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"An Evaluation of Oral Colonisation of Candida in Cigarette Smoking and Tobacco Toxicity"

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Abstract – The aim of the present study was Smoking and Tobacco chewing is associated with a variety of effects on the saliva, oral commensal bacteria and fungi, mainly Candida, which causes Oral Candidiasis, the most common opportunistic fungal infection in man. This evaluation is an attempt to address the effect of cigarette smoking and tobacco chewing on the colonisation of oral candida.

Keywords: Evaluation, Oral Colonization, Candida, Cigarette, Smoking, Tobacco, Toxicity, etc.

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INTRODUCTION

Epidemiological studies on Oral Candidiasis in HIV positive persons have identified Cigarette smoking as a major risk factor for symptomatic infection in those with higher CD4 cell counts than those usually predisposing to infection, with possible immunological consequences (Scully, *et al.,* 1994). Evidence of this possibility comes from the observation that such persons exhibited decreased interferon-gamma (IFN-γ) concentrations and a trend towards increased interleukin-4 (IL-4) concentrations in whole saliva as compared to the non-smokers. Although the association of oral candidiasis with smoking is unclear, hypothesis have included increased fungal burden caused by Candida, reduced numbers of Langerhans cells and/or increased prevalence of human papillomavirus in smokers (Soysa, Ellepola, 2005). This evaluation will discuss the relationship between oral candida colonisation and tobacco chewing/cigarette smoking and the possible mechanisms of pathogenicity (Bastiaan, Reade, 1982).

REVIEW OF LITERATURE:

One of the most commonly encountered opportunistic microorganisms in humans is Candida albicans, a ubiquitous fungus that is a part of the normal microbial flora found on mucosal surfaces such as those of the oral cavity, gastrointestinal tract, and vagina in human beings. It was stated that the tobacco content provided a nutritious medium to enhance proliferation of Candida species (Terai, Shimahara, 2005). Consumption of tobacco products remains to be an important problem of public health (Banoczy, *et al.,* 2001).

1. Candida strain: The cultures were then grown to the stationary phase in a shaking water bath for 18 h at 30°C, after which time the yeast cells were collected, washed with phosphate buffered saline (PBS), counted by means of a haemocytometer, and adjusted to 106 /ml prior to use.

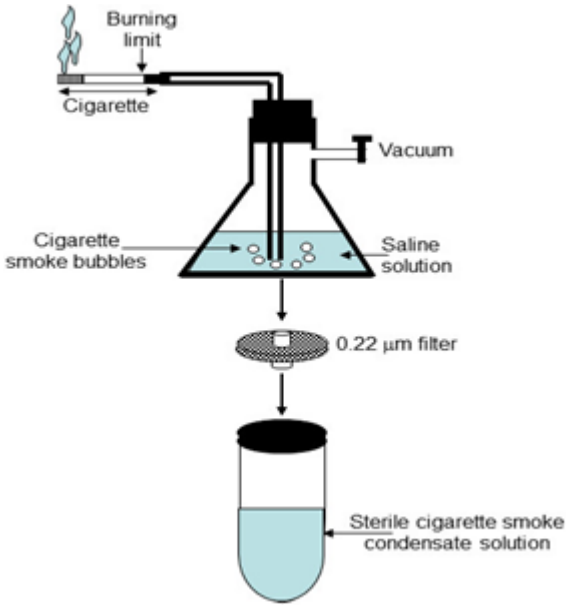

Figure 1 Generation of sterile CSC. Smoke from a burning cigarette was passed through a 0.9% saline solution.

2. Cigarette smoke condensate promoted C albicansbiofilm formation: Because CSC contributed to increasing Candida albicans adhesion and growth, we tested its potential to promote Candida albicans biofilm formation. Using SEM

analyses and a crystal violet assay, we were able to demonstrate the stimulatory effect of CSC on biofilm formation. SEM analyses revealed a high C. albicans density in the CSC-treated culture. A high Candida albicans density was observed in the scaffold in the presence of 30% CSC and this density increased with 50% CSC.

3. Cigarette smoke condensate promoted C albicans adhesion and growth: C. albicans attachment to the surface of glass slides for 1, 3, and 6 h was measured by means of crystal violet staining. C. albicans adhesion was significant ($p < 0.05$) at 3 and 6 h of incubation. All of the tested CSC concentrations promoted the adhesion of C. albicans. Adhesion was related to incubation period, with low adhesion reported at 1 h and high adhesion at 6 h. This result indicates that CSC can increase C. albicans adhesion and that this effect can lead to significant C. albicans growth.

4. Cigarette smoke condensate modulated HWP1, EAP1, and SAP2 expression: Based on the data showing that CSC increased C. albicans adhesion, growth, and biofilm formation, we sought to determine whether this took place through the regulation of certain genes. Reveals that HWP1 gene expression significantly increased following exposure of C. albicans to CSC The activation of this gene significantly ($p < 0.001$) increased according to CSC concentration. a twofold increase in HWP1 gene expression was recorded with a concentration of 30% CSC, compared to that observed in the controls, and with 50% CSC, this increase was over threefold. Similarly, EAP1 gene, which encodes a glycosylphosphatidylinositol-anchored, glucancross-linked cell wall protein involved in adhesion and biofilm formation, was also affected by CSC treatment.

CONCLUSION:

Smoking and Tobacco chewing is associated with Many factors can predispose to oral Candida infection. Whether cigarette smoking/tobacco chewing is included among one, has been considered for many years. The major component among both is Nicotine, an addictive stimulant drug. Smoking is evidently associated with systemic diseases like cardiovascular diseases, various lung disorders and different types of cancer. It has been proved that smoking is hazardous especially to women and children. Cigarette smoking negatively influences oral cavity.

REFERENCES:

[1]. Scully C, El-Kabir M, Samaranayake LP, 1994. Candida and oral candidosis: A review. Crit Rev Oral Biol Med 5: pp. 125-157.

[2]. Soysa N, Ellepola A, 2005. The impact of cigarette/tobacco smoking on oral candidosis: an overview. Oral Diseases 11: pp. 268–273.

[3]. Bastiaan RJ, Reade PC, 1982. The prevalence of Candida albicans in the mouths of tobacco smokers with and without oral mucous membrane keratoses. 53: pp. 148-151.

[4] Terai H, Shimahara M, 2005. Atrophic tongue associated with Candida. J Oral Pathol Med ; 34: pp. 397-400.

[5] Banoczy J, Gintner Z, Dombi C. 2001. Tobacco use and oral leukoplakia. J Dent Educ ; 65: pp. 322-7.