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ATP synthase subunit beta monoclonal antibody

CATALOG #:	A21351
COMPONENTS:	100 µg monoclonal antibody
APPLICATIONS:	Western blotting and Immunocytochemistry
CLONE ID OF MONOCLONAL ANTIBODY (mAb):	3D5AB1
SPECIES CROSS-REACTIVITY:	human, bovine, mouse, monkey, rat, and *C. elegans*
HOST SPECIES AND ISOTYPE:	Mouse IgG1, k
IMMUNOGEN:	Human Heart Mitochondria
CONCENTRATION:	1 mg/mL in Hepes-Buffered Saline (HBS) with 0.02% azide as a preservative.
SUGGESTED WORKING CONCENTRATION:	0.5 µg/mL for Western blotting, 1-2 µg/mL for Immunocytochemistry
mAb PURITY:	Near homogeneity as judged by SDS-PAGE. The antibody was produced *in vitro* using hybridomas grown in serum-free medium, and then purified by biochemical fractionation.
STORAGE CONDITIONS:	Store at 4°C. Do not freeze.
COUNTRY OF ORIGIN:	USA

BACKGROUND:

The epitope recognized by the anti-complex V-beta subunit mAb 3D5AB1 is in the region containing the active site of the β-subunit and is centered approximately at amino acid residue 83. The complete amino acid sequence of the epitope is not known. See Chi, S.L., Wahl, M.L., Kenan, D.J., Johnson, C.E., Marusich, M.F., Capaldi, R.A., and Pizzo, S.V. (2007). Angiostatin-like activity of a monoclonal antibody to the catalytic subunit of F_1F_0 ATP synthase. Cancer Research, 67, 4716-4724.

Complex V, also called F_1F_0ATPase or ATP synthase, is responsible for ATP production in oxidative phosphorylation and can work in reverse as a proton pumping ATPase. The enzyme was thought to be localized exclusively to mitochondria. However, it has recently been identified on the plasma membrane of several cell types including hepatocytes where it functions as the HDL receptor, on endothelial cells where it may act as the angiostatin receptor, and on the surface of cancer cells.

The enzyme in mammals is composed of 17 subunits, five of which make up the easily detached F_1. The remainder subunits are components of two stalk domains and the proton pumping F_0 part of the machinery. Two of the subunits of the F_0 part are encoded on mitochondrial DNA while the other subunits are nuclear encoded. Mutations in the mitochondrial-encoded subunits of ATP synthase (Complex V) cause OXPHOS disease.

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