PRPS1/2（Phospho-Ser180/183）Antibody

#58023

Number: 58023

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 Ser180/183 of human PRPS1/2

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.

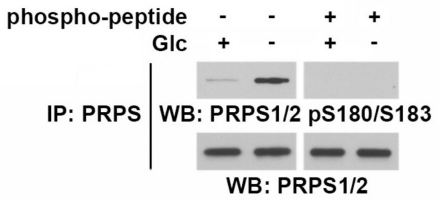
Specificity/Sensitivity: PRPS1/2（Phospho- Ser180/183)antibody detects endogenous levels of PRPS1/2 only when phospholated at Serine180/183 .

Reactivity: Human

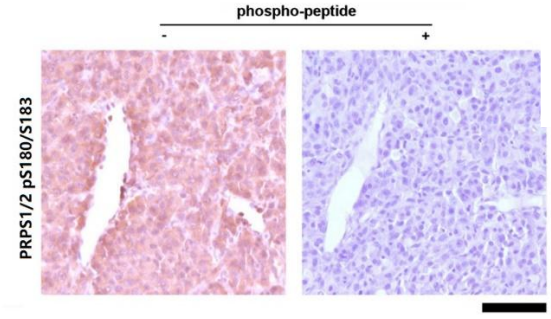
Applications:

Predicted MW: 100KD

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



U87 cells were cultured in the presence or absence of Glc for 3 h. Cell lysates were subjected to an immunoblot analysis and incubated with the indicated antibodies in the presence or absence of the corresponding phospho-blocking peptide.



Antibody specificity was validated in xenografted tumor specimens in the presence or absence of the blocking peptide that was specific for phosphorylated PRPS1 S180 and PRPS2 S183. Bar, 100μm.

Background :Glucose deprivation or hypoxia results in the AMPK-mediated phosphorylation of phosphoribosyl pyrophosphate synthetase 1 (PRPS1) S180 and PRPS2 S183, AMPK converts PRPS1/2 hexamers to monomers, and inhibits PRPS1/2 activity and subsequent nucleotide and NAD synthesis to maintain tumor cell growth and survival. The expression of nonphosphorylatable PRPS1/2 mutants greatly decreased cellular ATP and NADPH levels, increased reactive oxygen species (ROS) levels and cell apoptosis, and inhibited brain tumorigenesis [1] .

Reference:[1] Qian X, Li X, Tan L, Lee JH, Xia Y, Cai Q, Zheng Y, Wang H, Lorenzi PL, Lu Z. Conversion of PRPS Hexamer to Monomer by AMPK-Mediated Phosphorylation Inhibits Nucleotide Synthesis in Response to Energy Stress. ***Cancer Discov.*** 2018 Jan;8(1):94-107. doi: 10.1158/2159-8290.CD-17-0712.