SANTA CRUZ BIOTECHNOLOGY, INC.

IscU1/2 (FL-142): sc-28860

SANTA CRUZ BIOTECHNOLOGY
The Power to Question

BACKGROUND

Iron-sulfur (Fe-S) clusters are cofactors that are essential for a wide variety of processes, including facilitation of electron transfer processes in oxidative phosphorylation, catalysis of enzymatic reactions in aconitase and dehydratases and maintenance of structural integrity in the DNA repair enzyme endonuclease III. In bacteria and eukaryotes, several new genes are implicated in the biogenesis of Fe-S cluster-containing proteins. IscU1 and IscU2, homologs to bacterial IscU and NifU, are iron cluster-assembly proteins. Deletion of either IscU1 or IscU2 results in increased accumulation of iron within the mitochondria, loss of activity of the [4Fe-4S] aconitase enzyme and suppression of oxidative damage in cells lacking cytosolic copper/zinc superoxide dismutase. IscU1 and IscU2 are regulated by the iron status of the cell and localize primarily in the mitochondria. In human cells, alternative splicing of IscU pre-mRNA results in synthesis of these two proteins, which differ at the N-terminus and localize either to the cytosol (IscU1) or the mitochondria (IscU2). IscU proteins interact with IscS, a cysteine desulfurase, to sequester inorganic sulfur for Fe-S cluster assembly. IscU-IscS protein complex localizes in both mitochondria and cytosol, implying that Fe-S cluster assembly takes place in multiple subcellular compartments in mammalian cells.

CHROMOSOMAL LOCATION

Genetic locus: ISCU (human) mapping to 12q23.3; Iscu (mouse) mapping to 5 F.

SOURCE

IscU1/2 (FL-142) is a rabbit polyclonal antibody raised against amino acids 1-142 representing full length IscU1 of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

IscU1/2 (FL-142) is recommended for detection of IscU1 and IscU2 of human origin and IscU of mouse and rat 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), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for IscU1/2 siRNA (h): sc-270108, IscU siRNA (m): sc-40712, IscU1/2 shRNA Plasmid (h): sc-270108-SH, IscU shRNA Plasmid (m): sc-40712-SH, IscU1/2 shRNA (h) Lentiviral Particles: sc-270108-V and IscU shRNA (m) Lentiviral Particles: sc-40712-V.

Molecular Weight of IscU1: 15 kDa.

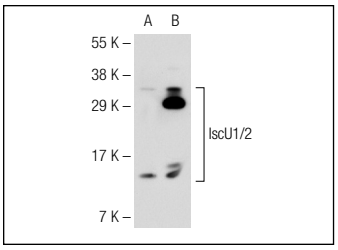
Molecular Weight of IscU2: 18 kDa.

Positive Controls: HeLa whole cell lysate: sc-2200, IscU1/2 (h): 293T Lysate: sc-174306 or IscU1/2 (m): 293T Lysate: sc-121116.

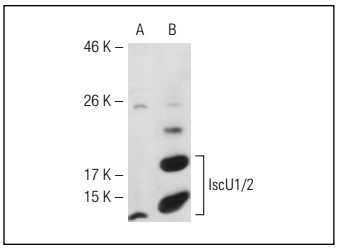
STORAGE

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

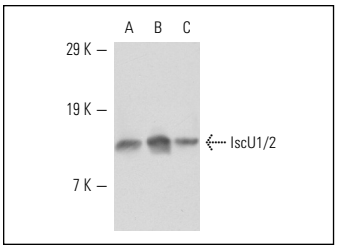
DATA



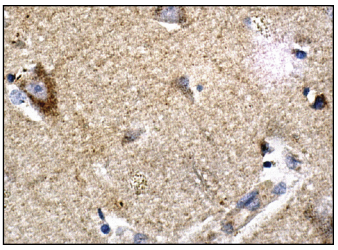
IscU1/2 (FL-142): sc-28860. Western blot analysis of IscU1/2 expression in non-transfected: sc-117752 (**A**) and human IscU1/2 transfected: sc-174306 (**B**) 293T whole cell lysates.



IscU1/2 (FL-142): sc-28860. Western blot analysis of IscU1/2 expression in non-transfected: sc-117752 (**A**) and mouse IscU1/2 transfected: sc-121116 (**B**) 293T whole cell lysates.



IscU1/2 (FL-142): sc-28860. Western blot analysis of IscU1 and IscU2 expression in HeLa (**A**), SW480 (**B**) and HCT 116 (**C**) whole cell lysates.



IscU1/2 (FL-142): sc-28860. Immunoperoxidase staining of formalin fixed, paraffin-embedded human brain tissue showing cytoplasmic staining of neuronal cells.

SELECT PRODUCT CITATIONS

1. Kollberg, G., et al. 2009. Clinical manifestation and a new ISCU mutation in iron-sulphur cluster deficiency myopathy. Brain 132: 2170-2179.
2. Nordin, A., et al. 2011. Tissue-specific splicing of ISCU results in a skeletal muscle phenotype in myopathy with lactic acidosis, while complete loss of ISCU results in early embryonic death in mice. Hum. Genet. 129: 371-378.
3. Kollberg, G., et al. 2011. Transient restoration of succinate dehydrogenase activity after rhabdomyolysis in iron-sulphur cluster deficiency myopathy. Neuromuscul. Disord. 21: 115-120.

RESEARCH USE

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