Revision 1

Store at -20C

#3827

# Phospho-PKM2 (Tyr105) Antibody

Cell Signaling TECHNOLOGY®

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**For Research Use Only. Not for Use in Diagnostic Procedures.**

| Applications: | Reactivity: | Sensitivity: | MW (kDa): | Source/Isotype: | UniProt ID: | Entrez-Gene Id: |
|---|---|---|---|---|---|---|
| W | H M R Mk | Endogenous | 60 | Rabbit | #P14618 | 5315 |

## Product Usage Information

| Application | Dilution |
|---|---|
| Western Blotting | 1:1000 |

## Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA and 50% glycerol. Store at –20°C. Do not aliquot the antibody.

## Specificity/Sensitivity

Phospho-PKM2 (Tyr105) Antibody detects endogenous levels of PKM2 protein only when phosphorylated at Tyr105. This antibody may slightly cross react with PKM1 phosphorylated at the equivalent site.

## Source / Purification

Polyclonal antibodies are produced by immunizing animals with a synthetic phosphopeptide corresponding to the sequence surrounding Tyr105 of human PKM2 protein. Antibodies are purified by protein A and peptide affinity chromatography.

## Background

Pyruvate kinase is a glycolytic enzyme that catalyses the conversion of phosphoenolpyruvate to pyruvate. In mammals, the M1 isoform (PKM1) is expressed in most adult tissues (1). The M2 isoform (PKM2) is an alternatively spliced variant of M1 that is expressed during embryonic development (1). Research studies found that cancer cells exclusively express PKM2 (1-3). PKM2 is shown to be essential for aerobic glycolysis in tumors, known as the Warburg effect (1). When cancer cells switch from the M2 isoform to the M1 isoform, aerobic glycolysis is reduced and oxidative phosphorylation is increased (1). These cells also show decreased tumorigenicity in mouse xenografts (1). Recent studies showed that PKM2 is not essential for all tumor cells (4). In the tumor model studied, PKM2 was found to be active in the non-proliferative tumor cell population and inactive in the proliferative tumor cell population (4). Additional studies show that the oncogenic forms of FGFR1 directly phosphorylate Tyr105 of PKM2 and thereby inhibit the formation of active, tetrameric PKM2 (5). A PKM2 missense mutation found in cancer cells results in the replacement of Tyr105 by phenylalanine and leads to reduced cell proliferation during hypoxia and tumor growth in nude mice xenografts (5). These findings suggest that the phosphorylation at Tyr105 is a critical switch for the metabolism in cancer cells that promotes tumor growth (5).

Phosphorylation of PKM2 on Tyr105 was identified at Cell Signaling Technology (CST) using PhosphoScan®, CST's LC-MS/MS platform for phosphorylation site discovery.

## Background References

1. Christofk, H.R. et al. (2008) *Nature* 452, 230-3.
2. Mazurek, S. et al. (2005) *Semin Cancer Biol* 15, 300-8.
3. Dombrauckas, J.D. et al. (2005) *Biochemistry* 44, 9417-29.
4. Israelsen, W.J. et al. (2013) *Cell* 155, 397-409.
5. Hitosugi, T. et al. (2009) *Sci Signal* 2, ra73.

## Species Reactivity

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

## Western Blot Buffer

IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

## Applications Key

**W:** Western Blotting

## Cross-Reactivity Key

**H:** Human **M:** Mouse **R:** Rat **Mk:** Monkey

## Trademarks and Patents



## Limited Uses

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