

Role of Environmental Factors in the Evolution of Cancer

Nemat Khansari

Medical Sciences University of Tehran, School of Medicine, Department of Immunology, Tehran, Iran

***Correspondence to:** Dr. Nemat Khansari, Medical Sciences University of Tehran, School of Medicine, Department of Immunology, Tehran, Iran.

Copyright

© 2018 Dr. Nemat Khansari. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Received: 08 November 2018

Published: 13 November 2018

Keywords: *Cancer; Chronic Infection*

Cancer is the second leading cause of death worldwide, accounting for an estimated 9.6 million deaths in 2018. This type of diseases elicits from uncontrolled growth and proliferation of malignant cells harbouring genetic alterations. These abnormally growing and proliferating cells can have a life-threatening effect when they physically or pathologically affect adjacent healthy cells in a vital organ. Distinct genetic alterations within a cell that result in out of control cell proliferation are responsible for the initiation of cancer formation. In this regard, genetic alterations in proto-oncogenes and tumour suppressor genes are frequently reported in several cancer cell types. Prolonged exposure to various mutagens can be involved in the induction of these genetic alterations in cancerous cells.

Chronic infection represents a risk factor for cancer development. It has been estimated that up to 20% of the global cancer burden is attributed to infectious agents, especially viruses and bacteria. The bacterium *Helicobacter pylori* and viruses Hepatitis B virus, Hepatitis C virus, certain strains of human papillomavirus, Epstein-Barr virus, human immunodeficiency virus type-1, and human T-cell lymphotropic virus type-1 have been identified as major carcinogenic infectious agents by International Agency for Research on Cancer (IARC). These infectious agents are highly prevalent in the world. Nevertheless, most infected individuals do not develop cancer, indicating that genetic susceptibility of host and environmental factors may be associated with cancer caused by these infectious agents.

Gastrointestinal tract is constantly exposed to many bacterial agents and some of these agents induce chronic inflammation in this organ. On the other hand, chronic inflammation may increase the rate of mutation in

epithelial cells leading to cancerous cell formation. As discussed below for gastric cancer, some evidences suggest that specific bacteria can be involved in cancer development or progression. These bacteria can trigger oxidative stress in host cells, activate some intracellular pathways such as nuclear factor-kappa B (NF- κ B) pathway, and promote production of various components involved in carcinogenesis.

In addition to the role of microbial factors causing inflammatory responses leading to cancers, epigenetics involvement due to environmental factors contribute to increasing incidence of cancers too. Epigenetic is heritable phenotype changes that do involve alterations in gene expression without any changes in DNA sequence leading altered expression or translation of the genome. DNA methylation, histone modification and RNA interference are main epigenetic factors contribute to cancer evolution. What factors influences epigenetic patterns? It has been shown that life style which include many factors such as nutrition, behavior, stress, physical activities, smoking, alcohol consumption, working habits and environmental pollutions are major factors that might modify epigenetic patters.

It should be noted that epigenetic factors may be cause tumors developments but the concept of inflammatory diseases contribution to cancer is a critical component of tumor progression.