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Regulation of cancer stem cell fates: From onco-metabolic-epigenetic reprogramming perspective

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The heterogeneity within cancer is proposed to be driven by the population of cells within the tumor mass endowed with stem cell characteristics, called cancer stem cells (CSCs). These CSCs have been attributed to resistance towards conventional cancer treatment, leading to treatment failure and thus poor therapeutic outcome. Different CSCs fates may be linked to distinct cellular programs governed by specific molecular switch and axes in a context-dependent manner, leading to tumor resistant phenotype. Thus, developing CSC-targeting therapies is of major interest which requires insight understanding of the unique molecular circuitry that regulates the CSCs and their cellular fates. Our current work focuses on the elucidation of cellular and molecular characteristics that are associated with CSCs associated with their resistant characteristics: stemness, proliferation, epithelial-mesenchymal transition and quiescence. Our results revealed distinct expression pattern exhibited in the CSC-associated gene expression pattern which is associated with different cellular fates in different *in vitro* models. The acquisition of CSC-phenotypes may be facilitated by intricate cellular program through the reprogramming of their cellular metabolism, known as onco-metabolic reprogramming/metabolic switch. Specifically, Lin28 has been previously identified as a possible regulator that governs the stem cell fate through metabolic reprogramming, which was exclusively associated with stemness and resistance characteristics. It also found to be involved in regulating CSC metabolic pathways. This pave a path to the investigation into the molecular circuitry of the heterogeneous CSC phenotypes, possibly via onco-metabolic reprogramming and stemness regulatory axis and thus this axis would be important target for the treatment of cancer. In summary, our research would likely have a significant impact on designing and developing novel approaches for targeting the key regulators in CSCs as novel targeted approach for the inhibition of cancer progression and treatment with better therapeutic outcome.

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