CG (i.e. 0.005%, 0.0025% and 0.00125%) for one hour. Thereafter the antiseptic was removed by dilution. Following drug removal, the PAFE, phospholipase and aspartyl proteinase production was determined by previously described turbidometric method, plate assay using an egg yolk-agar medium and solid medium containing bovine serum albumin respectively.

Mean *in vitro* PAFE ± SEM (h) of *C. albicans* following 1 h exposure to 0.005%, 0.0025% and 0.00125% CG was 6.97±0.77, 1.85±0.58 and 0.62±0.01, respectively. Compared with the unexposed controls, phospholipase production of these isolates were significantly suppressed with a mean percentage reduction (± SEM) of 21.68± 1.11%, 18.20± 1.08% and 14.04± 0.52% following exposure to 0.005%, 0.0025% and 0.00125% CG, respectively. Similarly, aspartyl proteinase production of these isolates were also significantly suppressed with a mean percentage reduction (± SEM) of 27.31± 2.32%, 21.53± 2.22% and 17.91± 2.67% following exposure to 0.005%, 0.0025% and 0.00125% CG, respectively. Brief exposure of *C. albicans* isolates to CG, a scenario all too familiar in the oral environment, would continue to wield an antifungal effect by suppressing growth as well as pathogenic extracellular enzyme production, thereby quelling its pathogenicity and exemplifies additional pharmacodynamics of CG.