Journal of Molecular Histology & Medical Physiology

Mol Histol Med Physiol 2021, 6:3

Editorial Note Open Access

Human Oral Cancer: Molecular Biology

Andrew Perry*

Editorial office, JOMHMP

***Corresponding author:** Perry A, Chaussee de la Hulpe 181, Box 21, 1170 Watermael-Boitsfort, Brussels, Belgium biomolecules@molecularbiologyjournals.com

Received date: May 04, 2021; **Accepted date:** May 7, 2021; **Published date:** May 10, 2021

Every year, roughly 40,000 Americans and 350,000 people worldwide are diagnosed with oral cancer for the first time. In the general population, oral cancer is the sixth most frequent disease for both men and women, and the third most common cancer in developing countries. About half of those affected will die within five years of being diagnosed, while those who survive will likely have significant cosmetic and/or functional problems. Oral cancer is defined as cancer of the oral cavity and pharynx, which includes cancers of the lip, tongue, salivary glands, gum, floor, and other regions of the mouth, oropharynx, nasopharynx, hypopharynx, pharynx, and other buccal areas, according to the International Classification of Diseases. Carcinomas account for 96 percent of all oral malignancies, with squamous cell carcinomas accounting for 91 percent. Although total mortality has decreased over the last two decades, oral cancer has one of the lowest five-year relative cancer survival rates, considerably below those of many other cancers such as skin melanoma and cancers of the testis, breast, colon, rectum, and kidney. According to the National Cancer Institute's Surveillance Epidemiology and End Result Program, only 33% of black patients and 54% of white patients survive five years after being diagnosed with oral cancer. Oral cancer survival rates have remained stable over the last two decades and are still among the poorest of all cancer locations. It is now evident that we need to enhance our understanding of the origin and progression of oral cancer before we can make any further progress. Tobacco and alcohol continue to be the most major risk factors for oral squamous cell carcinomas. Oral carcinogenesis has long been related to viruses, most notably the human papillomavirus. Due to the fact that the majority of people exposed to these risk factors do not acquire mouth cancer, as well as the fact that rare occurrences of oral cancers occur in young adults and non-users of tobacco and alcohol, a hereditary predisposition has been postulated. Many genetic events generated by these risk factors' chromosomal changes have recently been postulated to underpin the histopathologic development of oral squamous cell carcinoma.

As a result, it's fair to believe that earlier identification and/or treatment contributed to a lower death rate compared to incidence. Both the mortality and incidence data, like with other types of cancer, show greater rates among males than women and blacks than whites. With the application of multi-factorial techniques such as molecular biology, virology, epidemiology, and clinical trials based on biological principles, the molecular foundation of human cancer is presently emerging at a rapid speed. This, as well as the molecular virology of human oral cancer, has been previously discussed. The present state of molecular biology in human oral cancer is the subject of this review.

GROWTH FACTORS

Oral keratinocyte proliferation can be aided by growth agents. Growth factors are de-regulated during oral carcinogenesis due to increased synthesis and autocrine activation. TGF-a is overexpressed by hyperplastic epithelium early in oral carcinogenesis and later by the inflammatory infiltrate surrounding the invading oral epithelium, notably eosinophils. Healy observed an increase in eosinophils in oral cancer as early as 1975, accounting for up to 17% of the total inflammatory infiltration, and this is a constant characteristic in all cases of mouth cancer studied.