

ACSS2(Phospho-Ser659) Antibody

Catalog No: #58003

Package Size: #58003 100ul

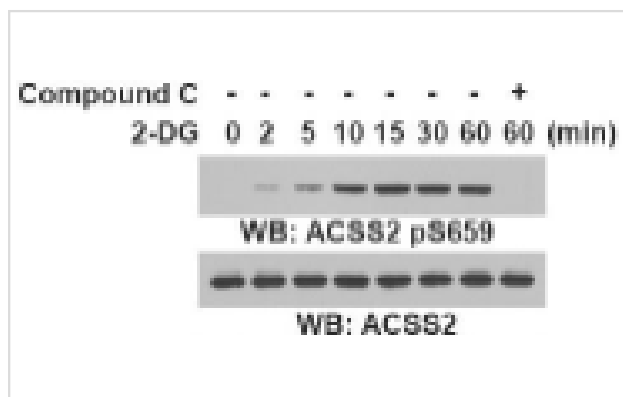
Description

Product Name	ACSS2(Phospho-Ser659) Antibody
Host Species	Rabbit
Clonality	Polyclonal
Isotype	IgG
Applications	WB
Species Reactivity	Hu Ms
Immunogen Description	The antiserum was produced against synthesized phosphopeptide derived from Human ACSS2 around the phosphorylation site of serine 659.
Conjugates	Unconjugated
Calculated MW	78kDa
Storage	Store at -20°C/1 year

Application Details

WB dilution: 1:500-1:2000

Images



ACSS2 S659 Phosphorylation in U87 Cells

Background

Overcoming metabolic stress is a critical step in tumor growth. Acetyl coenzyme A (acetyl-CoA) generated from glucose and acetate uptake is important for histone acetylation and gene expression. However, how acetyl-CoA is produced under nutritional stress is unclear. We demonstrate here that glucose deprivation results in AMP-activated protein kinase (AMPK)-mediated acetyl-CoA synthetase 2 (ACSS2) phosphorylation at S659, which exposed the nuclear localization signal of ACSS2 for importin α binding and nuclear translocation. In the nucleus, ACSS2 binds to transcription factor EB and translocates to lysosomal and autophagy gene promoter regions, where ACSS2 incorporates acetate generated from histone acetylation turnover to locally produce acetyl-CoA for histone H3 acetylation in these regions and promote lysosomal biogenesis, autophagy, cell survival, and brain tumorigenesis. In addition, ACSS2 S659 phosphorylation positively correlates with AMPK activity in glioma specimens and grades of glioma malignancy. These results underscore the significance of nuclear ACSS2-mediated histone acetylation in maintaining cell homeostasis and tumor development.

Published Papers

el at., Targeting ACSS2 activity suspends the formation ofchemoresistance through suppressed histone H3acetylation in human breast cancer, ,
(2024)
PMID:

Note: This product is for in vitro research use only and is not intended for use in humans or animals.