

NDUFS1 (G-6): sc-271387

BACKGROUND

The multisubunit NADH:ubiquinone oxidoreductase (complex I) is the first enzyme complex in the electron transport chain of mitochondria. Through use of chaotropic agents, complex I can be separated into three different fractions: a flavoprotein fraction, an iron-sulfur protein (IP) fraction, and a hydrophobic protein (HP) fraction. The IP fraction contains NDUFS1-7. NDUFS1, a 75 kD protein, is the largest subunit of complex I, and is thought to be the first of the Fe-S proteins to accept electrons from an NADH-flavoprotein reductase within the complex. NDUFS1 may even form part of the active site crevice where NADH is oxidized. NDUFS1 is also a critical caspase substrate in mitochondria, and caspase cleavage of NDUFS1 is required for several mitochondrial changes associated with apoptosis.

REFERENCES

1. Chow, W., et al. 1991. Determination of the cDNA sequence for the human mitochondrial 75 kDa FeS protein of NADH-coenzyme Q reductase. *Eur. J. Biochem.* 201: 547-550.
2. Duncan, A.M., et al. 1992. Localization of the human 75 kDa FeS protein of NADH-coenzyme Q reductase gene (NDUFS1) to 2q33-q34. *Cytogenet. Cell Genet.* 60: 212-213.
3. Bénit, P., et al. 2001. Large-scale deletion and point mutations of the nuclear NDUFV1 and NDUFS1 genes in mitochondrial complex I deficiency. *Am. J. Hum. Genet.* 68: 1344-1352.
4. Smeitink, J.A., et al. 2004. Cell biological consequences of mitochondrial NADH: ubiquinone oxidoreductase deficiency. *Curr. Neurovasc. Res.* 1: 29-40.
5. Karahan, O.I., et al. 2005. Ultrasound evaluation of peritoneal catheter tunnel in catheter related infections in CAPD. *Int. Urol. Nephrol.* 37: 363-366.
6. Martin, M.A., et al. 2005. Leigh syndrome associated with mitochondrial complex I deficiency due to a novel mutation in the NDUFS1 gene. *Arch. Neurol.* 62: 659-661.

CHROMOSOMAL LOCATION

Genetic locus: NDUFS1 (human) mapping to 2q33.3; Ndufs1 (mouse) mapping to 1 C2.

SOURCE

NDUFS1 (G-6) is a mouse monoclonal antibody raised against amino acids 428-727 mapping at the C-terminus of NDUFS1 of human origin.

PRODUCT

Each vial contains 200 µg IgG₁ kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

STORAGE

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

APPLICATIONS

NDUFS1 (G-6) is recommended for detection of NDUFS1 of mouse, rat and human origin by Western Blotting (starting dilution 1:100, 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).

Suitable for use as control antibody for NDUFS1 siRNA (h): sc-61164, NDUFS1 siRNA (m): sc-61165, NDUFS1 shRNA Plasmid (h): sc-61164-SH, NDUFS1 shRNA Plasmid (m): sc-61165-SH, NDUFS1 shRNA (h) Lentiviral Particles: sc-61164-V and NDUFS1 shRNA (m) Lentiviral Particles: sc-61165-V.

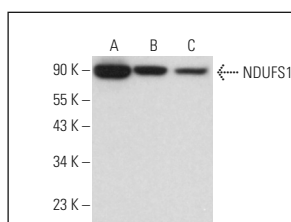
Molecular Weight of NDUFS1: 75 kDa.

Positive Controls: Raji whole cell lysate: sc-364236, NAMALWA cell lysate: sc-2234 or Hep G2 cell lysate: sc-2227.

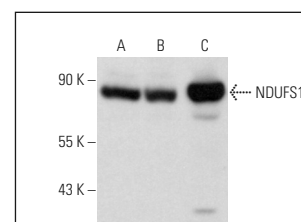
RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended: 1) Western Blotting: use m-IgGκ BP-HRP: sc-516102 or m-IgGκ BP-HRP (Cruz Marker): sc-516102-CM (dilution range: 1:1000-1:10000), Cruz Marker™ Molecular Weight Standards: sc-2035, UltraCruz® Blocking Reagent: sc-516214 and Western Blotting Luminol Reagent: sc-2048. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml). 3) Immunofluorescence: use m-IgGκ BP-FITC: sc-516140 or m-IgGκ BP-PE: sc-516141 (dilution range: 1:50-1:200) with UltraCruz® Mounting Medium: sc-24941 or UltraCruz® Hard-set Mounting Medium: sc-359850.

DATA



NDUFS1 (G-6): sc-271387. Western blot analysis of NDUFS1 expression in NAMALWA (A), c4 (B) and WEHI-231 (C) whole cell lysates.



NDUFS1 (G-6): sc-271387. Western blot analysis of NDUFS1 expression in Raji (A) and Hep G2 (B) whole cell lysates and rat heart tissue extract (C).

SELECT PRODUCT CITATIONS

1. Elouej, S., et al. 2020. Loss of MTX2 causes mandibuloacral dysplasia and links mitochondrial dysfunction to altered nuclear morphology. *Nat. Commun.* 11: 4589.
2. Sato, T., et al. 2022. Enhanced glucose metabolism through activation of HIF-1α covers the energy demand in a rat embryonic heart primordium after heartbeat initiation. *Sci. Rep.* 12: 74.

RESEARCH USE

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