## **Epidemiology and Immunological Parameters of Oral Cancer** Mack Carter<sup>\*</sup>

Department of Oral Medicine and Surgery, UCLA School of Dentistry, Los Angeles, United States

**Received:** 12-Jan-2023, Manuscript No. OHDM-23-19744; **Editor assigned:** 16-Jan-2023, Pre QC No. OHDM-23-19744 (PQ); **Reviewed:** 06-Feb-2023, QC No. OHDM-23-19744; **Revised:** 16-Feb-2023, Manuscript No. OHDM-23-19744 (R); **Published:** 24-Feb-2023, DOI: 10.35248/2247-2452.23.22.1039

## Description

Oral carcinoma is a heterogeneous group of cancers that occurs in oral cavities includes lip, cheek, salivary gland, soft and hard palate, uvula, gums, tonsils, tongue, and inner tongue. Worldwide oral carcinoma occurs more than 2%-4% among all cancer in which 95% represented by Oral Squamous Cell Carcinoma (OSCC). The oral carcinoma has been ranked sixteenth globally, with an incidence rate of 354,864 (2%) and a mortality rate of 177,384 (1.9%). In the Indian subcontinent, the new cases of oral carcinoma 1,19,992 and more than 72,616 deaths annually. The most important anatomical bases in the oral carcinoma are considered the tongue (26.0%) followed by the lips (23.0%), the lower floor of the mouth (16.0%), and salivary glands with (11.0%). Oral carcinoma is usually preceded by oral pre-malignant lesions in patients in which the initial stage of cancer is characterized by white spots (leukoplakia), complete mouth openings (sub mucous fibrosis), rash inside the mouth (erythroplakia). Leukoplakia, thick, white patches in the mouth arise on the gums, inside the cheeks, under the mouth, and, sometimes, on the tongue. These patches cannot be scratched away and might not be clinically or pathologically characterized. Erythroplakia, any lesion of the oral mucosa, is presented as dark red velvet plaques. Although erythroplakia is much lower in incidence than leukoplakia, it carries a significantly higher risk in a condition with dysplasia or carcinoma. Eventually, it changes to invasive squamous cell carcinoma, which can be easily be detected by visual inspection or palpation. These red lesions and some white lesions have a possible for malignant alter and have been categorized as Oral Precancerous Lesions (OPL) that are closely linked with malignant transformation into oral cancer. Oral cancer is caused by concurrent changes in biochemical, cellular, and molecular alterations in conjunction with clinical developments affecting epithelial tissues. The World Health Organization (WHO) has categorized precancerous changes into different stages of mild, moderate and severe or invasive carcinoma in Introduction 2005, and the malignant transformation risk has 6.6% to 36.4% reported. However, only 25.0% of leukoplakia, as detected, involves precancerous changes that progress to cancer in 10 years if not treated appropriately. However, in the case of erythroplakia, 70.0% to 95.0% of these lesions become cancerous and progress to cancer. Oral Sub-Mucous Fibrosis (OSMF) is another chronic disease characterized by the presentation of fibrosis, increased loss of tissue mobility with lamina propria and sub mucosa accompanied by burning, and stimulation of the oral mucosa, oropharynx, and trismus. The most distinguishing feature is the formation of a vertical fibrous ridge within the cheekbone.

In the advanced stages, vertical fibrous bands appear in the cheeks, duct pillars, and surround the lips. Lichen planus is an autoimmune disorder of mouth membranes that usually affects middle aged persons; the most frequent site for Lichen planus is buccal mucosa, tongue, and gingiva, while palatal lesions are rare; with interlinking white streaks forming a reticular pattern similar to a spider web. The background membrane might have red, while some cases blisters and ulcers with the white lines. Oral lichen planus is one of the potentially malignant disorders seen in popular, reticular, plaque-like, atrophic, erosive, and bullous subtypes. Epidemiological literature revealed that tobacco smoking alcohol, consumption, diet, and Human Papilloma Virus (HPV) a 90% primary causative of oral cancer. In India, oral carcinoma has been causally linked with tobacco smoking and chewing alone or together with betel quid, and heavy alcohol intake is the main etiological factor. On the other hand, approximately 75% of oral carcinoma associated tobacco (smokeless) and gutka, the rate of risk depends upon the chewed amount and the duration of exposure. However, smokers are six times more frequent in developing cancer than non-smokers. The individuals who used filtered cigarettes and quit smoking are at a substantially lower risk of OC than the other forms of tobacco users and current smokers. Cigarette smoke contains more than four thousand chemical compounds, including approximately sixty known carcinogens. Smokers have a lifetime increased risk of oral cancer, which is 5 to 25 times higher, and twenty years after smoking cessation, the exsmokers risk for OSCC starts to exceed the risk in the general population. Many risk factors involved in OSCC rather than smoking; tobacco products, including chewing tobacco, gutka, and sucked (moist oral) tobacco. Smokeless tobacco contains specific nitrosamines, major culprits for oral cavity cancer. The smokeless tobacco is direct contact with buccal mucosa and inner other soft tissue of the lips having various risk sites for cancer development. Betel nut chewers who also smoke and drink are 195 times more likely to develop cancer of the oral than those who do not smoke. Khaini has been found to contain the highest level of tobacco-specific nitrosamines. The tobacco processing method, which favours the reduction of nitrate to nitrite and other nitro sating agents, appears to be responsible for the high nitrosamine levels. Correspondingly, nitrite level was highest among the smokeless such as khaini and zarda which is produced by boiling tobacco leaves in water with lime and spices until evaporation, and these nitrosamines have been reported in the saliva of tobacco chewers. All these products are commonly used in Asian countries and India.

Corresponding author: Mack Carter, Department of Oral Medicine and Surgery, UCLA School of Dentistry, Los Angeles, United States, Email: mac\_cart@gmail.com