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Summary

INTEGRATIVE PROTEOGENOMIC ANALYSIS REVEALS IMMUNE DYSREGULATION AND LACTYLATION-DEPENDENT METABOLIC REPROGRAMMING IN ESOPHAGEAL CANCER METASTASIS

Qiu-Xin Li

UEG Week Berlin 2025 Poster

Oesophagus

October 4, 2025

Multi-omics analysis of 36 metastatic esophageal squamous cell carcinoma patients reveals lysine lactylation as a key driver of metastasis through metabolic pathway modulation and identifies PGAM1 K251 lactylation as promoting metastasis via enhanced actin gamma 1 interaction.

KEY POINTS

- Integration of genomic, transcriptomic, proteomic, and lactylome data from 99 samples across 36 metastatic ESCC patients established comprehensive molecular landscapes of metastatic disease
- Lysine lactylation was extensively involved in ESCC metastasis primarily through metabolic pathway modulation and exhibited comprehensive interactions with genomic and transcriptomic elements through cis- and trans-regulatory mechanisms
- PGAM1 K251 lactylation promoted metastasis both in vitro and in vivo by enhancing molecular interaction with actin gamma 1, thereby affecting actin kinetics and cell motility
- Intercellular adhesion molecule 1 was identified as a critical regulator of invasion and migration in prognosis-relevant immunity-driving waves in the metastatic cascade
- Findings were validated through immunohistochemical assays on clinical samples and in vitro and in vivo experiments

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