Revision 5

Store at -20C

#11988

HES1 (D6P2U) Rabbit mAb

Cell Signaling TECHNOLOGY®

Orders: 877-616-CELL (2355)
orders@cellsignal.com

Support: 877-678-TECH (8324)

Web: info@cellsignal.com
cellsignal.com

3 Trask Lane | Danvers | Massachusetts | 01923 | USA

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Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, IP, IHC-P	H M R Mk	Endogenous	30	Rabbit IgG	#Q14469	3280

Product Usage Information

Application	Dilution
Western Blotting	1:1000
Immunoprecipitation	1:200
Immunohistochemistry (Paraffin)	1:3200 - 1:12800

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.

For a carrier free (BSA and azide free) version of this product see product #33699.

Specificity/Sensitivity

HES1 (D6P2U) Rabbit mAb recognizes endogenous levels of total HES1 protein.

Source / Purification

Monoclonal antibody is produced by immunizing animals with recombinant protein specific to human HES1 protein. The epitope has been mapped to residues surrounding Ala230.

Background

HES1 (Hairy and Enhancer of Split 1) is one of seven members of the HES family of basic helix-loop-helix (bHLH) transcription factors which function primarily to repress transcription of bHLH-dependent genes (1). HES1 is understood to play an important conserved role in maintaining pluripotency of embryonic and adult stem/progenitor cells via the transcriptional repression of genes that promote differentiation (1,2). HES1 is particularly well known as a repressive mediator of the canonical Notch signaling pathway (3). HES1 plays a key role in mediating Notch-dependent T cell lineage commitment (4), and has been reported to be an essential mediator of Notch-induced T cell acute lymphoblastic leukemia (T-ALL) (4,5). HES1 is also reported to mediate Notch-induced repression of differentiation in a number of cancer cell types. A conditional deletion of HES1 from intestinal tumor cells in APC-mutant mice reduced tumor cell proliferation, while promoting differentiation toward epithelial lineages (6). Overexpression of HES1 in a human osteosarcoma (OS) cell line was shown to repress expression of the Notch antagonist *Dtx1*, leading to increased OS cell invasiveness (7). Other genes subject to transcriptional repression by HES1 include *Neurogenin-2*, *Math1/Atoh1* and the NOTCH ligands *DLL1* and *Jagged1* (6,8,9).

Background References

1. Kageyama, R. et al. (2007) *Development* 134, 1243-51.
2. Hatakeyama, J. et al. (2004) *Development* 131, 5539-50.
3. Kobayashi, T. and Kageyama, R. (2010) *Genes Cells* 15, 689-98.
4. Wendorff, A.A. et al. (2010) *Immunity* 33, 671-84.
5. Espinosa, L. et al. (2010) *Cancer Cell* 18, 268-81.
6. Ueo, T. et al. (2012) *Development* 139, 1071-82.
7. Zhang, P. et al. (2010) *Oncogene* 29, 2916-26.
8. Kageyama, R. et al. (2008) *Dev Growth Differ* 50 Suppl 1, S97-103.
9. Kobayashi, T. et al. (2009) *Genes Dev* 23, 1870-5.

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 **IP:** Immunoprecipitation **IHC-P:** Immunohistochemistry (Paraffin)

Cross-Reactivity Key

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

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