

Would carcinogenesis have a synergistic relationship with bacteria and virus?

Eduardo Emery

Sociedade Brasileira de Patologia Clínica (SBPC), Rio de Janeiro, RJ, Brazil.

According to data from the World Health Organization (WHO)⁽¹⁾, updated in September 2018, cancer is the second leading cause of death worldwide, totaling 9.6 million. The same survey mentions that, globally, one in six deaths is due to cancer and 70% of them occur in low- and middle-income countries. Another significant finding is that 22% of these deaths in these countries are associated with infectious conditions⁽²⁾.

The WHO, in 2017, approved a resolution in the UN General Assembly containing actions^(3,4) for cancer prevention and control, in which, in partnership with the International Agency for Research on Cancer (IARC), recommends the need for research on the causes of the disease and the mechanisms of carcinogenesis, that is, it highlights the significant importance of the studies related to the attempt to clarify its causal processes.

It is known that cancer is a pathology in which normal cells become abnormal, with high rate of multiplication, invading other areas and organs of the body, circumventing all the processes of control of multiplication and destruction of cells, especially the immunological ones, so very similar to bacterial and viral infectious processes, as shown in the literature⁽⁵⁻¹⁰⁾.

Considering, therefore, the importance of cancer as the second leading cause of mortality worldwide, as well as the imperious need for further studies and the fact that its pathophysiology in several immunological aspects present similarity with the survival of bacteria and viruses in our body. In this issue, the *Jornal Brasileiro de Patologia e Medicina Laboratorial* (JBPML) publishes an excellent article by Pêgas (2018)⁽¹¹⁾, in which the author points out several similar mechanisms of survival between neoplastic cells, bacteria and viruses, in which they circumvent the immune system, even proposing a different perspective at some forms of cancer, in whose etiopathogenesis may present a possible synergy with these microorganisms.

Enjoy reading.

REFERENCES

1. Organização Panamericana de Saúde [Internet]. Folha Informativa – Câncer. [Updated on: Sep 2018]. Available at: https://www.paho.org/bra.../index.php?option=com_content&view=article&id=5588:folha-informativa-cancer&Itemid=839.
2. Plummer M, de Martel C, Vignat J, Ferlay J, Bray F, Franceschi S. Global burden of cancers attributable to infections in 2012: a synthetic analysis. *Lancet Glob Health*. 2016 Sep; 4(9): e609-16. doi: 10.1016/S2214-109X(16)30143-7.
3. World Health Organization. Seventieth world health assembly. Geneva, 22-31 May 2017. Available at: http://apps.who.int/gb/ebwha/pdf_files/WHA70-REC1/A70_2017_REC1-en.pdf?ua=1&ua=1#page=27.
4. World Health Organization [Internet]. Global action plan for the prevention and control of NCDs 2013-2020. Available at: <https://www.who.int/nmh/publications/ncd-action-plan/en/>.
5. Baxt IA, Garza-Mayers AC, Goldberg MB. Bacterial subversion of host immune pathways. *Science*. 2013; 340: 697-701.
6. Finlay BB, McFadden G. Anti-immunology: evasion of the host immune system by bacterial and viral pathogens. *Cells*. 2006; 124: 767-82.
7. Dorhol A, Kaufmann SH. Fine-tuning T-cell responses during infection. *Curr Opin Immunol*. 2009; 21: 367-77.
8. Burnet FM. The concept of immunological surveillance. *Prog Exp Tumor Res*. 1970; 13: 1-27.
9. Schreiber RD, Old LJ, Smyth MJ. Cancer immunoediting: integrating immunity's roles in cancer suppression and promotion. *Science*. 2011; 331(6024): 1565-70.
10. Grivennikov SI, Greten FR, Karin M. Immunity, inflammation and cancer. *Cell*. 2010; 140: 883-99.
11. Pêgas KL. Cancer: but what if it were a disease caused by na association between microorganisms? *J Bras Patol Med Lab*. 2018; 54(6): 412-8.



This is an open-access article distributed under the terms of the Creative Commons Attribution License.