SANTA CRUZ BIOTECHNOLOGY, INC.

PDK3 (RR-2): sc-100535

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Pyruvate dehydrogenase kinase family members (PDK1, 2, 3, 4) are serine kinases that catalyze phosphorylation of the E1α subunit of the pyruvate dehydrogenase complex (PDC). PDC activity is controlled through phosphorylation and dephosphorylation of the E1α subunit, which leads to inactivation and reactivation, respectively. PDK3 binding to a free lipoyl domain (L2) in dihydrolypoyl acetyltransferase (E2), which comprises the core of PDC, leads to a large increase in E1α phosphorylation. Upregulation of PDK isoenzymes occurs during starvation conditions, where acetyl-CoA is alternatively generated through fatty acid oxidation. PDKs contain five conserved regions and are mechanistically similar to bacterial His-kinases in that both require histidine residues for activity. In mammals, transcripts for PDK3 are most abundant in testis and moderately expressed in heart and skeletal muscle.

REFERENCES

1. Gudi, R., et al. 1995. Diversity of the pyruvate dehydrogenase kinase gene family in humans. J. Biol. Chem. 270: 28989-28994.
2. Bowker-Kinley, M.M., et al. 1998. Evidence for existence of tissue-specific regulation of the mammalian pyruvate dehydrogenase complex. Biochem. J. 329: 191-196.
3. Sugden, M.C., et al. 2000. Selective modification of the pyruvate dehydrogenase kinase isoform profile in skeletal muscle in hyperthyroidism: implications for the regulatory impact of glucose on fatty acid oxidation. J. Endocrinol. 167: 339-345.
4. Mooney, B.P., et al. 2000. Histidine modifying agents abolish pyruvate dehydrogenase kinase activity. Biochem. Biophys. Res. Commun. 267: 500-503.
5. Baker, J.C., et al. 2000. Marked differences between two isoforms of human pyruvate dehydrogenase kinase. J. Biol. Chem. 275: 15773-15781.
6. Wu, P., et al. 2000. Starvation increases the amount of pyruvate dehydrogenase kinase in several mammalian tissues. Arch. Biochem. Biophys. 381: 1-7.

CHROMOSOMAL LOCATION

Genetic locus: PDK3 (human) mapping to Xp22.11; Pdk3 (mouse) mapping to X C3.

SOURCE

PDK3 (RR-2) is a mouse monoclonal antibody raised against recombinant PDK3 of human origin.

PRODUCT

Each vial contains 100 µg IgG_{2a} 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

PDK3 (RR-2) is recommended for detection of PDK3 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)] and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for PDK3 siRNA (h): sc-39029, PDK3 siRNA (m): sc-152139, PDK3 shRNA Plasmid (h): sc-39029-SH, PDK3 shRNA Plasmid (m): sc-152139-SH, PDK3 shRNA (h) Lentiviral Particles: sc-39029-V and PDK3 shRNA (m) Lentiviral Particles: sc-152139-V.

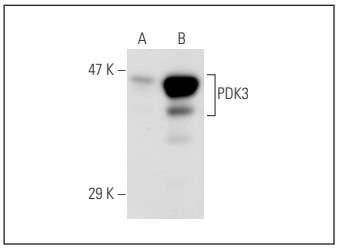
Molecular Weight of PDK3: 47 kDa.

Positive Controls: PDK3 (m): 293T Lysate: sc-122470, PDK3 (h2): 293 Lysate: sc-158838 or MCF7 whole cell lysate: sc-2206.

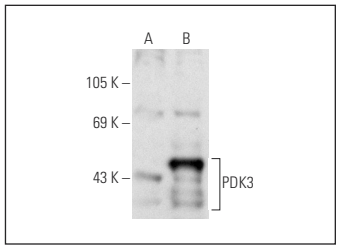
RECOMMENDED SUPPORT REAGENTS

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).

DATA



PDK3 (RR-2): sc-100535. Western blot analysis of PDK3 expression in non-transfected: sc-117752 (**A**) and mouse PDK3 transfected: sc-122470 (**B**) 293T whole cell lysates.



PDK3 (RR-2): sc-100535. Western blot analysis of PDK3 expression in non-transfected: sc-110760 (**A**) and human PDK3 transfected: sc-158838 (**B**) 293 whole cell lysates.

SELECT PRODUCT CITATIONS

1. Kluza, J., et al. 2011. Exploiting mitochondrial dysfunction for effective elimination of imatinib-resistant leukemic cells. PLoS ONE 6: e21924.
2. André, F., et al. 2017. Metabolic rewiring in cancer cells overexpressing the glucocorticoid-induced leucine zipper protein (GILZ): activation of mitochondrial oxidative phosphorylation and sensitization to oxidative cell death induced by mitochondrial targeted drugs. Int. J. Biochem. Cell Biol. 85: 166-174.
3. Zhao, J., et al. 2020. Deamidation shunts RelA from mediating inflammation to aerobic glycolysis. Cell Metab. 31: 937-955.e7.

RESEARCH USE

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