

Editorial

Genome's Chaotic Behavior for Adaptation may Explain Carcinogenesis! Suggestion from Surgical Oncologist

Mesut Tez*

Department of Radiology, Ankara Numune Teaching and Research Hospital, Turkey

EDITORIAL

Cancer, especially epithelial cancer (lung, breast, colon etc.), is the most important global public health problem [1]. Understanding of the mechanism of cancer has been gaining momentum for some years on account of its high incidence and impact on the lives of individuals' affected. There are a number of theories of carcinogenesis and these theories may be used to justify various alternative cancer treatments [2,3]. The small variations in cancer mortality observed during the previous years indicate that the clinical applications of these theories have been very limited.

Until now, scientists have spent a lot of time breaking things down into ever smaller component parts – known as reductionism – to understand how each part works in isolation of other parts. Biological systems consist of numerous dynamic networks of biochemical reactions and signaling interactions between active cellular components. This cellular complexity has made it difficult to build a complete understanding of cellular machinery [4-6]. Increased understanding of stem cell biology over the past two decades has uncovered several similarities between cancer cells and normal stem cells, leading to the notion that cancers may arise due to the accumulation of mutations within normal, tissue-resident stem cells.

I try to explain carcinogenesis based on complexity theory.

I described epithelium as a system, stem cell as active adaptive agent and stem cell genome behavior as an agent's behavior.

Ordered behavior

Stem cell has capacities for self-renewal and for asymmetric division leading to generation of epithelial differentiated cells (progenies). The differentiated epithelial cells have to be faithfully maintained to provide a secure barrier and guarantee the body's homeostasis. This is achieved by the constant replacement of exhausted cells with newly formed healthy progeny originating from the stem cells. In many tissues, stem cells are thought to reside within a niche formed by a group of surrounding cells and their extracellular matrix, which provides an optimal environment for stem cell homeostasis. [7]

*Corresponding author

Mesut Tez, Department of Radiology, Ankara Numune Teaching and Research Hospital, Turkey, Tel: 90-312-215 38 34; Fax: 90-312-310 34 60; E-mail: mesuttez@yahoo.com

Submitted: 09 September 2014

Accepted: 14 September 2014

Published: 16 September 2014

Copyright

© 2014 Tez

OPEN ACCESS

The environment around differentiated cell fluctuates regularly and predictably under physiological conditions. The flexibility of stem cell allows to differentiated cells (progeny) to deal with their constantly fluctuating environment. Aging , unpredictable ,previously genetically unknown (such as radiation, new chemicals etc.), weaken the normal function of differentiated cells. Declining functionality is paralleled by diminishing capacity to respond to microenvironmental injury or stress [8].

Edge of chaos

Stem cells can change flexibly according to the environment, while differentiated cells have no such flexibility. Stem cell provide the previously- known, another type of resistant differentiated cells for the protection of epithelial system (transition of epithelium another type of epithelium: such as Barret esophagus, ductal or lobular hyperplasia of breast) or re-organized architecture of epithelium (colo-rectal polyps, gastric polyps etc.)At this state, the system is optimized for adaptation to stressed microenvironment. In this state system is predictable and controllable.

Chaotic behavior

When, microenvironmental stress persists and new epithelial cells continue to die, system gone into a crisis. The crisis causes a series of changes which result in the renewal of the adaptability structure. The stem cell genome jumps to chaotic state for adaptation. Chaotic behavior of the stem cell allows to production of new stress resistant and previously unknown progenies (cancer cells) or atavistic cells (metazoan 1.0). Since this stochastic" behavior is generated by a deterministic chaotic mechanism. Clinically this state represent as local advanced or metastatic carcinomas. Presences of mutated tumor suppressor and/or genes onco-genes facilitate chaotic behavior. Genome chaos acts as a mechanism of rapid, adaptive, genome-based evolution that plays an essential role in promoting rapid macroevolution of new genome-defined systems during crisis. This is consistent with experimental observations that genome chaos, a process of complex, rapid genome re-organization [9].

CONCLUSION

A variety of mathematical approaches to carcinogenesis have been proposed during the past several decades. These modeling studies have generally focused on intracellular processes such as genomic mutations or the interactions of the mutant cells with other cellular populations. Very little attention has been directed to system behavior. Furthermore, modeling system behavior must allow full understanding of the dynamics of carcinogenesis.

REFERENCES

1. Braithwaite D, Boffetta P, Rebbeck TR, Meyskens F. Cancer prevention for global health: a report from the ASPO International Cancer Prevention Interest Group. *Cancer Epidemiol Biomarkers Prev.* 2012; 21: 1606-1610.
2. Croce CM. Oncogenes and cancer. *N Engl J Med.* 2008; 358: 502-511.
3. López-Lázaro M. A new view of carcinogenesis and an alternative approach to cancer therapy. *Mol Med.* 2010; 16: 144-153.
4. McCarthy, Thierry Rakotobe-Joel, Gerry Frizelle. Complex systems theory: implications and promises for manufacturing organizations. *Int. J. Manufacturing Technology and Management.* 2000; 2: 559-579.
5. Jochen F. *The Emergence of Complexity.* 2004; 1-200. Kassel; Kassel University Press GmbH.
6. Couture M. Complexity and chaos - state-of-the-art; overview of theoretical concepts, Tech. Rep. TN 2006-453, DRDC Valcartier.
7. Gallego GA, Villaamil VM, Prado SD, Aparicio LMA. Cancer Stem Cells and Their Niche, *Cancer Stem Cells Theories and Practice*, Prof. Stanley Shostak (Editors). 2011.
8. Sperka T, Rudolph KL. Intestinal Stem Cell Aging. In: Rudolph KL, editors. *Molecular Mechanisms of Adult Stem Cell Aging.* Basel: S. Karger AG. 2010; 63-78.
9. Liu G, Stevens JB, Horne SD, Abdallah BY, Ye KJ, Bremer SW, et al. Genome chaos: survival strategy during crisis. *Cell Cycle.* 2014; 13: 528-537.

Cite this article

Tez M (2014) Genome's Chaotic Behavior for Adaptation may Explain Carcinogenesis! Suggestion from Surgical Oncologist *J Cancer Biol Res* 2(2): 1047.