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Opinion Article

Stemness Regulators of Cancer and Embryonic Stem Cells

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DESCRIPTION

The two unique properties of Embryonic Stem Cells (ESCs) are self-renewal and pluripotency. Mouse ESCs (mESCs) are isolated from day 3.5 blastocyst and have ground state pluripotency whereas human ESCs (hESCs) are isolated from late blastocyst and correspond to the epiblast stem cells of the mouse. The pluripotency of ESCs is determined by the intensive action of signaling pathways that react to external stimuli, basically expressed transcription factors and complexes that govern the epigenetic state. The prolonged transcriptional network of ESCs is centered on the triad of master regulators of pluripotency Oct4, Sox2 and Nanog. In the last decade the introduction in somatic cells of transcription factors (Oct4, Sox2, Klf4, c-Myc), microRNAs and small molecules allowed the generation of Induced Pluripotent Stem (iPS) Cells. Due to their ability to give rise to any type of distinguished cells and tissues, both ES and iPS ells offer many opportunities for modeling human diseases and progress of regenerative medicine.

ESCs and tumor cells share numerous common properties demonstrated by rapid proliferation, similar metabolic requirements and inhibition of differentiation. Pluripotent ESCs have essential tumorigenic potential and they produce benign tumors and teratomas when injected in immunodeficient mice. Reprogramming of somatic cells into pluripotency by oncogenes like Myc and Klf4 suggest a strong connection between pluripotency and tumorigenicity. Currently, rising experimental evidence has disclosed that tumors contain a variable number of cells that have self-renewal and partial differentiation capacities. Because these cells share these properties with the adult tissue stem cells from which they are likely derived they were termed Cancer Stem Cells (CSCs). The procedures of somatic cell reprogramming and CSC formation are both dependent on transitions between epithelial and mesenchymal states (EMT/MET). In addition, CSCs from epithelial tumors also exhibit ESC-like signatures that include the oncogene *c-Myc* and factors important for pluripotency such as Sox2, Dnmt1, Cbx3 and HDAC1.

The CSC model eventually links Cancer with Stem cell biology and delivers a common framework that is suggested to account for all the properties of stem cells, irrespective of their initial or late developmental origin in normal or pathological states. This is analyzed that common regulatory mechanisms of embryonic and CSCs focusing on biomarkers, signaling pathways, transcription factors and epigenetic complexes. This information can explain the risks stopping from the tumorigenic potential of pluripotent ESC and iPS cells used in tissue regeneration therapies. Furthermore, knowledge about their stem cell properties is essential for the eradication of CSCs that are responsible for therapy resistance, tumor invasion and metastasis.

CONCLUSION

Pluripotency of Embryonic Stem Cells (ESCs) and induced pluripotent stem cells is controlled by a well categorized gene transcription circuitry. The circuitry is assembled by ESC exact transcription factors, signal transducing molecules and epigenetic regulators. Developing understanding of stem-like cells, albeit of more complex phenotypes, present in tumors (cancer stem cells), delivers a common conceptual and research framework for basic and applied stem cell biology. Gene signatures, signaling pathways and epigenetic regulators that are common in embryonic and cancer stem cells. Their potential use to design next generation biological and pharmaceutical approaches for regenerative medicine and cancer therapies.

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