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Editorial

Introduction to the Special issue “Advances in Molecular Oncology and personalized medicine: individuating molecular targets for intervention and amelioration of patients status”. Editorial.

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Scope of the thematic issue is to provide an update on the effects of modifications (at single nucleotide changes or chromosome translocation), gene silencing by chromatin repression or by mRNA translation block by microRNA activation, and to discuss the possible amplification effects and collaboration between different oncogenic pathways to the development and progression of cancerogenesis process. Driver mutations, epigenetic deregulation, DNA damage, telomere control escape, and changes in RNAs levels affect the cell differentiation state, potentially contributing to the stemness and the proliferation at an embryo-like state, as for cancer stem cells.

Knowledge on deregulation of signaling pathways and altered gene expression in cancer are rapidly progressing. In this special issue we invited contributors to discuss various ways cancer cells evade the control of gatekeeper genes determining the cell fate and finality of cellular processes (DNA damage repair, cell cycling, G/M phase transition), the transcription and post-transcriptional regulation (microRNAs, nucleotide mutations, altered splicing) and various aspects of structural RNAs regulating the assembling and function of protein complexes (HOTAIR, HOTAIRM1, SAMMSON, BANCR, FAL1) in euchromatin /heterochromatin organization), and the Epithelial to Mesenchymal Transition (EMT), the higher metabolic rate, hypoxia response and the intercellular communication and inhibition of growth. Finally, few reviews described the benefits and drawbacks of inhibitors targeting the DNA repair proteins and the ongoing trials that will bring new perspectives on the applicability of these compounds in anticancer therapies.

We open the special presenting the discussion on the highly complicated nature of EMT by Simeone et al. The authors point out on the network of factors involved in the control of the transition [1]. Mughal and collaborators open their review describing the DNA replication licensing complex and its deregulated expression as hallmark in numerous cancers. They focus on the impact of DNA replication complex during normal and cancer development and on the possibility to develop drugs able to reduce the expression of licensing proteins for anticancer therapies [2]. Oliver Schildgen and Verena Schildgen first defined the concept of driver mutations and then provided an overview about how this concept has progressively changed after the advent of NGS technology. Different mutations can occur even in a single cell and they can determine the level of malignancy as well as the therapeutic outcome [3]. DNA damage and DNA repair physiology and their aberrations in cancer are reviewed by Motegi et al. They further indicated the principal alteration of the DNA response leading to cancer and discuss the impact on therapeutic potential in cancer treatment [4]. Altered expression of microRNAs (miRNAs) is often found in several cancer types. Farooqi and collaborators review the transcriptional control of miRNAs at epigenetic level, the interplay of non coding-RNAs (nc-RNAs) in controlling epigenetically miRNAs expression and the effects of sponge nc-RNAs. Following, they give examples of the recently discovered function of miRNAs as epigenetic regulators of gene expression [5]. Fayyaz et al. describe the importance of miRNAs in the apoptosis control. In particular they comprehensively explained the function of miRNAs in layered regulation of TRAIL mediated apoptosis speculating on the possibility to translate these findings into therapeutic approaches [6]. In a further review [7], Farooqi et al. explain the complexity of the signal transduction deregulation in colorectal cancer (CC) describing the putative dynamic role of expression changes in voltage-gated ion channel, while Chen et al. [8] underline the feasibility of computational analysis in the understanding the signaling network alteration in gastroenteropancreatic neuroendocrine cancers. Arnaiz, Sole and coworkers review two important aspects of non-coding RNA biology related to cancer. First, they describe physiological and pathological function of a novel class of ncRNAs, the circular RNAs, emphasizing on the possibility to use them as biomarkers in tissue biopsy [9], then they highlight how studies on the circulating transcriptome positively impacted on the finding for novel non invasive cancer biomarkers [10]. Many attempts to find drugs that affect tumor cells with light effects on normal cells are undergoing in several labs. Tang et al. bring into focus the main knowledge on the oxidative-stress modulating drugs that allow preferential anticancer effect [11]. Resistance to therapy is a common feature of glioblastoma malignancy. Tomiyama et al. summarize the current knowledge on the molecular network running the glioma biology and the impact of the signal transduction pathway on the therapy resistance [12]. In addition, BalÇa-Silva and collaborators describe the relevance of the blood-brain barrier in determination of therapy resistance and give example of novel drug delivery strategies in glioblastoma [13]. The Guest Editors and contributors to this special issue of the journal Seminars in Cancer Biology hope that basic researchers and clinicians will read these review articles with great interest, wishing that these reviews can help and satisfy the broadest range of scientific readers.

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Massimo Mallardo

Department of Molecular Medicine and Medical Biotechnology, University of Naples, "Federico II"via Pansini 5, Napoli, Italy

E-mail address: massimo.mallardo@unina.it.

Palmiro Poltronieri

Agrofood Department, National Research Council, CNR-ISPA via Monteroni km. 7, Lecce, Italy

E-mail address: palmiro.poltronieri@ispa.cnr.it.

Ammad A. Farooqi

Department of Molecular Oncology, Institute of Biomedical and Genetic Engineering (IBGE), Islamabad, Pakistan

E-mail address: ammadfarooqi@rlmclahore.com.