Silencing of *STIM1* Inhibits Epstein-Barr Virus (EBV)-Related Nasopharyngeal Carcinoma Cell Lines Survival by Regulating MIR-200A-3p and MIR-375 Activities

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Epstein-Barr virus (EBV) infection is associated with many cancers pathogenesis, especially nasopharyngeal cancer (NPC). This infection has been reported to promote metastatic potential via stromal interaction molecule 1 (*STIM1)*. However, the post-transcriptional regulation of *STIM1* activities in EBV-related NPC is not well understood yet. This study aims to investigate the post-transcriptional regulatory roles of *STIM1* silencing in EBV-related NPC. C666-1 cells is an NPC cell line model harbouring EBV. The C666-1 cells were transfected with Dicer substrate short interfering RNA (*DsiRNA*) targeting *STIM1* for 48 and 72 h periods. The expression of *STIM1* and a set of microRNAs were determined via qRT-PCR. The post-translational expressions of RAC1 and CDKN1B proteins were determined via western blot profile. The functional profile of *STIM1* silencing showed inhibition of C666-1 cells proliferation and migration activities. Our molecular finding suggests that *STIM1* silencing could promote tumor suppressor activities via miR-375 and CDKN1B expression. This study suggests that *STIM1* silencing could suppress the survival of EBV-related NPC cells via post-transcriptional regulation of miR-375 and miR-200a-3p as well as post translational regulation of RAC1 and CDKN1B proteins. Further comprehensive work is needed to support this finding especially involving in vivo work.

Keywords: stromal interaction molecule 1 (*STIM1)*, Dicer substrate short interfering RNA (*DsiRNA*), EBV-related NPC, miRNA

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