# SMALL-MOLECULE INHIBITOR OF SLC25A1 AS ANTI-CANCER AGENT

GMU-16-019

## ATTACKING CANCER AT ITS SOURCE: POWERING A NEW ERA OF RESISTANCE-FREE TREATMENT

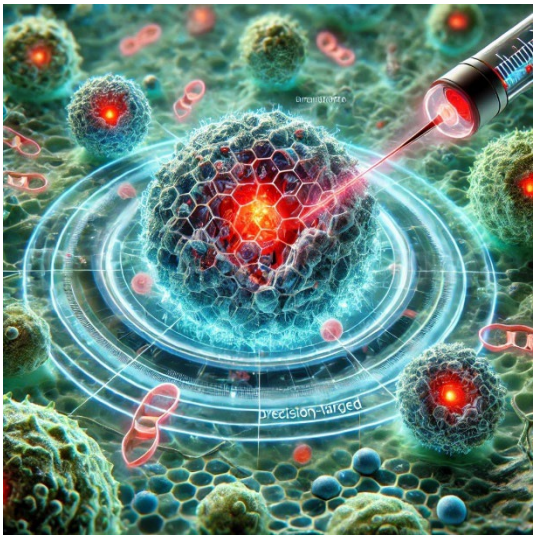
**Mason's cutting-edge research has identified the mitochondrial citrate carrier, SLC25A1, as a key driver of therapy resistance and stemness in non-small cell lung cancer (NSCLC). This breakthrough discovery reveals how targeting SLC25A1 can suppress cancer stem cells (CSCs), which are responsible for therapy resistance, tumor relapse, and metastasis. A novel inhibitor of SLC25A1 has demonstrated the potential to restore sensitivity to conventional treatments like cisplatin and EGFR inhibitors.**

### Key Features

- **Targeted Cancer Stem Cell Therapy:** Specifically inhibits SLC25A1 to attack the cancer stem cells responsible for therapy resistance and tumor relapse
- **Enhanced Treatment Response:** Combines with existing therapies such as cisplatin or EGFR inhibitors to significantly improve treatment outcomes
- **Novel SLC25A1 Inhibitor:** Newly developed inhibitor disrupts cancer cell metabolism, particularly mitochondrial respiration, which is vital for cancer stem cell survival
- **Broad Efficacy:** A Demonstrated success in preclinical models of drug-resistant lung cancer, showing promise for overcoming treatment resistance in patients

### Applications

- **Advanced non-small cell lung cancer (NSCLC) therapy**
- **Potential expansion to other cancers exhibiting SLC25A1-mediated therapy resistance**

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