

# PDK3 (RR-2): sc-100535

## BACKGROUND

Pyruvate dehydrogenase kinase family members (PDK1, 2, 3, 4) are serine kinases that catalyze phosphorylation of the E1 $\alpha$  subunit of the pyruvate dehydrogenase complex (PDC). PDC activity is controlled through phosphorylation and dephosphorylation of the E1 $\alpha$  subunit, which leads to inactivation and reactivation, respectively. PDK3 binding to a free lipoyl domain (L2) in dihydrolipoyl acetyltransferase (E2), which comprises the core of PDC, leads to a large increase in E1 $\alpha$  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

- Gudi, R., et al. 1995. Diversity of the pyruvate dehydrogenase kinase gene family in humans. *J. Biol. Chem.* 270: 28989-28994.
- 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.
- 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.
- Mooney, B.P., et al. 2000. Histidine modifying agents abolish pyruvate dehydrogenase kinase activity. *Biochem. Biophys. Res. Commun.* 267: 500-503.
- Baker, J.C., et al. 2000. Marked differences between two isoforms of human pyruvate dehydrogenase kinase. *J. Biol. Chem.* 275: 15773-15781.
- 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  $\mu$ g IgG<sub>2a</sub> 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  $\mu$ g per 100-500  $\mu$ g 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.

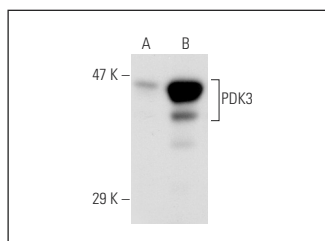
Molecular Weight of PDK3: 47 kDa.

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

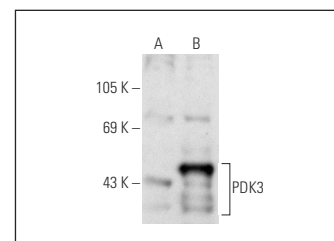
## RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended: 1) Western Blotting: use m-IgG $\kappa$  BP-HRP: sc-516102 or m-IgG $\kappa$  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

- Kluza, J., et al. 2011. Exploiting mitochondrial dysfunction for effective elimination of imatinib-resistant leukemic cells. *PLoS ONE* 6: e21924.
- 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.
- 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.