SANTA CRUZ BIOTECHNOLOGY, INC.

# SUCLA2 (G-17): sc-79114

SANTA CRUZ BIOTECHNOLOGY

*The Power to Question*

## BACKGROUND

SUCLA2 (succinate-CoA ligase, ADP-forming, β subunit), also known as A-β, SCS-βA or renal carcinoma antigen NY-REN-39, is a 463 amino acid mitochondrial matrix enzyme that belongs to the succinate/malate CoA ligase β subunit family. Widely expressed, SUCLA2 dimerizes with the SCS α subunit to form SCS-A, an essential component of the tricarboxylic acid cycle. Defects in SUCLA2 may be involved in a group of autosomal recessive disorders known as mitochondrial DNA depletion syndromes (MDSs) that are characterized by a decrease in mitochondrial DNA copy numbers in affected tissues. Progressive external ophthalmoplegia (PEO), ataxia-neuropathy and mitochondrial neurogastrointestinal encephalomyopathy (MNGIE) may also be associated with mutations in SUCLA2. Two isoforms of SUCLA2 exists due to alternative splicing events.

## REFERENCES


1. Furuyama, K., et al. 2000. Interaction between succinyl CoA synthetase and the heme-biosynthetic enzyme ALAS-E is disrupted in sideroblastic anemia. J. Clin. Invest. 105: 757-764.
2. Elpeleg, O., et al. 2005. Deficiency of the ADP-forming succinyl-CoA synthase activity is associated with encephalomyopathy and mitochondrial DNA depletion. Am. J. Hum. Genet. 76: 1081-1086.
3. Ostergaard, E., et al. 2007. Mitochondrial encephalomyopathy with elevated methylmalonic acid is caused by SUCLA2 mutations. Brain 130: 853-861.
4. Carrozzo, R., et al. 2007. SUCLA2 mutations are associated with mild methylmalonic aciduria, Leigh-like encephalomyopathy, dystonia and deafness. Brain 130: 862-874.
5. Bourdon, A., et al. 2007. Mutation of RRM2B, encoding p53-controlled ribonucleotide reductase (p53R2), causes severe mitochondrial DNA depletion. Nat. Genet. 39: 776-780.
6. Copeland, W.C. 2008. Inherited mitochondrial diseases of DNA replication. Annu. Rev. Med. 59: 131-146.


## CHROMOSOMAL LOCATION

Genetic locus: SUCLA2 (human) mapping to 13q14.2; Sucla2 (mouse) mapping to 14 D3.

## SOURCE

SUCLA2 (G-17) is an affinity purified rabbit polyclonal antibody raised against a peptide mapping near the N-terminus of SUCLA2 of human origin.

## PRODUCT

Each vial contains 100 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-79114 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

## STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

## APPLICATIONS

SUCLA2 (G-17) is recommended for detection of SUCLA2 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

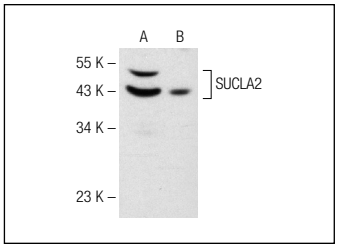
SUCLA2 (G-17) is also recommended for detection of SUCLA2 in additional species, including equine, canine, bovine and porcine.

Suitable for use as control antibody for SUCLA2 siRNA (h): sc-76598, SUCLA2 siRNA (m): sc-76599, SUCLA2 shRNA Plasmid (h): sc-76598-SH, SUCLA2 shRNA Plasmid (m): sc-76599-SH, SUCLA2 shRNA (h) Lentiviral Particles: sc-76598-V and SUCLA2 shRNA (m) Lentiviral Particles: sc-76599-V.

Molecular Weight of SUCLA2: 50 kDa.

Positive Controls: Hep G2 cell lysate: sc-2227, MOLT-4 cell lysate: sc-2233 or ES-2 cell lysate: sc-24674.

## DATA




SUCLA2 (G-17): sc-79114. Western blot analysis of SUCLA2 expression in Hep G2 (**A**) and MOLT-4 (**B**) whole cell lysates.

## RESEARCH USE

For research use only, not for use in diagnostic procedures.

## PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.

MONOS Satisfaction Guaranteed

Try **SUCLA2 (A-9): sc-374107** or **SUCLA2 (F-2): sc-373959**, our highly recommended monoclonal alternatives to SUCLA2 (G-17).