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PFKP (Phospho-Tyr64) Antibody

#58022

Number: 58022

Amount: 100μg/100μl

Form of Antibody: Rabbit IgG in phosphate buffered saline (without Mg2+ and Ca2+), pH 7.4, 150mM NaCl,0.02% sodium azide and 50% glycerol.

Storage/Stability: Store at -20℃/1 year

Immunogen: synthetic phosphopeptide corresponding to residues surrounding Tyr64 of human PFKP

Purification: The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific phosphopeptide. The antibody against non-phosphopeptide was removed by chromatography using non-phosphopeptide corresponding to the phospholation site.

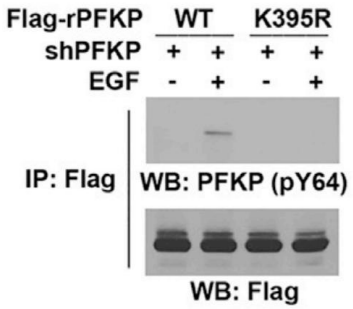
Specificity/Sensitivity: PFKP（Phospho-Tyr64)antibody detects endogenous levels of PFKP only when phospholated at Tyrosine64 .

Reactivity: Human

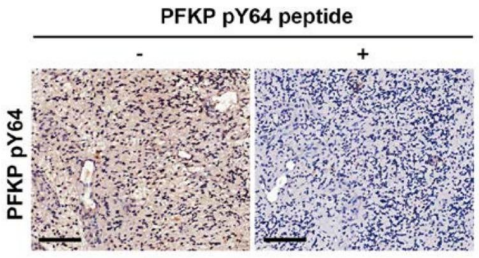
Applications:

Predicted MW: 80KD

WB **:**1:500~1:1000 IHC:1:50-200



U251 cells with or without PFKP depletion and reconstituted expression of WT Flag-rPFKP or Flag-rPFKP K395R were treated with or without EGF (100 ng/mL) for 15 min. Immunoprecipitation analyses were performed with the indicated antibodies.



PFKP Y64 Phosphorylation Antibody specificity was validated in human GBM specimens, in the presence or absence of a blocking peptide that was specific for phosphorylated PFKP Y64. Scale bar, 100 μm.

Background :Activated EGFR induced PFKP phosphorylation at Y64. Phosphorylated PFKP Y64 bound to the N-terminal SH2 domain of p85a and promoted activation of PI3K and AKT, leading to enhanced AKT-dependent PFK2 activation, F-2,6-BP-production-dependent PFK1 activation, and glucose transporter type 1 (GLUT1) expression. The feedback regulation of glycolytic enzymes by PFKP Y64 phosphorylation promoted the Warburg effect, cell proliferation, and tumorigenesis [1] .

Reference:[1] Lee JH, Liu R, Li J, Wang Y, Tan L, Li XJ, Qian X, Zhang C, Xia Y, Xu D, Guo W, Ding Z, Du L, Zheng Y, Chen Q, Lorenzi PL, Mills GB, Jiang T, Lu Z. EGFR-Phosphorylated Platelet Isoform of Phosphofructokinase 1 Promotes PI3K Activation. ***Mol Cell.*** 2018 Apr 19;70(2):197-210.e7. doi: 10.1016/j.molcel.2018.03.018.