**RNA interference-mediated targeting of CTNNB1 gene expression in vulva carcinoma cells causes increased tumor cell invasion and migration**

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BACKGROUND: Alterations in cell adhesion are among the hallmark characteristics of a malignant epithelial tumor, including irregularities in expression and distribution of adhesion molecules, such β-catenin. Stabilization of cytoplasmic β-catenin by aberrant activation of Wnt signaling leads to its accumulation, complexed with lymphoid enhancer factor/Tcf (LEF/Tcf) transcription factors and transactivation of LEF/Tcf target genes. Some studies reported that reduced assembly of membranous β-catenin induces up-regulation of Slug and the mesenchymal marker, Vimentin and show reduced intercellular adhesion as well as increased motility. β-catenin may act as an oncoprotein, becoming one of the key downstream effectors in the Wnt signalling pathway and represents an essential role in tumor invasion and migration. HYPOTHESIS: For this reason, we aimed to determine the role of β-catenin in vulva carcinoma cell invasion and migration by short hairpin RNAs (shRNAs) technology. METHODS: Vulva cancer cell was transfected at high efficiency with specific CTNNB1 shRNA. RT-PCR and western blot analysis were used to determine the mRNA and protein levels of β-catenin in shRNA-treated and -untreated cells. In addition, the invasion and migration of the vulva cancer cell was detected by invasion and migration assays. RESULTS: Stable transfection of CTNNB1 shRNA significantly inhibited the mRNA and protein levels of β-catenin. Markedly increased cell invasion and migration was observed following treatment with CTNNB1 shRNA when compared with the negative control shRNA-treated and shRNA-untreated cells. The RNAi-mediated targeting of CTNNB1 gene expression in vulva cancer cell resulted in increased tumor cell invasion and migration *in vitro*. β-catenin was identified as an stimulator of vulva cell invasion and migration via its novel role in the Wnt signaling pathway.