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Mimicking the effect of prolactin on STAT3/STAT5 activity in breast cancer

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ABSTRACT

Signal transducers and activators of transcription (STAT) 3 and 5 are commonly constitutively activated in breast cancer. STAT5 can outcompete STAT3 and reduce cell proliferation and metastasis. STAT5 activation is stimulated by prolactin, a natural hormone that can be harmful at high levels. The aim of this study is to identify some possible previously developed drugs that mimic the effect of prolactin and STAT5 without the added risk in MDA-MB231 breast cancer cells. Using the CLUE database query app and STAT5 up- and downregulation signatures, three drugs (X, K, and M) were chosen based on their similarity in signatures to those of STAT5. These drugs were then analyzed for cell viability to determine dosage. qPCR was used to compare the expression of STAT5 target genes after drug treatment and the expression after STAT5 activation with prolactin. The drug K was found to show a similar impact on target gene expression to prolactin. Further research is required, but from this study K seems to be a promising option for stimulating STAT5 and improving breast cancer prognosis.