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TNF-α Increases IP-10 expression in MCF-7 breast Cancer Cells via Activation of the JNK/c-Jun pathways

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IP-10 (also called CXCL10) plays a significant role in leukocyte homing to inflamed tissues, and increased IP-10 levels are associated with the pathologies of various inflammatory disorders, including type 2 diabetes, atherosclerosis, and cancer. TNF-α is a potent activator of immune cells and induces inflammatory cytokine expression in these cells. However, it is unclear whether TNF-α is able to induce IP-10 expression in MCF-7 breast cancer cells. We therefore determined IP-10 expression in TNF-α-treated MCF-7 cells and investigated the mechanism involved. Our data show that TNF-α induced/upregulated the IP-10 expression at both mRNA and protein levels in MCF-7 cells. Inhibition of JNK (SP600125) significantly suppressed the TNF-α-induced IP-10 in MCF-7 cells, while the inhibition of p38 MAPK (SB203580), MEK1/2 (U0126), and ERK1/2 (PD98059) had no significant effect. Furthermore, TNF-α-induced IP-10 expression was abolished in MCF-7 cells deficient in JNK. Similar results were obtained using MCF-7 cells deficient in c-Jun. Moreover, the JNK kinase inhibitor markedly reduced the TNF-α-induced JNK and c-Jun phosphorylation. The kinase activity of JNK induced by TNF-α stimulation of MCF-7 cells was significantly inhibited by SP600125. Altogether, our novel findings provide the evidence that TNF-α induces IP-10 expression in MCF-7 breast cancer cells via activation of the JNK/c-Jun signaling pathway.

Biography

Amnah Al-Sayyar (BEng, MSc), a chemical engineer and a certified associate project manager with an interest in medical research and disease management. She is currently working with the Immunology and Microbiology lab at Dasman Diabetes Institute in Kuwait for almost two years and at the early stages of her research career. Her main research focus revolves around identifying novel metabolic markers and signaling pathways that are associated with diabetes and its related complications. Such research focus will enable the development of therapeutic targets and prevention methods which in turn will assist the drug discovery process for enhancing metabolic health.