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Molecular modulation of autophagy: New venture to target resistant cancer stem cells

Mandhair HK *et al.* Modulation of autophagy in CSCs

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Abstract

Autophagy is a highly regulated catabolic process in which superfluous, damaged organelles and other cytoplasmic constituents are delivered to the lysosome for clearance and the generation of macromolecule substrates during basal or stressed conditions. Autophagy is a bimodal process with a context dependent role in the initiation and the development of cancers. For instance, autophagy provides an adaptive response to cancer stem cells to survive metabolic stresses, by influencing disease propagation *via* modulation of essential signaling pathways or by promoting resistance to chemotherapeutics. Autophagy has been implicated in a cross talk with

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Moreover, it is likely that emerging research regarding the role of **autophagy** in **cancer stem cells**, in **epithelial-to-mesenchymal transition**, in DNA damage and cell cycle control, in the regulation of inflammatory signalling and in other aspects of **cancer biology** will further shift the existing paradigms that dictate our current understanding of **autophagy** as a **target** in **cancer therapy**.

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Autophagy-Induced Resistance of Cancer Stem Cells to Chemotherapy Diverse **molecular mechanisms** contribute to **CSC resistance** to drug treatment, including cellular plasticity, highly efficient DNA damage repair, expression of genes related to **multi-drug resistance**, and prevention of **apoptosis**.

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[Autophagy, cancer stem cells and drug resistance](#)

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Autophagy also promotes the survival of **mesenchymal stem cells** and **human embryonic stem cells** [37,38] and is required for the quiescent state of **muscle stem cells**. **Autophagy** induces pluripotency with pluripotency factor SOX2 repressing mTOR expression, resulting in the increased **autophagy** necessary to reprogram somatic cells into induced **pluripotent stem cells** [50,51].

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