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Histone H3 (Phospho-Thr11)

Number: 58031

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 phosphopeptide corresponding to residues surrounding Thr11 of human Histone H3

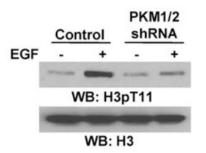
Purification: The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific phosphopeptide. The antibody against non-phosphopeptide was removed by chromatography using non-phosphopeptide corresponding to the phospholation site.

Specificity/Sensitivity: Histone H3 (Phospho-Thr11)antibody detects endogenous levels of Histone H3 only when phospholated at Threonine11.

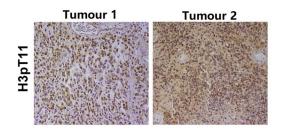
Reactivity: Human

Applications:

Predicted MW: 17KD WB :1:500~1:1000 IHC:1:50-200



U87/EGFR cells expressing a control shRNA or shRNA against a coding sequence for both PKM1 and PKM2 were treated with or without EGF (100 ng/ml) for 6 hr. Immunoblotting analysis of endogenous histone H3 was performed with the indicated antibodies.



Immunohistochemical staining with anti-phospho-H3-T11 antibodies was performed on 45 GBM specimens. **Background** :Tumor-specific pyruvate kinase M2 (PKM2) is essential for the Warburg effect. In addition to its well established role in aerobic glycolysis, PKM2 directly regulates gene transcription. PKM2 directly binds to histone H3 and phosphorylates histone H3 at T11 upon EGF receptor activation. PKM2-dependent histone H3 modifications are instrumental in EGF-induced expression of cyclin D1 and c-Myc, tumor cell proliferation, cell-cycle progression, and brain tumorigenesis. In addition, levels of histone H3 T11 phosphorylation correlate with glioma malignancy grades and prognosis [1].

Reference:[1] Yang W, Xia Y, Hawke D, Li X, Liang J, Xing D, Aldape K, Hunter T, Alfred Yung WK, Lu Z. PKM2 phosphorylates histone H3 and promotes gene transcription and tumorigenesis. Cell. 2012 Aug 17;150(4):685-96. doi: 10.1016/j.cell.2012.07.018.