

PFKL (A-6): sc-393713



The Power to Question

BACKGROUND

Phosphofructokinases (PFKs) are regulatory glycolytic enzymes that catalyze the irreversible conversion of fructose-6-phosphate to fructose-1,6-bisphosphate. Mammalian PFK is a tetramer made up of diverse combinations of three isoenzymes: muscle (PFK-1), liver (PFKL) and platelet (PFKP). PFKL (phosphofructokinase, liver), also referred to as PFK-B (phosphofructo-1-kinase isozyme B), phosphofructokinase 1 or phosphohexokinase, predominates in organs with active gluconeogenesis, such as liver and kidney. Overexpression of PFKL in transgenic mice results in a diminished glucose-induced Insulin response, which suggests that PFKL may play a role in glucose-induced Insulin secretion. PFKL is expressed at high levels in Down's syndrome (DS) patients, suggesting a possible role for PFKL in the pathogenesis of DS.

CHROMOSOMAL LOCATION

Genetic locus: PFKL (human) mapping to 21q22.3; Pfk1 (mouse) mapping to 10 C1.

SOURCE

PFKL (A-6) is a mouse monoclonal antibody raised against amino acids 46-81 mapping near the N-terminus of PFKL of human origin.

PRODUCT

Each vial contains 200 µg IgG₁ kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

PFKL (A-6) is available conjugated to agarose (sc-393713 AC), 500 µg/0.25 ml agarose in 1 ml, for IP; to HRP (sc-393713 HRP), 200 µg/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-393713 PE), fluorescein (sc-393713 FITC), Alexa Fluor® 488 (sc-393713 AF488), Alexa Fluor® 546 (sc-393713 AF546), Alexa Fluor® 594 (sc-393713 AF594) or Alexa Fluor® 647 (sc-393713 AF647), 200 µg/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor® 680 (sc-393713 AF680) or Alexa Fluor® 790 (sc-393713 AF790), 200 µg/ml, for Near-Infrared (NIR) WB, IF and FCM.

APPLICATIONS

PFKL (A-6) is recommended for detection of PFKL of mouse, rat and human origin by Western Blotting (starting dilution 1:100, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for PFKL siRNA (h): sc-106400, PFKL siRNA (m): sc-152180, PFKL shRNA Plasmid (h): sc-106400-SH, PFKL shRNA Plasmid (m): sc-152180-SH, PFKL shRNA (h) Lentiviral Particles: sc-106400-V and PFKL shRNA (m) Lentiviral Particles: sc-152180-V.

Molecular Weight of PFKL: 80 kDa.

Positive Controls: HeLa whole cell lysate: sc-2200, Hep G2 cell lysate: sc-2227 or A-431 whole cell lysate: sc-2201.

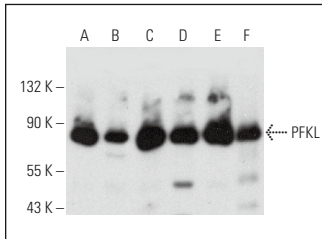
RESEARCH USE

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

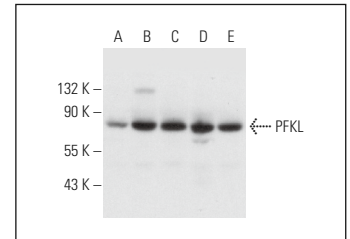
STORAGE

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

DATA



PFKL (A-6): sc-393713. Western blot analysis of PFKL expression in MCF7 (A), WI-38 (B), C2C12 (C), RAW 264.7 (D), KNRK (E) and RPE-J (F) whole cell lysates.



PFKL (A-6): sc-393713. Western blot analysis of PFKL expression in HeLa (A), Hep G2 (B), SK-BR-3 (C), A-431 (D) and LNCaP (E) whole cell lysates.

SELECT PRODUCT CITATIONS

1. Ichhaporia, V.P., et al. 2018. SIL1, the endoplasmic-reticulum-localized BiP co-chaperone, plays a crucial role in maintaining skeletal muscle proteostasis and physiology. *Dis. Model. Mech.* 11: dmm033043.
2. Liu, L., et al. 2020. Triose kinase controls the lipogenic potential of fructose and dietary tolerance. *Cell Metab.* 32: 605-618.e7.
3. Cheung, R.A., et al. 2021. Relocation of phosphofructokinases within epithelial cells is a novel event preceding breast cancer recurrence that accurately predicts patient outcomes. *Am. J. Physiol., Cell Physiol.* 321: C654-C670.
4. Lim, J.S., et al. 2022. Mutual regulation between phosphofructokinase 1 platelet isoform and VEGF promotes glioblastoma tumor growth. *Cell Death Dis.* 13: 1002.
5. Gandhirajan, A., et al. 2023. SIRT2-PFKP interaction dysregulates phagocytosis in macrophages with acute ethanol-exposure. *Front. Immunol.* 13: 1079962.
6. Sun, X., et al. 2023. The diapause-like colorectal cancer cells induced by SMC4 attenuation are characterized by low proliferation and chemotherapy insensitivity. *Cell Metab.* 35: 1563-1579.e8.
7. Park, S.H., et al. 2024. The m⁶A writer RBM15 drives the growth of triple-negative breast cancer cells through the stimulation of serine and glycine metabolism. *Exp. Mol. Med.* 56: 1373-1387.
8. Chen, T., et al. 2024. AKT1 phosphorylation of cytoplasmic ME2 induces a metabolic switch to glycolysis for tumorigenesis. *Nat. Commun.* 15: 686.

PROTOCOLS

See our web site at www.scbt.com for detailed protocols and support products.

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