

# Preface

Recently, there has been increasing support for the “cancer stem cell” hypothesis, which postulates that cancer arises from a subpopulation of tumor-initiating cells or cancer stem cells (CSCs). There are currently two conflicting views that attempt to explain tumor formation. The classical stochastic model suggests that every cell within a tumor is a potential tumor-initiator, but that entry into the cell cycle is governed by a low probability of stochastic mutations. According to this model, it would be impossible to tell which cell initiated the tumor since each cell has an equal ability to be malignant. By contrast, the hierarchy theory (upon which the CSC hypothesis is based) proposes that only a subset of cells within a tumor is capable of initiating tumor growth, but that these cells all do so at a high frequency. According to this theory, it should be possible to identify and target the cells responsible for tumor initiation and progression because not all cells have the same phenotypic and functional characteristics.

While the idea of CSCs has been around for more than 100 years, evidence from the hematology field has now demonstrated the critical role of stem cells in hematological malignancies and suggested that these same mechanisms could also be central to the initiation, progression, and treatment of solid cancers. Indeed, several pivotal studies have recently provided compelling evidence that these cells do exist in solid tumors of many types including breast, brain, colorectal, pancreas, prostate, melanoma, lung, ovarian, liver, and head and neck cancer. Furthermore, clinical and experimental studies have demonstrated that CSCs exhibit many classical properties of normal stem cells, including a high self-renewal capacity and the ability to generate heterogeneous lineages; the requirement for a specific “niche”/microenvironment to grow; and an increased capacity for self-protection against harsh environments, toxins, and drugs.

This multi-authored volume focuses specifically on the role of CSCs in solid cancers. The authors are all active investigators with research programs related to oncology and/or stem cell biology, and are leaders in their field. Part I (Chap. 1) serves to introduce the concept of CSCs vs. normal stem cells, including a historical perspective and the contributing lessons from leukemia. Part II (Chaps. 2–11) describes the identification and role of CSCs in various forms of solid cancer,

organized according to disease site. Part III (Chaps. 12–14) elaborates on molecular pathways that are involved in driving CSC function, with a particular focus on the convergence of embryonic and tumorigenic signaling pathways. Part IV (Chaps. 15–18) describes available model systems and modalities for studying CSC biology and therapeutic development, including in vitro and in vivo model systems and assays and imaging modalities. Part V (Chaps. 19–23) discusses the importance of CSCs for cancer management and treatment, including implications for prognosis, prediction, and treatment resistance. Finally, Part VI (Chap. 24) provides the concluding thoughts for the book, including consideration of the controversy surrounding the CSC hypothesis. The editor and the authors hope that this work will provide a comprehensive overview of this evolving and important field.

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