



## The Causes and the Pathophysiology of Cancer

Tianwei Guo\*

Department of Pathophysiology, Harbin Medical University, Harbin, China

### ARTICLE HISTORY

Received: 30-Jan-2023, Manuscript No. JMOLPAT-23-90214;  
Editor assigned: 02-Feb-2023, PreQC No. JMOLPAT-23-90214(PQ); Reviewed: 17-Feb-2023, QC No JMOLPAT-23-90214;  
Revised: 24-Feb-2023, Manuscript No. JMOLPAT-23-90214(R);  
Published: 03-Mar-2023

### Description

Cancer is a group of diseases that can invade or spread to various parts of the body as a result of abnormal cell proliferation. They contrast with benign tumors, which stay always. Some warning signs and symptoms include a lump, irregular bleeding, a chronic cough, unexplained weight loss, and a change in bowel habits. There may be some cancer symptoms, but there may also be other causes. More than 100 different cancers can affect humans.

Tobacco use is responsible to 22% of cancer-related deaths. An additional 10% of instances are caused by obesity, a poor diet, inactivity, or excessive alcohol use. Ionizing radiation exposure, certain illnesses, and environmental toxins are additional issues. 15% of cancer cases in underdeveloped nations are caused by infections, such as *Helicobacter pylori*, hepatitis B, hepatitis C, human papillomavirus infection, Epstein-Barr virus, and human immunodeficiency virus (HIV).

### Pathophysiology

Cancer is essentially a disorder of the control of tissue growth. Changes in the genes that control cell development and differentiation are required for a normal cell to develop into a cancer cell. Two main categories can be used to classify the impacted genes. Genes called oncogenes encourage cell division and development [1]. Tumor suppressor genes prevent cell proliferation and survival. Novel oncogenes can develop, normal oncogenes can be overexpressed inappropriately, tumor suppressor genes can be silenced or under expressed, or tumor suppressor genes can be overexpressed inappropriately. To turn a healthy cell into a cancer cell, usually several different genes must be altered [2].

Different degrees and processes can lead to genetic

alterations. Errors in mitosis can result in the acquisition or loss of a whole chromosome. The nucleotide sequence of genomic DNA can alter, and these mutations are more frequent [3]. A significant section of a chromosome may be lost or gained in large-scale changes. When a cell acquires additional copies of a short chromosomal region, typically containing one or more oncogenes and nearby genetic material, genomic amplification takes place. When two distinct chromosomal regions merge incorrectly, frequently at a specific site, translocation occurs [4, 5].

Point mutations, deletions, and insertions are examples of small-scale changes. They can affect a gene's expression in the promoter region or change the function or stability of the protein that is produced by the gene by occurring in the gene's coding sequence [6, 7]. The production of viral oncogenes in the afflicted cell and its progeny might come from the disruption of a single gene caused by the incorporation of genomic material from a DNA virus or retrovirus. Probabilistically, there will be some inaccuracies in the data replicated from living cells' DNA. Comprehensive mistake prevention and repair are incorporated into the procedure to protect the cell from cancer. A damaged cell may self-destruct through a process known as apoptosis if a serious mistake occurs. In the event that the error correction procedures are unsuccessful, the mutations will endure and be transmitted to daughter cells [8].

According to the traditional understanding of cancer, it is a group of illnesses brought on by chromosomal abnormalities, tumor-suppressor gene alterations, and oncogene mutations. Functionally significant genome modifications are epigenetic changes, which do not alter the nucleotide sequence. A few of these alterations are DNA methylation, chromosomal rearrangements, and histone modifications. Without al-

tering the base DNA sequence, each of these alterations regulates gene expression [9]. These changes could resemble mutations in that they could endure through multiple generations of cell divisions.

## References

- [1] Gavila J, Seguí MÁ, Calvo L, López T, Alonso JJ, Farto M, et al. Evaluation and management of chemotherapy-induced cardiotoxicity in breast cancer: a Delphi study. *Clin Transl Oncol* 2017; 19:91-104.
- [2] Procter M, Suter TM, De Azambuja E, Dafni U, Van Dooren V, Muehlbauer S, et al. Longer-term assessment of trastuzumab-related cardiac adverse events in the Herceptin Adjuvant (HERA) trial. *J Clin Oncol* 2010; 28(21):3422-3428.
- [3] Santoro C, Esposito R, Lembo M, Sorrentino R, De Santo I, Luciano F, et al. Strain-oriented strategy for guiding cardioprotection initiation of breast cancer patients experiencing cardiac dysfunction. *Eur Heart J Cardiovasc Imaging* 2019; 20(12):1345-1352.
- [4] Lyon AR, López-Fernández T, Couch LS, Asteggiano R, Aznar MC, Bergler-Klein J, et al. 2022 ESC Guidelines on cardio-oncology developed in collaboration with the European Hematology Association (EHA), the European Society for Therapeutic Radiology and Oncology (ESTRO) and the International Cardio-Oncology Society (IC-OS) Developed by the task force on cardio-oncology of the European Society of Cardiology (ESC). *Eur Heart J* 2022; 43(41):4229-4361.
- [5] Smiseth OA, Torp H, Opdahl A, Haugaa KH, Urheim S. Myocardial strain imaging: how useful is it in clinical decision making?. *Eur Heart J* 2016; 37(15):1196-1207.
- [6] Moslehi JJ, Witteles RM. Global longitudinal strain in cardio-oncology. *J Am Coll Cardiol* 2021; 77(4):402-404.
- [7] Boe E, Skulstad H, Smiseth OA. Myocardial work by echocardiography: a novel method ready for clinical testing. *Eur Heart J Cardiovasc Imaging* 2019; 20(1):18-20.
- [8] Guan J, Bao W, Xu Y, Yang W, Li M, Xu M, et al. Assessment of myocardial work in cancer therapy-related cardiac dysfunction and analysis of CTRCD prediction by echocardiography. *Front Pharmacol* 2021; 12:770580.
- [9] Sahiti F, Morbach C, Cejka V, Tiffe T, Wagner M, Eichner FA, et al. Impact of cardiovascular risk factors on myocardial work—insights from the STAAB cohort study. *J Hum Hypertens* 2022; 36(3):235-245.