

## A Review on Causes of Oral Cancer

Johnny Fujimoto\*

Department of Periodontics, Comprehensive Dental Care and University of Pennsylvania, USA

### Introduction

In many regions of the world, including Europe, oral cancer-defined as cancers of the lip, tongue, and mouth-is a serious and expanding issue. Together, oral and pharyngeal cancers rank as the sixth most prevalent malignancy in the world. South Asia, the Pacific regions, Latin America, and portions of central and Eastern Europe are the regions with a high incidence. Current details on the global epidemiology are available from a recent review.

In 2006, 5,325 additional cases were identified. Since the 1980s, there have been an annual increase in the number of oral cancer incidents reported to cancer registries; more current data indicate an increase of 41.2 percent over 10 years. The most recent findings were verified by the statistics released by Cancer Research in August 2009, which also showed that other malignancies linked to tobacco use, like lung cancer, had decreased. No other cancer location has actually experienced such a sharp increase in incidence over the last 25 years, according to these unrefined (age unadjusted) data [1].

In great part, oral cancer is a self-inflicted illness. It's crucial to comprehend the risk factors linked to the disease in order to develop preventive measures. The main risk variables are well recognised, have undergone a recent review<sup>4</sup>, and won't be covered in-depth in this essay. Dentists should be aware of several newly discovered mouth cancer risk factors, though, as well as some on-going debates over the origin of this illness.

### Description

This essay's goal is to offer a viewpoint on these hotly contested issues. There is insufficient evidence to link a number of factors that have been frequently mentioned as being likely to be associated with oral cancer, including heredity and familial risk, marijuana (cannabis) smoking, khat chewing, medicinal nicotine use, HIV infection, and alcohol-containing mouthwashes. In order for dentists to communicate just the significant and pertinent dangers with their patients, it is crucial to dispel several beliefs about the disease's cause [2].

### Three major risk factors: Alcohol, Tobacco and Betel nut

Cigarette smoking and excessive alcohol consumption is two major risk factors for oral cancer in the UK population. The related risk with these two lifestyle choices is supported by a number of important epidemiological researches from numerous nations. While there is no conclusive proof that particular alcoholic beverages (wine, beer, spirits) have differing effects on oral cancer, all forms of smoking (cigars or cigarettes) have the same additional risks. The alcoholic beverage with the highest risk in a particular demographic would be that population's most popular one [3].

Use of smokeless tobacco (ST) greatly raises the risk of mouth cancer. The public cannot purchase ST because it is illegal to do so in the UK. However, betel quid is frequently combined with the chewing tobacco that is sold (areca nut). Betel quid is a significant risk factor for those who have this habit among Asian ethnic minority living in the UK and is carcinogenic to humans (both with and without additional tobacco). Being South Asian (ethnicity, not race) may therefore be seen as a risk factor.

The estimated higher risks from these several agents and exposures (among smokers, regular drinkers, and betel quid users; adjusted for each other) are consistently reported from multiple populations compared to non-users. Multiple meta-analyses or systematic reviews have verified the raised risks, demonstrating the significant hazards of certain lifestyle choices for oral cavity malignancies. There is evidence linking increased alcohol use to an increase in oral cancer cases, especially among younger people. Alcohol use has a well-documented synergistic effect on tobacco's ability to cause mouth cancer [4].

Additionally, there is strong evidence that suggests quitting these behaviours lowers risk, albeit it may take as long as ten years to achieve the low-risk condition of never using. The primary responsibility of oral health experts is to encourage quit efforts among their patients through brief interventions, and when necessary, to send individuals for additional treatment of tobacco use to cessation clinics.

Recent research (mainly conducted in the USA) provides enough proof that HPV infection is a risk factor, especially for the oropharynx (posterior tongue, tonsil and the [visible] part of pharynx in continuity with the oral cavity). A portion of oral and Oropharyngeal cancers can be linked to HPV infection and seropositive status, according to new findings that support the aetiological significance of HPV in cervical cancer (particularly among young subjects with no tobacco or alcohol history) [5]. Some writers have hypothesised that sexual activities may play a role in how this virus infects the oral cavity (and integrates with the oral mucosa).

Cervix and Gardasil, two HPV vaccinations, are now readily available. When administered to young adolescent women, they are not therapeutic but are anticipated to provide protection against cervical cancer. Results are awaited to establish their efficacy in preventing infections in organs other than the cervix, such as the oral cavity or oropharynx, in young adult women who have previously been exposed to HPV [6].

### Conclusion

Dentists must be aware of the main risk factors that are relevant to the general community in order to prevent oral malignancies. Evidence for recognised and modifiable risk factors is presented in this article. It is crucial to assess reported conflicts since treatments should be supported by solid scientific evidence. In order to enable dentists to ignore characteristics for which the epidemiological evidence is either

**\*Corresponding author:** Johnny Fujimoto, Department of Periodontics, Comprehensive Dental Care and University of Pennsylvania, USA, E-mail: johnyfujimoto@yahoo.com

**Received:** 31-May-2022, Manuscript No: JOHH-22-67727, **Editor assigned:** 02-Jun-2022, PreQC No: JOHH-22-67727(PQ), **Reviewed:** 16-Jun-2022, QC No: JOHH-22-67727, **Revised:** 21-Jun-2022, Manuscript No: JOHH-22-67727(R), **Published:** 28-Jun-2022, DOI: 10.4172/2332-0702.1000321

**Citation:** Fujimoto J (2022) A Review on Causes of Oral Cancer. J Oral Hyg Health 10: 321.

**Copyright:** © 2022 Fujimoto J. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

insufficient or not suggestive, this study offers scientific information based on recent findings. It's critical to dispel some misconceptions about variables that are thought to be unrelated to this malignancy in order to avoid misinforming patients and the general public and to make it easier to develop prevention methods.

#### References

1. Warnakulasuriya S (2009) Global epidemiology of oral and oropharyngeal cancer. *Oral Oncol* 45: 309-316.
2. Warnakulasuriya S (2002) Areca nut use following migration and its consequences. *Addict Biol* 7: 127-132.
3. Thomas SJ, Bain CJ, Battistutta D, Ness AR, Paissat D, et al. (2007) Betel quid not containing tobacco and oral cancer: a report on a case-control study in Papua New Guinea and a meta-analysis of current evidence. *Int J Cancer* 120: 1318-1323.
4. Warnakulasuriya S, Sutherland G, Scully C (2005) Tobacco, oral cancer, and treatment of dependence. *Oral Oncol* 41: 244-260.
5. Scully C (2005) Oral cancer; the evidence for sexual transmission. *Br Dent J* 199: 203-207.
6. Petridou E, Zavras AI, Lefatzis D, Dessypris N, Laskaris, et al. (2002) The role of diet and specific micronutrients in the aetiology of oral carcinoma. *Cancer* 94: 2981-2988.