

# ATP5B (10): sc-135903

## BACKGROUND

Mitochondrial ATP synthase is composed of two multi-subunit complexes that utilize an inner membrane electrochemical gradient to catalyze the synthesis of ATP during oxidative phosphorylation. The two multi-subunit complexes are designated  $F_1$  and  $F_0$ , the former of which comprises the soluble catalytic core and the latter of which comprises the membrane-spanning proton channel of ATP synthase.  $F_1$  consists of five distinct subunits, designated ATP5A, ATP5B, ATP5C1, ATP5D and ATP5E, while  $F_0$  consists of ten subunits, designated ATP5H, ATP5G1, ATP5I, ATP5G2, ATP5J2, ATP5J, ATP5G3, ATP5S, ATP5F1 and ATP5L. ATP5B, also designated ATPMB, ATPSB or mitochondrial ATP synthetase,  $\beta$  subunit, is a 529 amino acid protein that localizes to the mitochondrial membrane and exists as a subunit of the  $F_0$  complex. ATP5B is encoded by a nuclear gene and assembled with the other subunits encoded by both mitochondrial and nuclear genes. The ATP5B gene is activated by members of the Ets family of transcription factors, suggesting that Ets transcription factors are involved in the enhanced expression of the ATP5B gene in highly proliferating cells and in the coordinate transcription of nuclear genes for mitochondrial proteins. ATP5B mRNA levels vary among species through transcriptional control with high expression levels in heart, lower levels in skeletal muscle and the lowest levels in liver and kidney.

## REFERENCES

- Ohta, S., et al. 1986. Human  $F_1$ -ATPase: molecular cloning of cDNA for the  $\beta$  subunit. *J. Biochem.* 99: 135-141.
- Neckelmann, N., et al. 1989. The human ATP synthase  $\beta$  subunit gene: sequence analysis, chromosome assignment, and differential expression. *Genomics* 5: 829-843.
- Rouslin, W. 1991. Regulation of the mitochondrial ATPase *in situ* in cardiac muscle: role of the inhibitor subunit. *J. Bioenerg. Biomembr.* 23: 873-888.
- LaNaue, K.F., et al. 1992. Kinetic studies of ATP synthase: the case for the positional change mechanism. *J. Bioenerg. Biomembr.* 24: 499-506.
- Villena, J.A., et al. 1994. Ets transcription factors regulate expression of the gene for the human mitochondrial ATP synthase  $\beta$ -subunit. *J. Biol. Chem.* 269: 32649-32654.
- Elston, T., et al. 1998. Energy transduction in ATP synthase. *Nature* 391: 510-513.
- Wang, H., et al. 1998. Energy transduction in the  $F_1$  motor of ATP synthase. *Nature* 396: 279-282.

## CHROMOSOMAL LOCATION

Genetic locus: ATP5B (human) mapping to 12q13.3; Atp5b (mouse) mapping to 10 D3.

## SOURCE

ATP5B (10) is a mouse monoclonal antibody raised against amino acids 428-539 of  $F_1$ -ATPase of human origin.

## RESEARCH USE

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

## PRODUCT

Each vial contains 50  $\mu$ g IgG<sub>1</sub> in 0.5 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

## APPLICATIONS

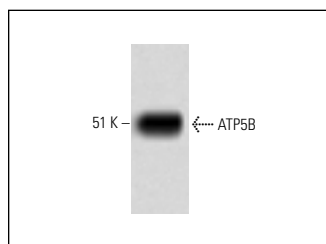
ATP5B (10) is recommended for detection of ATP5B of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2  $\mu$ g per 100-500  $\mu$ g of total protein (1 ml of cell lysate)] and immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500).

Suitable for use as control antibody for ATP5B siRNA (h): sc-40565, ATP5B siRNA (m): sc-40566, ATP5B shRNA Plasmid (h): sc-40565-SH, ATP5B shRNA Plasmid (m): sc-40566-SH, ATP5B shRNA (h) Lentiviral Particles: sc-40565-V and ATP5B shRNA (m) Lentiviral Particles: sc-40566-V.

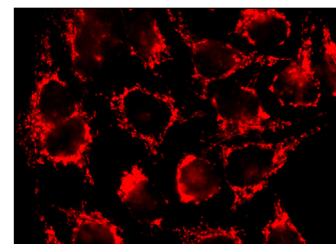
Molecular Weight of ATP5B: 51 kDa.

Positive Controls: Jurkat whole cell lysate: sc-2204, Hep G2 cell lysate: sc-2227 or DU 145 cell lysate: sc-2268.

## DATA



ATP5B (10): sc-135903. Western blot analysis of ATP5B expression in Jurkat whole cell lysate.



ATP5B (10): sc-135903. Immunofluorescence staining of methanol-fixed HeLa cells showing cytoplasmic localization.

## SELECT PRODUCT CITATIONS

- Zeng, Z., et al. 2015. Cyclic mechanical stretch promotes energy metabolism in osteoblast-like cells through an mTOR signaling-associated mechanism. *Int. J. Mol. Med.* 36: 947-956.
- Agrawal, S., et al. 2019. Novel proteomic changes in brain mitochondria provide insights into mitochondrial dysfunction in mouse models of Huntington's disease. *Mitochondrion* 47: 318-329.

## STORAGE

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

## PROTOCOLS

See our web site at [www.scbt.com](http://www.scbt.com) for detailed protocols and support products.