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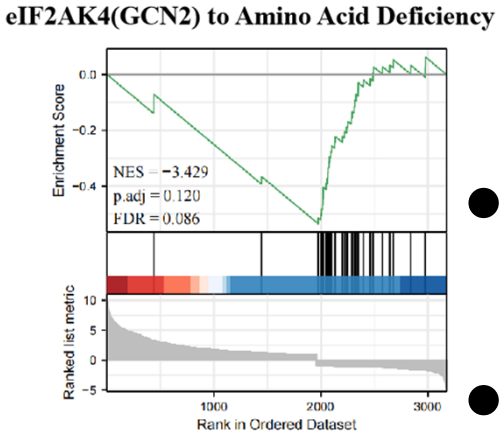
UPF1 increases amino acid levels and promotes cell proliferation in lung adenocarcinoma via the eIF2α-ATF4 axis

Key words: UPF1, ATF4, Amino acid, Lung adenocarcinoma

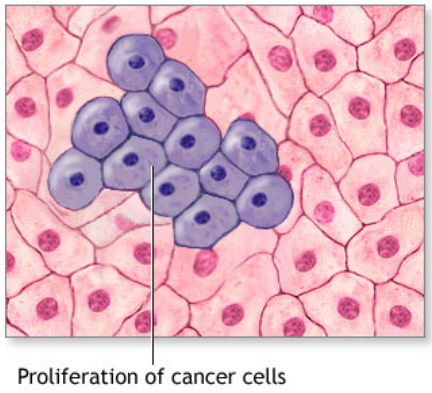
Research Summary

This review mainly focused on the core components of nonsense-mediated mRNA decay(NMD), UPF1 proteins, and summarized the key roles it played in the following aspects:



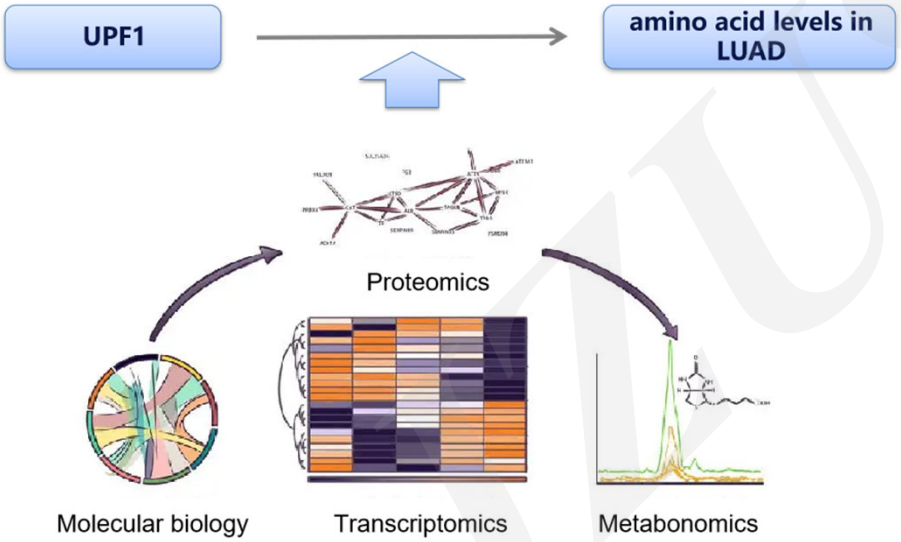


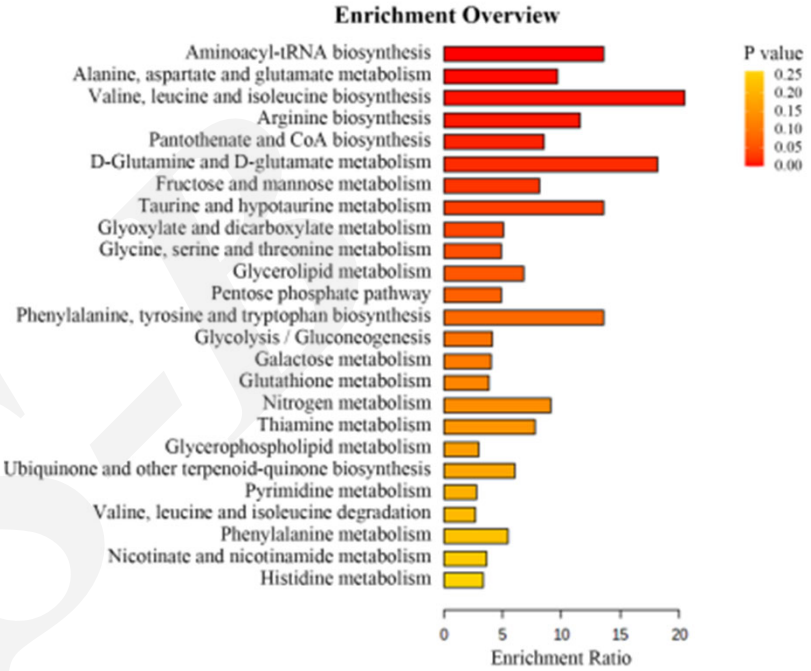
- **Regulation of phosphorylation of eIF2A-Ser51 and ATF4**
- **Amino acid levels in LUAD cells**
- **LUAD cells proliferation in vitro and vivo**
- **UPF1 and ATF4 were significantly correlated with overall survival (OS) in LUAD patients**



Innovation points

- **Introduction of UPF1 to increase amino acid levels depending on integrated analysis of multiple omics in LUAD for the first time.**





- **Introduction of UPF1 to regulate phosphorylation of eIF2A-Ser51 and ATF4 in LUAD.**

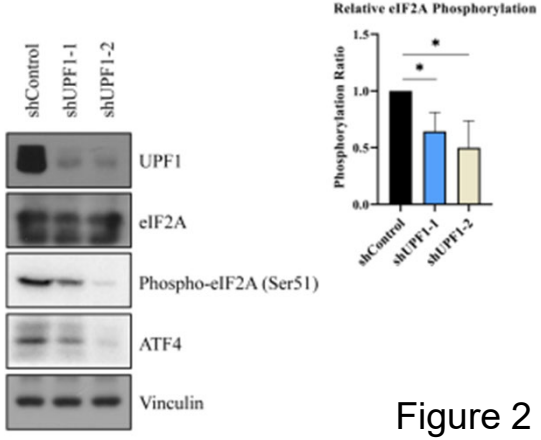


Figure 2

Innovation points

A series of comprehensive figures were generated to introduce the function of UPF1 to increase amino acid levels in LUAD

Figure 1 | UPF1 knockdown in LUAD cells altered metabolomics profiling based on GC-MS.

Figure 2 | UPF1 knockdown inhibited Ser51 phosphorylation of eIF2A and ATF4 expression.

Figure 3 | UPF1 knockdown inhibited LUAD cell proliferation via ATF4 in vitro and in vivo.

Figure 4 | UPF1 enhanced amino acid levels depending upon ATF4 in LUAD cells.

Figure 5 | Expression of UPF1 in LUAD tissues and its relationship with patient prognosis.

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