EFFECTS OF MODULATING KDM5B EXPRESSION IN PROSTATE CANCER CELLS

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Prostate cancer (PCa) remains one of the leading causes of death among men, with over 370,000 annual deaths worldwide [1]. It has become evident that epigenetics plays a significant role in the onset and progression of PCa, particularly histone methylation and its associated genes. One of these genes is *KDM5B*, a lysine-specific demethylase, which has been identified in recent years as an oncogene associated with many cancers [2]. In PCa, *KDM5B* is often overexpressed, which correlates with lower patient survival rates [3]. Therefore, lowering *KDM5B* expression in cancer cells could be a potential target for PCa treatment.

This study aimed to modulate *KDM5B* gene expression in PCa cells by transfection with small interfering RNA (siRNA) and to assess the induced potential changes in cell behavior. PC-3 and LNCaP cell lines were used for this study. The relative expression of *KDM5B* was measured using quantitative PCR, and commercial assays were used to assess cell viability, proliferation, invasiveness, and migration.

Our preliminary results demonstrated successful downregulation of *KDM5B* expression in both cell lines following siRNA transfection, with the statistically significant decrease observed in LNCaP cells ($P < 0.01$). Functional analyses revealed that *KDM5B* silencing reduced PC-3 cell viability and proliferation ($P = 0.0125$ and $P = 0.0190$, respectively), while no significant changes in apoptotic or necrotic cell populations were detected. Furthermore, decreased migratory capacity was observed in both PC-3 and LNCaP cells, suggesting a potential role of *KDM5B* in regulating prostate cancer cell motility.

In conclusion, our preliminary results indicate that siRNA-mediated downregulation of *KDM5B* expression in PCa cells reduces cell viability, proliferation, and migration, suggesting a potential role in PCa progression and its relevance for drug development.

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