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BCAT1 promotes lung adenocarcinoma progression through enhanced mitochondrial function and NF-κB pathway activation

Key words: BCAT1, LUAD, mitochondrial function, ROS, NF-κB pathway

Research Summary

This study mainly focused on the function and molecular mechanism of BCAT1 in LUAD development in the following aspects:

- **BCAT1 expression levels in tumor and surrounding normal lung tissues of LUAD patients**
- **Effect of BCAT1 on LUAD development *in vitro* and *in vivo***
- **BCAAs consumption, mitochondrial respiration, and ROS content**
- **RNA sequencing and NF- κ B pathway**
- **NF- κ B pathway inhibitor (PDTC) treatment on LUAD cells**

Innovation points

In LUAD cells:

- BCAT enhanced BCAAs consumption and mitochondrial respiration ability
- BCAT1 decreased ROS content
- BCAT1 decreased transcription level of *NFKBIB* and activated NF- κ B pathway
- NF- κ B pathway inhibitors were potential therapeutic drugs for LUAD patients.

