

Histone 3.3 G34V

Cat.No. HS-388 011; Monoclonal mouse antibody, 100 µg purified IgG (lyophilized)

Data Sheet

Reconstitution/ Storage	100 µg purified IgG, lyophilized. Azide was added before lyophilization. For reconstitution add 100 µl H ₂ O to get a 1mg/ml solution in PBS. Then aliquot and store at -20°C until use.
Applications	WB: not recommended ICC: yes IHC: yes IHC-P/FFPE: 1 : 1000 up to 1 : 2000
Clone	329E5
Subtype	IgG2b
Immunogen	Synthetic peptide corresponding to AA 29 to 38 from human H3F3A G34V (UniProt Id: P84243)
Epitop	Epitop: AA 29 to 38 from human H3F3A G34V (UniProt Id: P84243)
Reactivity	Reacts with: human (P84243). Other species not tested yet.
Specificity	Specific for the H3.3 G34V mutant. Negligible cross-reactivity to H3.3 G34R, and no cross-reactivity to unmutated H3.3. (K.O. verified)

Selected General References

Mutations in chromatin machinery and pediatric high-grade glioma.

Lulla RR, Saratsis AM, Hashizume R

Science advances (2016) 2(3): e1501354.

Histone H3.3 mutations drive pediatric glioblastoma through upregulation of MYCN.

Bjerke L, Mackay A, Nandhabalan M, Burford A, Jury A, Popov S, Bax DA, Carvalho D, Taylor KR, Vinci M, Bajrami I, et al. Cancer discovery (2013) 3(5): 512-9.

Driver mutations in histone H3.3 and chromatin remodelling genes in paediatric glioblastoma.

Schwartzenbacher J, Korshunov A, Liu XY, Jones DT, Pfaff E, Jacob K, Sturm D, Fontebasso AM, Quang DA, Tönjes M, Hovestadt V, et al.

Nature (2012) 482(7384): 226-31.

Somatic histone H3 alterations in pediatric diffuse intrinsic pontine gliomas and non-brainstem glioblastomas.

Wu G, Broniscer A, McEachron TA, Lu C, Paugh BS, Becksfort J, Qu C, Ding L, Huether R, Parker M, Zhang J, et al. Nature genetics (2012) 44(3): 251-3.

TO BE USED IN VITRO / FOR RESEARCH ONLY NOT TOXIC, NOT HAZARDOUS, NOT INFECTIOUS, NOT CONTAGIOUS

The gene mutations **H3.3 G34V** and H3.3 G34R of histone 3.3 (H3.3 or H3F3A) have been recently identified as driver mutations in paediatric glioblastoma. G34V/R mutations are restricted to tumors of the cerebral hemispheres and are most prevalent in adolescents and young adults. These mutations cause profound upregulation of MYCN, a potent oncogene. Emerging evidence strongly suggests that paediatric glioblastomas with H3F3A mutations can be subclassified into distinct entities.

This antibody is part of the HistoSure® product line, specifically developed and tested for human pathology.