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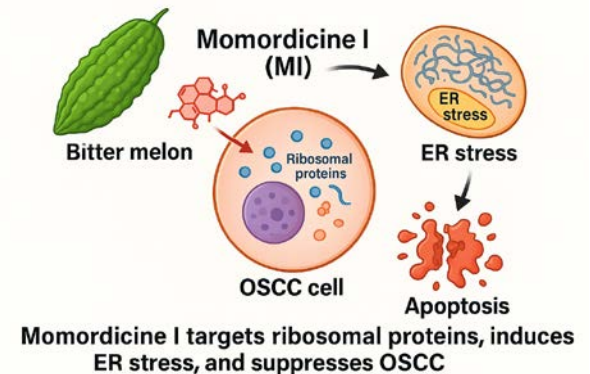
Momordicine I induces ER stress and inhibits OSCC by targeting ribosomal proteins

Key words: Oral squamous cell carcinoma (OSCC); Momordicine I (MI); Endoplasmic reticulum (ER) stress; Ribosomal protein

Research Summary

This study mainly focused on the anticancer effects of Momordicine I (MI) from bitter melon extract, and summarized the key roles it played in the following aspects:

- Momordicine I (MI) from bitter melon strongly inhibited OSCC proliferation, migration, and tumor growth.
- Transcriptomic analysis revealed MI triggered unfolded protein response (UPR) and ER stress, leading to apoptosis in OSCC cells.
- Cellular thermal shift assay and mass spectrometry (CETSA-MS) analysis and molecular docking identified multiple ribosomal proteins as direct MI targets, uncovering a new mechanism of MI in OSCC inhibition.



Innovation points

- This study revealed novel mechanism of natural triterpenoids in OSCC inhibition.
- RNA-seq showed that MI induced UPR and ER stress. CETSA-MS & molecular docking identified ribosomal proteins (RPL7, RPL11, RPL12, RPL18, RPL30, RPL38, RPS13, RPS25) as direct MI targets. Targeting ribosomes disrupts protein folding, causing ER stress and apoptosis.

