

Uncover novel pharmacological approach in renal cell carcinoma

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INTRODUCTION

Renal cell carcinoma (RCC) represents the most common urological malignancy with an incidence around 1% of all cancer. RCC consists of heterogeneous histologic and molecular subtypes. The most common form of RCC is clear cell renal cell carcinoma (ccRCC), approximately 75-80%, and an invariant common pathway is pathway "PI3K/AKT/mTOR". Therapeutic strategies are largely resistant to conventional therapies such as chemotherapy and radiotherapy. Therapeutic have focused over the last particular therapies that target tumor signaling pathways and modulate the current strategy for the treatment of advanced and metastatic RCC. Indeed, patients with advanced/metastatic ccRCC are treated with combination therapies of mTOR inhibitors (mTORi) and tyrosine kinase inhibitors (TKI). Despite the evolution of therapies, an optimal panel of patients to use in first years after the initiation of systemic treatment will have disease progression. So, a better understanding of the molecular pathogenesis of RCC will help to choose and identify the best therapeutic approach to improve the patients' outcome.

In last years, several evidence highlighted the importance of mTOR and AKT/mTOR inhibition in cancer cells. This includes, mTOR inhibition was observed in RCC^{1,2} and it has been identified as a pathway of cancer cell. The integrally and biologically significance of AKT/mTOR pathway have been largely tested by genetic, through "Wortmannin effect". Some experimental approaches (PI3K) in the mouse model, in the treatment of cancer cells, have also identified an increase in the concentration of lipid droplets with metastatic membranes. These results could be interpreted as a potential event from lipotoxicity for the progression of malignancy. Our study found a substantial role for pharmacological inhibition of PI3K/mTOR pathway in the effect on metastatic and tumor maintenance. Our study found a substantial role for pharmacological inhibition of PI3K/mTOR pathway in the effect on metastatic and tumor maintenance. Our study found a substantial role for pharmacological inhibition of PI3K/mTOR pathway in the effect on metastatic and tumor maintenance.

RESULTS

Figure 1: Overview of sample collection, experimental and data analysis.

Figure 2: Single cell transcriptomic analysis of RCC cells.

Figure 3: Single cell transcriptomic analysis of RCC cells.

Figure 4: Pharmacological inhibition of PI3K/mTOR pathway.

CONCLUSIONS

- PI3K/mTOR inhibition in RCC cells induce normal lipid.
- AKT/mTOR inhibition impact on AKT/mTOR and PI3K/mTOR pathway.
- PI3K/mTOR inhibition induces a lipid droplet in RCC cells.
- PI3K/mTOR inhibition induces a higher lipid in RCC cells.
- PI3K/mTOR inhibition leads to be activated by the treatment with AKT/mTOR inhibitors.
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Role of Znf148 in squamous cell carcinomas

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ABSTRACT

High and low Znf148 expression in squamous cell carcinoma (SCC) is associated with different clinical outcomes. Znf148 is an transcription factor that regulates the expression of genes involved in cell growth, differentiation and survival. In this study, we investigate the role of Znf148 in squamous cell carcinoma (SCC) and its potential as a prognostic marker. We found that Znf148 expression is significantly correlated with tumor progression and survival. Znf148 expression is significantly correlated with tumor progression and survival. Znf148 expression is significantly correlated with tumor progression and survival.

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RESULTS

Figure 1: Role of the transcription factor ZNF148 in squamous cell carcinoma.

Figure 2: Analysis of ZNF148 expression in tumor cells.

Figure 3: Effect of ZNF148 knockdown on cell growth and survival.

Figure 4: Effect of ZNF148 knockdown on cell growth and survival.

CONCLUSIONS

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