Short Communication

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Archives in Cancer Research
ISSN 2254-6081

2018
Vol.6 No.4:18

DOI: 10.21767/2254-6081.100184

Head and Neck Carcinogenesis

Ajaz S*

Dr. Panjwani Center for Molecular Medicine and Drug Research (PCMD), International Center for Chemical and Biological Sciences (ICCBS), University of Karachi, Pakistan

*Corresponding author: Ajaz S

sadiaajaz.pcmd@iccs.edu

Dr. Panjwani Center for Molecular Medicine and Drug Research (PCMD), International Center for Chemical and Biological Sciences (ICCBS), University of Karachi, Pakistan.

Tel: +922134824924-5

Abstract

Cancer is caused by mutation(s) in driver gene(s), leading to the transformation of normal cell into malignant one, followed by rapid proliferation. Head and neck cancers (HNCs) comprise epithelial malignancies of the mucosal lining in upper aero-digestive tract. These cancers are heterogeneous in nature. Carcinogenesis therein is correlated with the use of tobacco, chewing of areca nut (betel quid), alcohol consumption and infection with the human papillomavirus (HPV). These factors in conjunction with molecular variations contribute to tumour development, pathology and response to treatment. Thus, the carcinogenesis of head and neck cancers comprise a complex system of gene-gene and gene-environment interactions.

Keywords: Species; Tobacco; Carcinogenesis; Neck cancers; Papillomavirus

Received: November 02, 2018; Accepted: November 12, 2018; Published: November 15, 2018

Citation: Ajaz S (2018) Head and Neck Carcinogenesis. Arch Can Res Vol.6 No.4:18.

Introduction

Cancer is caused by mutation(s) in driver gene(s), leading to the transformation of normal cell into malignant one, followed by rapid proliferation [1]. Head and neck cancers (HNCs) comprise epithelial malignancies of the mucosal lining in upper aero-digestive tract [2]. These cancers are heterogeneous in nature [3]. Carcinogenesis therein is correlated with the use of tobacco, chewing of areca nut (betel quid), alcohol consumption and infection with the human papillomavirus (HPV) [4]. These factors in conjunction with molecular variations contribute to tumour development, pathology and response to treatment [5]. Thus, the carcinogenesis of head and neck cancers comprise a complex system of gene-gene and gene-environment interactions. The risk factors for head and neck cancers are elucidated in **Figure 1.**

Table 1 Phase I and II metabolism enzymes participating in response to carcinogens.

Sr. No.	Phase (Category)	Enzymes
1.	I (microsomal)	Cytochrome b_5/cytochrome b_5 reductase
2.		Cytochrome P450
3.		CYP3A4
4.		Flavin-containing mono-oxygenises (FMOs)
5.		Cyclooxygenases
6.	I (non-microsomal)	Alcohol dehydrogenase
7.		Aldehyde dehydrogenase
8.		Amine oxidases
9.		Monoamine oxidase
10.		Diamine oxidase
11.	I (reductases)	Carbonyl reductases
12.		Sulphonyl reductases
13.	I (hydrolases)	Esterase/amidase enzymes
14.		Epoxide hydrolases
15.	II (metabolism)	Glucuronoxylan transferases
16.		Glucosyltransferases
17.		Sulfotransferases
18.		Methyltransferases
19.		N-Methylases
20.		O-Methylases
21.		S-Methylases
23.		Glutathione S-transferases
25.		Cysteine conjugate β-lyase
27.		Acetylases
28.		N-acetyltransferase
29.		N, O-acyltransferases

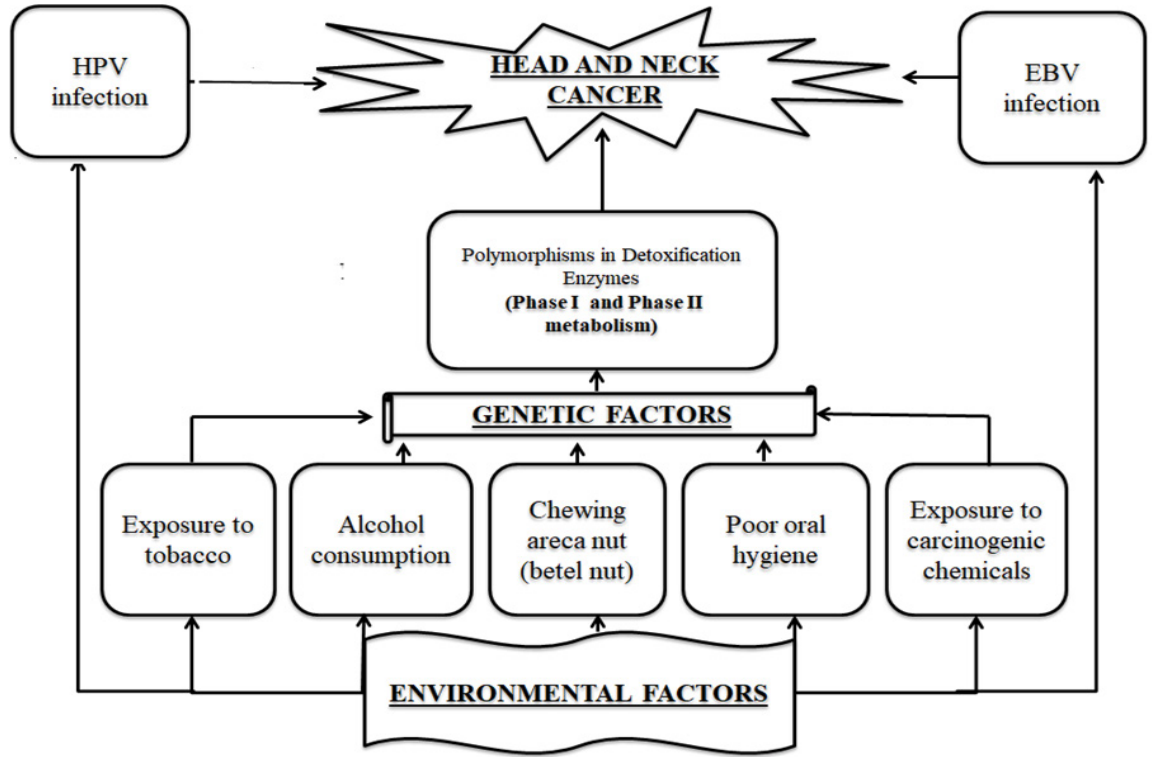

Figure 1 Environmental and genetic risk factors in head and neck cancers.

Discussion and Conclusion

Functional variations in genes encoding detoxification enzymes are candidate markers for these cancers. These enzymes catalyse bio-conversion of exogenous and endogenous carcinogens into hydrophilic, non-toxic metabolites [6]. Biotransformation decreases lipophilicity and consists of two phases. In phase I, a polar group is introduced into the molecule.

Thus, the carcinogen is converted into:

- Water-soluble form that is eliminated or,
- More electrophilic compound, which is recognized by phase II enzymes.

In phase II enzymes, the polar group serves as a center for conjugation with an endogenous metabolite. The phase I and phase II enzymes are listed in **Table 1** [7].

The exogenous risk factors for head and neck cancers, including betel quid ingredients, nitrosamines, tobacco, alcohol, and other environmental toxins, are metabolized by phase I and phase II enzymes. On the other hand, endogenous risk factor like reactive oxygen species (ROS) are metabolized by phase II enzymes. In case of genetic alterations, ranging from single nucleotide polymorphisms (SNPs) to deletion of whole gene, there is either decreased enzymatic activity or no activity. Consequently, the carriers of these genetic variations are susceptible to head and neck cancers.

Increasingly, human papilloma virus (HPV) infection is associated with head and neck cancers. The vaccination against this virus has also significantly decreased the number of cases with this infection. Two known viral oncogenes, E6 and E7, play major role in carcinogenesis. E6 encodes protein that inhibits p53, whereas E7 protein inhibits pRb pathway [8,9]. Among head and neck cancers, persistent infection with Epstein-Barr Virus (EBV) is one of the risk factors for nasopharyngeal cancers. However, EBV infection alone is insufficient causative factor as majority of the population is EBV +ve without developing this form of cancer [10].

Epi-genetic changes in p53 and p16INK4A–cyclin D1–RB are mainly implicated in head and neck cancers and comprise earliest steps in neoplastic transformation [11]. Additional epigenetic changes are currently being elucidated [12].

With the increase in incidence of head and neck cancers and their high-mortality rate, the understanding of epidemiological and biological phenomenon underlying their carcinogenesis has become urgent.

Acknowledgement

Ms. Rabbia Muneer for her contribution in preparation of **Figure 1**.

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