

Cellular Polarity in Physiology and Cancer

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Needless to say that all the multicellular organisms consists of remarkably organized and widely distributed epithelial tissues, which have specific structural and functional roles. The integrity of the architecture of these tissues is vital and is maintained by millions of epithelial cells functioning as a dynamic system. The epithelial cells are integrated and regulated by various biological mechanisms, governing the cellular proliferation, death and their replenishment.¹

One such regulatory factor is cellular polarity. It is characterized by asymmetry in the shape of the cell, distribution of the cellular organelles and functions of the cell. A subtle balance of the cell polarity signaling pathways is essential for maintenance of diverse physiological processes such as cell growth and development, molecular transport, cell migration, etc.² Consequently, any deregulation in the polarity of the cell can change its course. Therefore, the loss of polarity of cell is often appreciated commonly in the epithelial neoplasms, leading to the formation of a disorganized tissue. Furthermore, evidence suggests that the proteins involved in cell polarity are targets of several oncogenes. In addition, some tumor suppressor genes are thought to regulate the pathways that maintain polarity of the cells.³

Various types of cellular polarity exist such as front-rear polarity, planar cell polarity, mitotic spindle polarity, and apical-basal polarity. The epithelial cells characteristically exhibit the apical-basal polarity and the loss of which is thought to be involved in the initiation of malignancy.² It is regulated by three major complexes namely; the Crumbs complex, the Par complex, and the Scribble complex.³

The fundamental functions of apical-basal polarity in relation to tumor suppression include the upkeep of the apical junctional complex (AJC) and the conservation of asymmetric cell division.³ The AJC comprises adherens and tight junctions. Acquirement of mutations disrupts the junctional complexes, and the cells lose their communication with their adjacent cells. These transformed cells may further undergo epithelial-mesenchymal transformation, which constitute one of the important tools of metastasis.⁴

As mentioned, the polarity proteins aid in the maintenance of the asymmetric division of the stem cells. The asymmetric cell division produces one self-renewal unit and one differentiated daughter cell. When the polarity proteins are disrupted, there is a shift from the asymmetric to symmetric cells division, initiating the formation of neoplastic cells.⁴

Compelling evidence has also been put forth in relation to the role of viruses in disruption of cellular polarity. The viruses which undergo aggressive lytic cycle are rarely associated with the development of malignancy. Whereas most of the oncogenic viruses are known to persist in the host cells for long-term, integrate into them and exploit the multiplication mechanism for their survival and propagation. In the process of doing so, the virus attacks on the various cell polarity signaling pathways, e.g., the human papillomavirus, known to target multiple cell polarity regulators, which further makes it challenging to ascertain the significant molecules in relation to the stages of cancer development and/or in the life cycle of the virus.⁵

To conclude, as the research progresses with respect to cancer etiopathogenesis and treatment, novel evidences are emerging to our knowledge day-to-day. At present, the role of polarity proteins seems to be shielding and guarding the cells from undergoing neoplastic transformations.

References

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