LINC00992 contributes to the oncogenic phenotypes in prostate cancer via targeting miR-3935 and augmenting GOLM1 expression

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Abstract

Background: Accumulating evidence has revealed the critical role of long non-coding RNAs (lncRNAs) in cellular processes during tumor progression. As documented in cancer-related literatures, LINC00992 expression is associated with cancer progression, whereas its function in tumors including prostate cancer has not been characterized yet.

Methods: Data from GEPIA database suggested LINC00992 expression in prostate cancer tissues. The expression levels of RNAs were monitored via qRT-PCR. Western blot evaluated the levels of proteins. The proliferation, apoptosis and migration of prostate cancer cells were assessed by CCK-8, EdU, TUNEL, Transwell and wound healing assays. Luciferase reporter, RNA pull down and RIP assays were applied to detect the interplays among LINC00992, miR-3935 and GOLM1.

Results: Elevated levels of LINC00992 and GOLM1 were detected in prostate cancer tissues and cells. LINC00992 exerted facilitating functions in prostate cancer cell proliferation and migration. Mechanically, LINC00992 interacted with and negatively regulated miR-3935 to elevate GOLM1 expression in prostate cancer cells. In addition, the in vitro suppressive effect of silenced LINC00992 on prostate cancer cell proliferation and migration was reversed by GOLM1 upregulation. Likewise, LINC00992 depletion restrained tumor growth in vivo was offset by enhanced GOLM1 expression.

Conclusions: LINC00992 competitively bound with miR-3935 to elevate GOLM1 expression and therefore facilitate the oncogenic phenotypes of prostate cancer cells, implying a potential LINC00992-targeted therapy for prostate cancer.

Keywords: LINC00992; miR-3935; GOLM1; prostate cancer