



EP03.15: Construction of a Phagocytosis Regulator-Related Risk Model in Lung Adenocarcinoma and Analysis of Single-Cell Molecular Characteristics

J. Deng¹, J. Xu¹, T. Luo², F. Yu^{1,2}, F. Qiu^{1,2}

¹Gaoxin Branch of The First Affiliated Hospital of Nanchang University, Nanchang/CN, ²The First Affiliated Hospital of Nanchang University, Nanchang/CN

Correspondence: djsx1012@126.com



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INTRODUCTION

Lung Adenocarcinoma (LUAD) is the most common lung cancer subtype. Abnormal phagocytosis is closely associated with LUAD progression, but the specific molecular mechanisms remain unclear. This study aimed to identify prognostic genes related to phagocytosis regulator, and provide new theoretical basis and potential targets for LUAD targeted therapy.

METHODS

LUAD-associated transcripts and phagocytosis regulator-related genes were retrieved from public databases. Prognostic genes were identified by integrating differential expression analysis and univariate Cox regression analysis, followed by the establishment of a risk prediction model. Subsequently, the immune and drug susceptibility features related to the risk score were analyzed. Single-cell sequencing data were further utilized to delineate the cellular landscape and characterize the expression patterns of prognostic genes.

DISCUSSION AND CONCLUSION

Our study identified 5 phagocytosis regulator-related prognostic genes, characterized their risk score in relation to immune features and drug sensitivity, and provided potential molecular targets for LUAD stratified management and mechanistic research. Our analysis is mainly based on database-derived data, the mechanistic investigation is not sufficiently in-depth, and further clinical trials are required to validate our findings.

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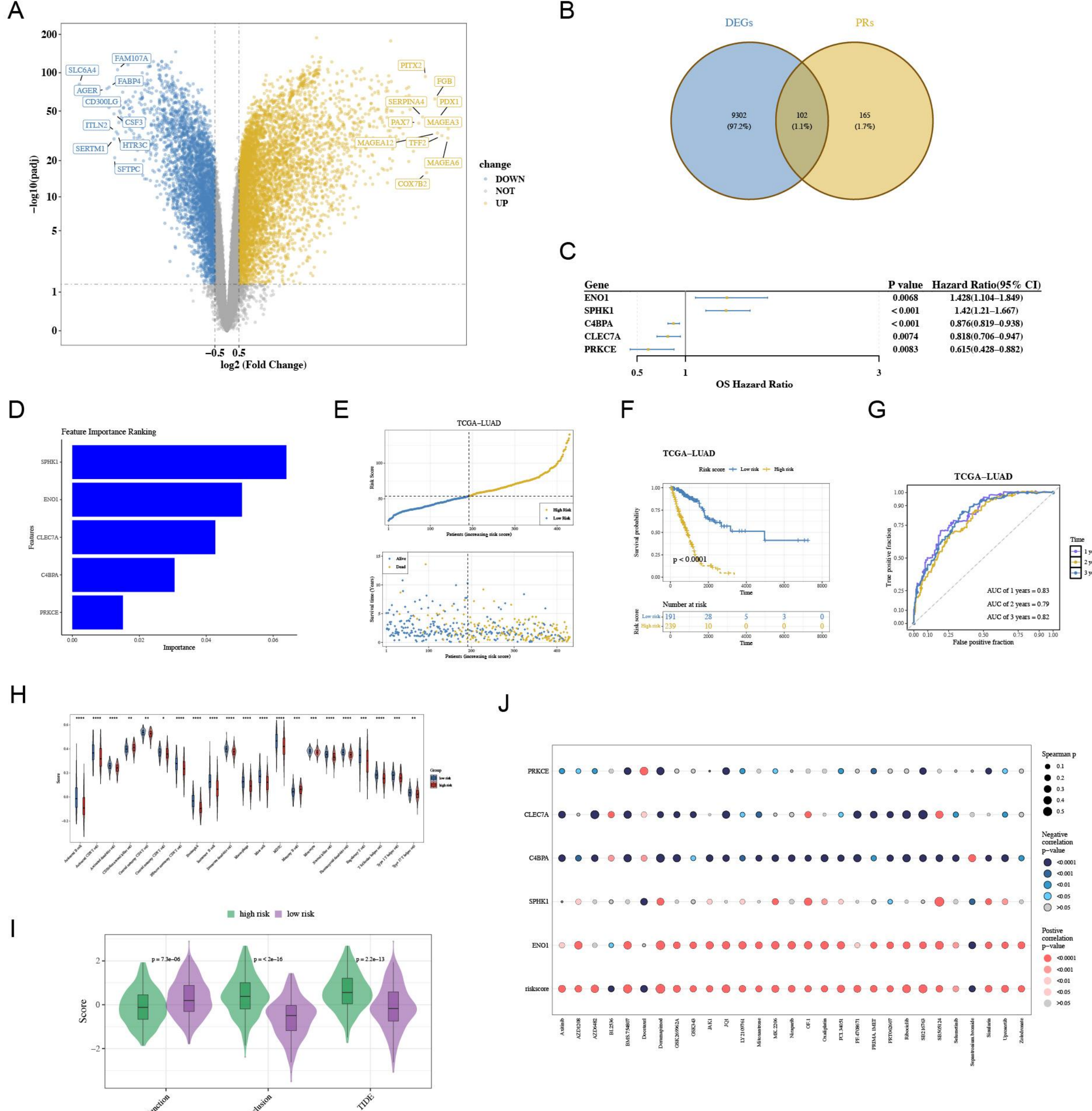
In accordance with the standards and guidance set forth by the Accreditation Council for Continuing Medical Education (ACCME), we hereby disclose that artificial intelligence (AI) tools were not utilized in the development of this educational material.

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RESULTS

The 102 genes were associated with phagocytosis regulator during LUAD tumorigenesis. Among them, ENO1 and SPHK1 were identified as risk factors, while C4BPA, CLEC7A, and PRKCE served as protective factors, collectively constituting the prognostic gene panel. The constructed risk model enabled robust stratification of LUAD samples, with the high-risk group exhibiting consistently poorer overall survival than the low_x0002_risk group. The two risk strata displayed distinct infiltration levels of 21 immune cells and varied immunotherapeutic responses, along with differential sensitivity to 102 chemotherapeutic agents (e.g., docetaxel).



At the single-cell level, 5 cell types showed significantly altered proportions in LUAD tissues compared with normal tissues, among which monocytes were recognized as key cells. Meanwhile, tumorigenesis was accompanied by enhanced intercellular communication. Prognostic genes exhibited dynamic expression patterns during monocyte differentiation; for instance, ENO1 showed an overall upward trend.

