

PTRF

- EPIC-1042 as a potent PTRF/Cavin1-caveolin-1 interaction inhibitor to induce PARP1 autophagic degradation and suppress temozolomide efflux for glioblastoma
- Molecular and clinical characterization of PTRF in glioma via 1,022 samples
- PTRF/Cavin-1 enhances chemo-resistance and promotes temozolomide efflux through extracellular vesicles in glioblastoma
- Neuronal STAT3/HIF-1 α /PTRF axis-mediated bioenergetic disturbance exacerbates cerebral ischemia-reperfusion injury via PLA2G4A
- Blood exosomes-based targeted delivery of cPLA2 siRNA and metformin to modulate glioblastoma energy metabolism for tailoring personalized therapy
- PTRF/Cavin-1 as a Novel RNA-Binding Protein Expedites the NF-kappaB/PD-L1 Axis by Stabilizing lncRNA NEAT1, Contributing to Tumorigenesis and Immune Evasion in Glioblastoma
- PTRF/cavin-1 remodels phospholipid metabolism to promote tumor proliferation and suppress immune responses in glioblastoma by stabilizing cPLA2
- PTRF/CAVIN1, regulated by SHC1 through the EGFR pathway, is found in urine exosomes as a potential biomarker of ccRCC

PTRF in Glioma

PTRF in glioma

Polymerase I and transcript release factor (PTRF) plays a pivotal role in regulating cellular senescence, glucose intolerance, lipid metabolism, and mitochondrial bioenergetics. The STAT3/HIF-1 α /PTRF-axis in neurons, aggravating cerebral I/R injury by regulating the activity and stability of PLA2G4A, might be a novel therapeutic target for ischemic stroke ¹⁾

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Jin W, Zhao J, Yang E, Wang Y, Wang Q, Wu Y, Tong F, Tan Y, Zhou J, Kang C. Neuronal STAT3/HIF-1 α /PTRF axis-mediated bioenergetic disturbance exacerbates cerebral ischemia-reperfusion injury via PLA2G4A. *Theranostics*. 2022 Apr 4;12(7):3196-3216. doi: 10.7150/thno.71029. PMID: 35547748; PMCID: PMC9065197.

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