The Editorial

The pluripotency feature of cancer cells; product of a harmony or an output of a disharmony

Hesam Dehghani[1,2,3]

1- *Embryonic and Stem Cell Biology and Biotechnology Research Group, Institute of Biotechnology, Ferdowsi University of Mashhad, Mashhad, Iran*
2- *Division of Biotechnology, Faculty of Veterinary Medicine, Ferdowsi University of Mashhad, Mashhad, Iran*
3- *Editorial Board Member of JCMR, JCMR Office, Department of Biology, Faculty of Sciences, Ferdowsi University of Mashhad, Mashhad, Iran*

Received 31 Jul 2014 Accepted 04 Sep 2014

Summary

Pluripotency is a central feature of stem and cancer cells. This feature enables cancer cells to self-renew and trans-differentiate. In this editorial, it is hypothesized that in cancer cells synchronized events rather than segmented procedures may lead to pluripotency.

***Keywords*:** Pluripotency, Stem cells, Cancer cells

The state of pluripotency is operationally defined by self-renewal and a capacity to differentiate into multiple lineages. However, numerous features have also been discovered that contribute to pluripotency and immortality. These features include high level of transcription, open chromatin and nuclear organization, re-expression of telomerase, activation of c-myc network, activity of specific transcription factors, and utilization of certain cell signaling pathways. In certain pluripotent states like what is existent in primordial germ cells, some other features like migratory behavior has also been observed.

Recent findings indicate that cancer cells and pluripotent cells (embryonic stem cells, adult stem cells, and primordial germ cells in some in vitro conditions) possess and use similar features. It seems that features of open organization of chromatin and increased level of transcription are shared between cancer and pluripotent cells (Dehghani et al., 2005, Efroni et al., 2008, Gaspar-Maia et al., 2011). Similarly, the increased activity of stemness transcription factors has been documented in both cancer and stem cells (Apostolou et al., 2012, Liu et al., 2013). On the other hand, the myc regulatory network frequently known for its involvement in cancer is also characterized to be a major player of pluripotency (Hurlin et al., 2013, Hirasaki et al., 2013, Chappell et al., 2013, Hann et al., 2014). With these similarities in mind and considering that cancer cells partially fulfill the requirements for the operational definition of stem cells, ability to self-renew and trans-differentiation into different cells (Sabe et al., 2011, Barneda-Zahonero et al., 2012, Shekhani et al., 2013), the important question that arises is that if the pluripotency feature is necessary for the cancerous nature of cancer cells, and whether this feature ever contributes to the pathogenesis of cancer.

Becoming a cancer cell is a much more coordinated and synchronized phenomenon that could be justified only by segmented cellular events such as mutations, cell-cycle control defects, and disengagement of apoptotic mechanisms. Inspired by Gottfried Wilhelm von Leibniz, the 17th century philosopher, there could be a pre-established synchronization and harmony among different features in order to reach a specific state of activity or a specific nature like cancer. Perhaps it is a harmony and synchronization between different features that provides a new identity for cancer cells. This is in prominent contrast to the idea that it is a disharmony and dysregulation that brings about cancer. If after all cancer is the result of a harmony of selected cellular features and abilities, there will be no doubt that the stemness and pluripotency is one of the most central features of cancer, which is actively regulated in sync with other cellular features.

∗Corresponding author E-mail:
Member of Editorial Board, JCMR
dehghani@um.ac.ir

References:

1. Apostolou P., et al. (2012) Cancer stem cells stemness transcription factors expression correlates with breast cancer disease stage. Current Stem Cell Research Therapy 7(6): p. 415-9.
2. Barneda-Zahonero, B., et al. (2012) Epigenetic regulation of B lymphocyte differentiation, transdifferentiation, and reprogramming. Computer Functional Genomics 564381.
3. Chappell J. and Dalton S. (2013) Roles for MYC in the establishment and maintenance of pluripotency. Cold Spring Harb Perspect Med 3(12): a014381.
4. Dehghani H., Dellaire G. and Bazett-Jones D.P. (2005) Organization of chromatin in the interphase mammalian cell. Micron 36(2): p. 95-108.
5. Efroni S., et al. (2008) Global transcription in pluripotent embryonic stem cells. Cell Stem Cell 2(5): p. 437-47.
6. Gaspar-Maia A., et al. (2011) Open chromatin in pluripotency and reprogramming. Nature Reviews Molecular Cell Biology 12(1): p. 36-47.
7. Hann S. R. (2014) MYC Cofactors: Molecular Switches Controlling Diverse Biological Outcomes. Cold Spring Harb Perspect Med. 17:4(9) a014399
8. Hirasaki M., et al. (2013) Striking similarity in the gene expression levels of individual Myc module members among ESCs, EpiSCs, and partial iPSCs. PLoS One 8(12): e83769.
9. Hurlin P. J. (2013) Control of vertebrate development by MYC. Cold Spring HarbPerspect Med,. 3(9): a014332.
10. Liu A., Yu X. and Liu S. (2013) Pluripotency transcription factors and cancer stem cells: small genes make a big difference. Clinical Journal of Cancer 32(9): p. 483-7.
11. Sabe H. (2011) Cancer early dissemination: cancerous epithelial-mesenchymal transdifferentiation and transforming growth factor beta signalling. Journal of Biochemistry 149(6): 633-9.
12. Shekhani M. T., et al. (2013) Cancer stem cells and tumor transdifferentiation: implications for novel therapeutic strategies. American Journal of Stem Cells 2(1): 52-61.