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Targeting resistin signaling pathway as a strategy for enhancing the effect of chemotherapy drugs in reducing breast cancer cells aggressiveness

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Obesity has been linked to increased cancer incidence and poor clinical outcome. Levels of resistin, a proinflammatory cytokine produced by the white adipose tissue adipocytes and macrophages, are found to be elevated in obese individuals. Our results demonstrated that in MCF-7 breast cancer cells, resistin induces the formation of filopodia and lamellipodia, reorganization of the cellular stress fibers and separation of single cells from the cellular clusters. We also observed increased breast cancer cell migration after resistin treatment of MCF-7 and MDA-MB-231 cells subjected to scratch migration wound healing assay. Further experiments revealed that resistin upregulates mRNA and protein expression of key transcription factors involved in the epithelial to mesenchymal transition (SNAIL, SLUG, ZEB1 and TWIST) and the mesenchymal markers fibronectin and vimentin. In the same time, the expression of the epithelial markers E-cadherin and claudin-1 was downregulated. Recent studies have shown that resistin also interferes with chemotherapy affecting the efficacy of doxorubicin, significantly attenuating doxorubicin-induced apoptosis in a dose- and time-dependent manner. Here, we propose a hypothesis that creating a monoclonal antibody drug that either blocks resistin signaling pathway or marks resistin for further destruction by lymphocytes, can be a potential strategy for enhancing the effect of chemotherapy drugs on apoptosis and/or attenuating the aggressiveness of cancer cells.

Biography

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