Preface

The classic hallmarks of cancer are a poorly differentiated phenotype, and a cellular and genetic heterogeneity. In the past, the cellular diversity of cancer has mostly been attributed to the genetic instability of its cells. As the tumor cell population expands, individual cells pick up random mutations, and their molecular identity starts to diverge. By the time the cancer is detected, the millions of cells that make up the tumor have become as different from each other.

Cancer stem cells (CSCs) or, as defined by other authors, tumor-maintaining cells or cancer stem-like cells are a subpopulation of cancer cells that acquired some of the characteristics of stem cells to survive and adapt to ever-changing environments. These include the ability to self-renew and the capacity to produce progenitors that differentiate into other cell types.

It has been originally hypothesized that CSCs could potentially arise from normal stem or early progenitors. Now, the longstanding notion that fully committed and specialized cells might de-differentiate over the course of tumor initiation and progression to originate CSCs has been reevaluated. At present, data emerge to indicate that cancer cells that resemble stem cells need not be part of the original tumor but rather may emerge during later stages of tumor development. The observed tumor heterogeneity is probably a combination of growing genomic instability and epigenetic instability associated with the acquisition of a stem cell-like phenotype. These instability promote a new a fundamental peculiarity of CSCs, i.e., genetic plasticity.

CSCs represent the ideal justification for a lot of intriguing and obscure aspects of cancer pathogenesis (i.e., cancer cell dormancy, chemoresistance, local and distant relapses). The complex pathophysiology of CSCs and its important direct and indirect implications in molecular and cellular biology of cancer, at present, render this topic particularly interesting for Chemists, Biochemists, Pharmacologists, Biologists, Geneticists who are studying different aspect of experimental oncology. Moreover, considering the enormity of the clinical implications related to CSCs and/or to "cancer cells like stem cell," a growing number of researchers should modify and/or adapt its field of study in consideration of this relatively new topic.

At last, the identification of a molecular phenotype for these modified stem cells, associated to an accurate definition of their typical derangement in cell differentiation and metabolism, can represent a fundamental advance in terms of early diagnosis and selective therapy of cancer. At last but not least, the knowledge of pathogenetic mechanisms at the basis of CSCs can enlarge and ameliorate the therapeutic applications of the normal adult stem cells (i.e., regenerative medicine, tissue engineering, biotechnology applications) by reducing the risk of a deranged, uncontrolled, and thereby potentially tumorigenic stem cell differentiation.

A critical and continuous updating to the different pathophysiological aspects of this CSC may certainly help the development of a research, not only limited to cancer but also really useful and harmless for patients, by stimulating potential clinical applications in terms of diagnosis and above all of therapy.

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