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Research Article

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Candida Albicans: Initiator or facilitator in Oral Carcinogenesis? A Short Review

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Review

In healthy individuals, the *Candida albicans* is a habitant of mucous membranes, consisting of oral cavity, nose, gastrointestinal tract, reproductive organs, etc. Bacteria, viruses, and fungi not only colonize different parts of the oral cavity but also connect and communicate with each other creating an oral ecosystem. These organisms are aerobic and anaerobic with a wide range of metabolic capabilities, such as capability to rebuild sugars and proteins and complex substrates found in the oral cavity thus affecting their host in many ways [1].

Candida albicans is abundantly found in the oral cavity and can cause a wide range of acute and chronic infections when the immune system is compromised. All components of the immune system play a vital role towards recognition and defense against *Candida albicans*. Innate immunity is accountable for detecting yeast cells, and cell-mediated immunity is involved in protecting the mucous membrane by its ability to produce cytokines. However, *Candida albicans* can replicate and invade host mucosal tissues because of dysfunction or disruption in oral epithelial barrier. *Candida albicans* produces hydrolytic enzymes and different virulence factors which helps invasion of oral epithelial tissues via hypha formation. Therefore, proliferation, protein degrading ability and infiltration of *Candida albicans* encourages a hyperplastic epithelial response and if it remains untreated for prolonged period, then epithelial lesions may become dysplastic and transform into carcinoma [2].

Oral Squamous Cell Carcinoma (OSCC) is an ulcer proliferative lesion affecting oral mucosa, with a 5-year survival rate of only 50%. Any site in the oral cavity can be affected starting from the lips to oropharynx. Etiology of OSCC is multifactorial including tobacco consumption, alcohol, betel nut chewing, nutritional deficiencies, and viral infections. Oral fungal colonization on neoplastic epithelial cells is considerably higher when compared to healthy surfaces which shows a positive co-relation between OSCC and *Candida albicans* can convert nitrates or nitrites into nitrosamine to produce acetaldehyde. Acetaldehyde is highly toxic with mutagenic and carcinogenic properties. This production of acetaldehyde is facilitated by production of an enzyme by *Candida albicans* called *Candida Albicans* Alcohol Dehydrogenase 1 (CaADH1), which is also associated with its pathogenicity. It interferes with DNA synthesis and repair by changing its structure and function which leads to genomic instability and inhibition of apoptotic system [3]. Furthermore, CaADH1 gene expression is higher in patients with OSCC as compared to healthy people which indicates a positive association between the two.

Moreover, in elderly patients continuing oral candidiasis has been observed leading to the development of OSCC as untreated candidiasis leads to dysplastic changes and finally transforming into carcinoma. There is convincing evidence advocating the idea that Candida helps carcinogenic events in the oral cavity but the exact mechanism by which Candida leads to OSCC still needs to be investigated. This is so because cancer patients have altered immune status and immunosuppression facilitates Candida infection in cancer patients [4]. *Candida albicans* infection is significantly associated, especially in cases of dysplastic and neoplastic forms of oral leukoplakia involving the tongue. Candida may influence and play a role in dysplastic and malignant transformations of leukoplakia. Finally, *Candida albicans* is responsible for production of IL1β, which triggers the production of proinflammatory cytokines. Significant colonization of *Candida albicans* genotype A has been identified through molecular analysis data in oral squamous cell carcinoma lesions, suggesting that genotypic diversity may affect the carcinogenic process [5].

The potential tumor promoting role of Candida in oral cancer cells is supported by different in vitro studies indicating enhancement of the migration ability, expression of matrix metalloprotein-

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ases, secretion of oncometabolites, and expression of metastasis-related genes by *Candida albicans* [6]. However, in general, the role of *Candida albicans* in oral carcinogenesis is complex due to presence of several virulence factors, host immune response and an imbalance in the microbiota, changes in their functional composition and metabolic activities, or a shift in their local distribution. Whether it acts as an initiator, or a facilitator still needs to be investigated.

Conflict of Interest

None.

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