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## Phospho-Myt1 (Ser83) Antibody

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

|                           |                         |                                   |                        |                                  |                               |                                |
|---------------------------|-------------------------|-----------------------------------|------------------------|----------------------------------|-------------------------------|--------------------------------|
| <b>Applications:</b><br>W | <b>Reactivity:</b><br>H | <b>Sensitivity:</b><br>Endogenous | <b>MW (kDa):</b><br>70 | <b>Source/Isotype:</b><br>Rabbit | <b>UniProt ID:</b><br>#Q99640 | <b>Entrez-Gene Id:</b><br>9088 |
|---------------------------|-------------------------|-----------------------------------|------------------------|----------------------------------|-------------------------------|--------------------------------|

### Product Usage Information

#### Application

Western Blotting

#### Dilution

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-Myt1 (Ser83) Antibody detects endogenous levels of Myt1 only when phosphorylated at serine 83.

### Source / Purification

Polyclonal antibodies are produced by immunizing animals with a synthetic phosphopeptide corresponding to residues surrounding Ser83 of human Myt1. Antibodies are purified by protein A and peptide affinity chromatography.

### Background

Entry of all eukaryotic cells into mitosis is regulated by activation of cdc2 kinase. The critical regulatory step in activating cdc2 during progression into mitosis appears to be dephosphorylation of Tyr15 and Thr14 (1,2). Phosphorylation at Tyr15 and Thr14 and inhibition of cdc2 is carried out by Wee1 and Myt1 protein kinases, while Tyr15 dephosphorylation and activation of cdc2 is carried out by the cdc25 phosphatase (1,3,4). Hyperphosphorylation and inactivation of Myt1 in mitosis suggests that one or more kinases activated at the G2/M transition negatively regulates Myt1 activity. Kinases shown to phosphorylate Myt1 include cdc2, p90RSK, Akt, and Plk1 (5-7).

Although Akt has been shown to phosphorylate Asterina (starfish) Myt1 at a consensus Akt phosphorylation site (7), the orthologous site, Ser83, in human Myt1 may be phosphorylated by a different kinase.

### Background References

1. Watanabe, N. et al. (1995) *EMBO J.* 14, 1878-1891.
2. Hunter, T. (1995) *Cell* 80, 225-236.
3. Galaktionov, K. et al. (1995) *Genes Dev* 9, 1046-58.
4. McGowan, C.H. and Russell, P. (1993) *EMBO J* 12, 75-85.
5. Booher, R.N. et al. (1997) *J Biol Chem* 272, 22300-6.
6. Palmer, A. et al. (1998) *EMBO J* 17, 5037-47.
7. Nakajima, H. et al. (2003) *J Biol Chem* 278, 25277-80.

### 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

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