



Nuclear M6a Reader YTHDC1-mediated CLU-IGF1R Axis Inhibits Lung Adenocarcinoma Progression by Targeting PI3K/AKT Signaling

L. Xia¹, L. Zhang¹, T. Wang¹, Y. Wang¹, T. Yang¹, G. Wu¹, Y. Liu¹, Y. Feng¹, H. Sun¹, H. Huang¹, Y. Zhao¹, H. Liu¹
¹Affiliatin Shanghai Pulmoanry Hospital Tongji University, School of Medicine, Shanghai/CN
Correspondence: xialang0207@tongji.edu.cn



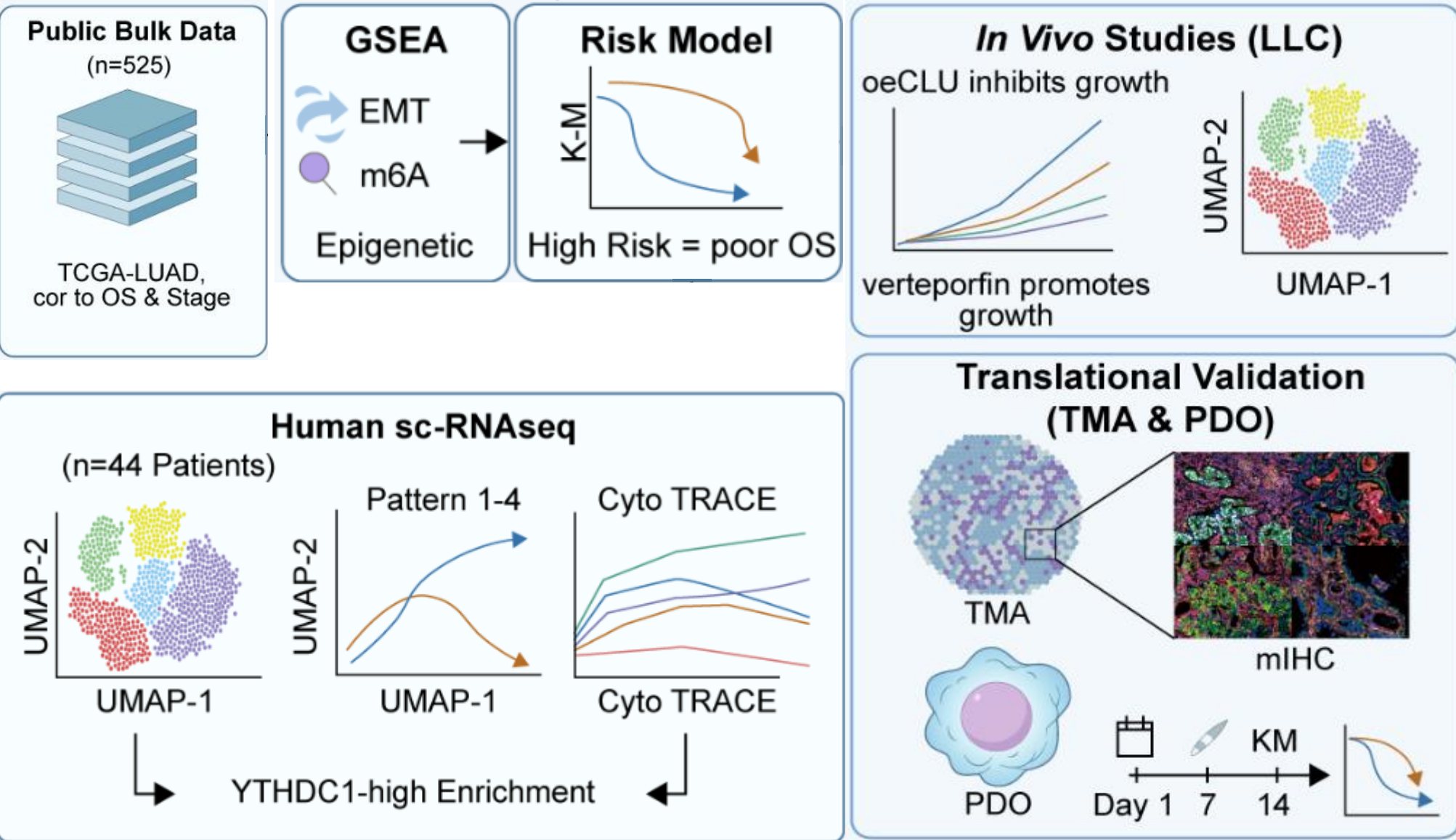
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INTRODUCTION

RNA N6-methyladenosine (m6A) modification and its regulatory factors precisely control gene expression and regulate tumorigenesis. This study aims to elucidate the role of the m6A reader protein YTHDC1 in suppressing the progression of lung adenocarcinoma and its underlying molecular mechanisms.

METHODS

Using in vitro and in vivo models to determine the inhibitory role of YTHDC1 in lung cancer progression. To identify the genuine YTHDC1-targeted RNAs, we performed RNA-seq, m6A-seq, RIP-seq, and single-cell sequencing techniques, followed by integrated data analysis and validation experiments.



DISCUSSION AND CONCLUSION

- We demonstrate that YTHDC1 plays a key tumor-suppressive role in lung cancer progression by inhibiting the activation of the PI3K/AKT signaling pathway through promoting the nuclear export and expression of CLU.
- Overall, our study shows that YTHDC1 suppresses lung cancer progression by inhibiting cell survival and EMT via CLU. We also propose the use of exogenous CLU supplementation as a potential therapeutic approach for lung cancer patients with low YTHDC1 expression.

REFERENCES & ACKNOWLEDGEMENTS

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RESULTS

