

1. Record Nr.	UNINA9910137088203321
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Titolo	Cellular and phenotypic plasticity in cancer [[electronic resource] /] / edited by Petranel Theresa Ferrao, Andreas Behren, Robin Anderson and Erik Thompson
Pubbl/distr/stampa	Frontiers Media SA, 2015 France : , : Frontiers Media SA, , 2015
ISBN	9782889196623 (ebook)
Descrizione fisica	1 online resource (77 pages) : illustrations
Collana	Frontiers Research Topics
Soggetti	Cancer
Lingua di pubblicazione	Inglese
Formato	Materiale a stampa
Livello bibliografico	Monografia
Nota di bibliografia	Includes bibliographical references.
Sommario/riassunto	The process of Epithelial-Mesenchymal-Transition (EMT) is known to result in a phenotype change in cells from a proliferative state to a more invasive state. EMT has been reported to drive the metastatic spread of various cancers and has also been associated with drug resistance to cytotoxics and targeted therapeutics. Recently phenotype switching akin to EMT has been reported in non-epithelial cancers such as metastatic melanoma. This process involves changes in EMT-Transcription Factors (EMT-TFs), suggesting that phenotype-switching may be common to several tumour types. It remains unclear as to whether the presence of both Epithelial-like and Mesenchymal-like cells are a pre-requisite for phenotype switching within a tumour, how this heterogeneity is regulated, and if alteration of cell phenotype is sufficient to mediate migratory changes, or whether drivers of cell migration result in an associated phenotype switch in cancer cells. Similarly it has yet to be clarified if cells in an altered phenotype can be refractory to drug therapy or whether mediators of drug resistance induce a concurrent phenotypic change. Little is known today about the underlying genetic, epigenetic and transient changes that accompany this phenotypic switch and about the role for the tumor micro-environment in influencing it. Hence this is currently an area of

speculation and keen interest in the Oncology field with wide-ranging translational implications. In this Frontiers Research Topic, we discuss our current understanding of these concepts in various cancer types including breast cancer, colorectal cancer and metastatic melanoma. This topic covers how these processes of cellular and phenotypic plasticity are regulated and how they relate to cancer initiation, progression, dormancy, metastases and response to cytotoxics or targeted therapies.

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