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PDK2 Antibody

Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP9827a

Product Information

Application	WB, FC, IHC-P, E
Primary Accession	Q15119
Reactivity	Human, Rat, Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	46154

Additional Information

Gene ID	5164
Other Names	[Pyruvate dehydrogenase (acetyl-transferring)] kinase isozyme 2, mitochondrial, Pyruvate dehydrogenase kinase isoform 2, PDH kinase 2, PDKII, PDK2, PDHK2
Target/Specificity	This PDK2 antibody is generated from rabbits immunized with recombinant protein of human PDK2.
Dilution	WB~~1:1000 FC~~1:10~50 IHC-P~~1:100~500 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	PDK2 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	PDK2
Synonyms	PDHK2
Function	Kinase that plays a key role in the regulation of glucose and fatty acid metabolism and homeostasis via phosphorylation of the pyruvate dehydrogenase subunits PDHA1 and PDHA2. This inhibits pyruvate dehydrogenase activity, and thereby regulates metabolite flux through the

tricarboxylic acid cycle, down-regulates aerobic respiration and inhibits the formation of acetyl-coenzyme A from pyruvate. Inhibition of pyruvate dehydrogenase decreases glucose utilization and increases fat metabolism. Mediates cellular responses to insulin. Plays an important role in maintaining normal blood glucose levels and in metabolic adaptation to nutrient availability. Via its regulation of pyruvate dehydrogenase activity, plays an important role in maintaining normal blood pH and in preventing the accumulation of ketone bodies under starvation. Plays a role in the regulation of cell proliferation and in resistance to apoptosis under oxidative stress. Plays a role in p53/TP53-mediated apoptosis.

Cellular Location Mitochondrion matrix.

Tissue Location Expressed in many tissues, with the highest level in heart and skeletal muscle, intermediate levels in brain, kidney, pancreas and liver, and low levels in placenta and lung

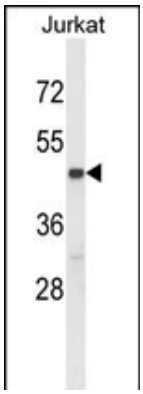
Background

PDK2 inhibits the mitochondrial pyruvate dehydrogenase complex by phosphorylation of the E1 alpha subunit, thus contributing to the regulation of glucose metabolism.

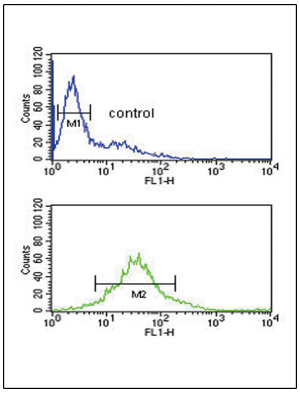
References

Li, J., et al. J. Biol. Chem. 284(49):34458-34467(2009) Fencl, F., et al. Pediatr. Nephrol. 24(5):983-989(2009) Sun, W., et al. Clin. Cancer Res. 15(2):476-484(2009) Hiromasa, Y., et al. Biochemistry 47(8):2312-2324(2008)

Images

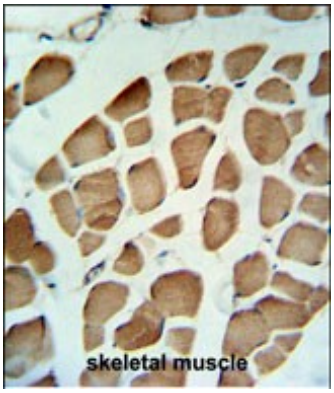


PDK2 Antibody (Cat. #AP9827a) western blot analysis in RD cell line and mouse heart,rat heart lysates (35ug/lane).This demonstrates the PDK2 antibody detected the PDK2 protein (arrow).



PDK2 Antibody (Cat. #AP9827a) flow cytometric analysis of Jurkat cells (bottom histogram) compared to a negative control cell (top histogram).FITC-conjugated goat-anti-rabbit secondary antibodies were used for the analysis.

PDK2 Antibody (Cat. #AP9827a) IHC analysis in formalin fixed and paraffin embedded skeletal muscle followed by



peroxidase conjugation of the secondary antibody and DAB staining. This data demonstrates the use of the PDK2 Antibody for immunohistochemistry. Clinical relevance has not been evaluated.

Citations

- Increasing cardiac pyruvate dehydrogenase flux during chronic hypoxia improves acute hypoxic tolerance.
- Selective Phosphorylation Inhibitor of Delta Protein Kinase C-Pyruvate Dehydrogenase Kinase Protein-Protein Interactions: Application for Myocardial Injury in Vivo.
- GAPDH (glyceraldehyde-3-phosphate dehydrogenase) Protein-Protein Interaction Inhibitor Reveals a Non-Catalytic Role for GAPDH Oligomerization in Cell Death.
- Increasing Pyruvate Dehydrogenase Flux as a Treatment for Diabetic Cardiomyopathy: A Combined 13C Hyperpolarized Magnetic Resonance and Echocardiography Study.
- Chronic CSE treatment induces the growth of normal oral keratinocytes via PDK2 upregulation, increased glycolysis and HIF1 □± stabilization.