THE STUDY OF THE RELATIONSHIP OF HS6ST2 WITH CXCL14 AND IFIT1 IN BREAST CANCER

From previous study, the silencing of HS6ST2 in MCF7 demonstrated enhanced migration and invasion, while the DNA microarray picked out two candidate genes: CSCL14 and IFIT1. Primarily, findings from HS6ST2 silencing were corroborated via HS6ST2 over-expression in MDA-MB-231 and MCF7, and they were in agreement. Through literature study, CXCL14 has a role in promoting migration and invasion, indicative of CXCL14's involvement in HS6ST2-silenced cells. Using double silencing, the invasion and migration capabilities seen in HS6ST2-silenced cells were abolished and the cells behaved more like nonsilenced cells. Additionally, DNA microarray together with gene ontology postulated that the changes observed could be further attributed to the TGF- β pathway through SMAD2/3 signalling. On the contrary, IFIT1 has unreported functions in breast cancer. Novel findings of IFIT1 have shown the protein to be pro-tumour advancement. Through IFIT1 silencing in MDA-MB-231 and MCF7, there were significant decrease in migration, invasion and proliferation. Additionally, IHC of HS6ST2, CXCL14 and IFIT1 in IDC TMAs were also performed to observe distribution in the tissues and various statistical analyses were done to evaluate the correlation of staining intensities with clinicopathological parameters, disease survival and recurrence. To date, this study is the first in exploring the possible relationship between HS6ST2 and CXCL14, as well as IFIT1, in breast cancer progression.

Speaker :

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