Cáncer - Póster

Extracellular vesicles derived from TNF-? conditioned macrophages promote endocrine resistance in breast tumor cells

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Breast cancer is one of the leading tumors diagnosed worldwide and the primary cause of cancer-related death in women. Nearly 70% of diagnosed breast tumors are estrogen receptor-positive (ER+), becoming the receptor a key therapeutic target. Despite the efficacy of hormonal therapies, some patients develop resistance, either initially or during the course of treatment. Understanding tumor progression and resistance mechanisms requires studying the tumor microenvironment, where tumor cells interact closely with endothelial cells, cytokines, soluble factors, fibroblasts, and immune cells, particularly macrophages, which are highly influential in tumor behavior. Cells communicate through direct contact, endocrine and paracrine soluble factors, and extracellular vesicles (EVs). These lipid bilayer vesicles facilitate the transfer of their cargo to recipient cells, inducing phenotypic changes. Here, we isolated EVs derived from TNF-?-conditioned macrophages and investigated their effects on MCF-7 ER+ breast tumor cells. Our findings indicated that these vesicles enhance proliferation, migration, induced epithelial-mesenchymal transition and a tumor stem cell phenotype. Furthermore, we identified a link between these EVs and endocrine resistance, demonstrating that vesicles not only increased cell proliferation in the presence of Tamoxifen, an ER modulator, but also transferred endocrine resistance to previously sensitive cells.