INSTRUCTIONS

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Epigenetic modulation of autophagy to induce cancer cell dormancy

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Tumor dormancy refers to the stable state of tumor mass and of micrometastases, a condition that can be reversed and give rise to cancer relapse and progression. Tumor dormancy is attained through three different mechanisms that may act simultaneously and synergy, i.e. by the lack of blood supply (angiogenic dormancy), the active tumor killing by T-lymphocytes and NK cells (immune-mediated dormancy), and by keeping the tumor cells in a resting state out of the cell cycle (cancer cell dormancy). The latter has been shown to rely on autophagy to sustain cell metabolism for survival under the lack of nutrients. Autophagy is a macromolecular and organelle degradation pathway accomplished through the sequestration of the redundant and damaged cellular structures within autophagosomes which would then fuse with lysosomes for the complete hydrolysis and recycling of molecular subunits. Autophagy negatively controls cell proliferation and migration. Consistently, oncogenes inhibit while tumor suppressor genes promote autophagy. Analogously, signaling pathways triggered by growth factors, amino acids and glucose inhibit autophagy while the lack of nutrients and of oxygen trigger autophagy. Autophagy is controlled through epigenetics by tumor microenvironmental triggers, including inflammatory cytokines. Here, we present data showing that extracellular triggers may stimulate autophagy to induce cancer cell dormancy by modulating the expression of the transcriptome and particularly of the micrornome.