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## PRPS1/2 (Acety1-Lys29 ) Antibody



Number: 58033

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°C/1 year

**Immunogen:** synthetic acetylpeptide corresponding to residues surrounding Lys29 of human PRPS1/2 **Purification:** The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific acetylpeptide. The antibody against non-acetylpeptide was removed

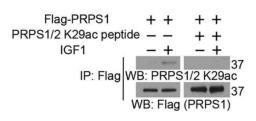
by chromatography using non-acetylpeptide corresponding to the acetylation site.

**Specificity/Sensitivity:** PRPS1/2 (Acetyl-Lys29 )antibody detects endogenous levels of PRPS1/2 only when acetylated at lysine29.

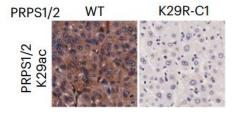
Reactivity: Human

## Applications:

Predicted MW: 37KD WB :1:500~1:1000 IHC:1:50-200



Immunoblotting analyses of human HCC samples were performed with the indicated antibodies and a PRPS1/2 K29 acetylation blocking peptide.



Parental Huh7 cells and the indicated clones with knock-in expression of PRPS1/2 K29R mutants were intrahepatically injected into athymic nude mice. IHC analyses of the indicated xenograft tumours from nude mice were performed with the indicated antibodies.

**Background** :PRPS1 catalyzes the synthesis of phosphoribosylpyrophosphate (PRPP) that is essential for nucleotide synthesis. In the cytosol, CLOCK acetylates PRPS1/2 K29 and blocks HSC70-mediated and lysosome-dependent PRPS1/2 degradation. Stabilized PRPS1/2 promote de novo nucleotide synthesis and HCC cell proliferation and liver tumour growth. PRPS1/2 K29 acetylation are positively correlated with HCC poor prognosis [1]

**Reference**:[1] Liu T, Wang Z, Ye L, Duan Y, Jiang H, He H, Xiao L, Wu Q, Xia Y, Yang M, Wu K, Yan M, Ji G, Shen Y, Wang L, Li L, Zheng P, Dong B, Shao F, Qian X, Yu R, Zhang Z, Lu Z, Xu D. Nucleus-exported CLOCK acetylates PRPS to promote de novo nucleotide synthesis and liver tumour growth. Nat Cell Biol. 2023 Feb;25(2):273-284. doi: 10.1038/s41556-022-01061-0.