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50748

Astrocyte Markers Antibody Sampler Kit



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

1 Kit (7 x 20 microliters)

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
GFAP (E4L7M) XP [®] Rabbit mAb	80788	20 µl	50 kDa	Rabbit IgG
EAAT1 (D44E2) XP [®] Rabbit mAb	5684	20 µl	58 kDa	Rabbit IgG
Survivin (71G4B7) Rabbit mAb	2808	20 µl	16 kDa	Rabbit IgG
EAAT2 (E3P5K) Rabbit mAb	20848	20 µl	65 kDa	Rabbit IgG
ALDH1L1 (E7I2Q) Rabbit mAb	85828	20 µl	98 kDa	Rabbit IgG
AQP4 (D1F8E) XP [®] Rabbit mAb	59678	20 µl	28 kDa	Rabbit IgG
GAT1 (E7J1B) Rabbit mAb	37342	20 µl	65 kDa	Rabbit IgG
Anti-rabbit IgG, HRP-linked Antibody	7074	100 µl		Goat

Please visit cellsignal.com for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

Description	The Astrocyte Markers Antibody Sampler Kit provides an economical means of detecting astrocyte markers by Immunofluorescence or Western Blot. The kit includes enough antibodies to perform at least two western blot or twenty IF tests with each primary antibody.
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. <i>Do not aliquot the antibodies.</i>
Background	Astrocytes are a population of cells with distinctive morphological and functional characteristics that differ within specific areas of the brain. Postnatally, astrocyte progenitors migrate to reach their brain area and related properties. They have a regulatory role in brain functions that are implicated in neurogenesis and synaptogenesis, controlling blood-brain barrier permeability and maintaining extracellular homeostasis. Mature astrocytes also express some genes enriched in cell progenitors, suggesting they can retain proliferative potential (1). Astrocytes in the human brain are characterized by a heavy expression of the glial fibrillary acidic protein (GFAP), comprised of interlaminar that are located in layers I and II, protoplasmic in layers III and IV, varicose projections in layers V and VI, and fibrous astroglia in white matter (2,3).
	Aquaporins (AQP) are integral membrane proteins that serve as channels in the transfer of water and small solutes across the membrane. AQP4 is present in the brain and it is enriched in astrocytes to regulate water homeostasis, preventing cerebral edema caused by solute imbalance (4,5). Excitatory amino acid transporters are members of sodium-dependent, high-affinity transporters that mediate the uptake of L-glutamate and D-aspartate (6). GABA transmitters are Na ⁺ /Cl ⁻ dependent transporters that regulate neurotransmitter transport, including GAT1 (SLC6), GAT2, GAT3, and BGT1 (7). GAT1 expresses in the brain, preferentially in glial cells, but is also found in neurons, regulating the uptake and release of neurotransmitters in terminal clefts (8-10). EAAT2, also known as GLT-1, is primarily expressed in astrocytes accounting for up to 90% of the total glutamate transport in the brain. EAAT1 upregulates increased concentrations of glutamate in astrocytes and has a neuroprotective potential following ischemia since reactive astrocytes and activated microglia express EAAT1 but not EAAT2 (11-13).
	Aldehyde dehydrogenase 1 family member L1 (ALDH1L1) is a member of the aldehyde dehydrogenase super family (14,15). ALDH1L1 has also been shown to be a useful astrocyte marker throughout the grey and white matter of the brain, labeling both the cell body and processes of astrocytes. ALDH1L1 does not label neurons or oligodendrocytes (16). It has been shown that expression of ALDH1L1 in astrocytes is stably expressed through normal tissue and during astrogliosis (17). Survivin is a 16 kDa anti-apoptotic protein highly expressed during fetal development and cancer malignancy. It binds and inhibits caspase-3, controlling the checkpoint in the G2/M-phase of the cell cycle by inhibiting apoptosis and promoting cell division (18). Survivin expression is associated with malignant phenotypes and prognosis of glioma (19).
Background References	1. Siracusa, R. et al. (2019) Front Pharmacol 10, 1114.

	 Vasile, F. et al. (2017) <i>Brain Struct Funct</i> 222, 2017-2029. Eng, L.F. et al. (2000) <i>Neurochem Res</i> 25, 1439-51. Takata, K. et al. (2004) <i>Prog Histochem Cytochem</i> 39, 1-83. Kobayashi, H. et al. (2004) <i>J Pharmacol Sci</i> 96, 264-70. Amara, S.G. and Fontana, A.C. (2002) <i>Neurochem Int</i> 41, 313-8. Kristensen, A.S. et al. (2011) <i>Pharmacol Rev</i> 63, 585-640. Borden, L.A. (1996) <i>Neurochem Int</i> 29, 335-56. Moldavan, M. et al. (2017) <i>J Neurophysiol</i> 118, 3092-3106. Lorenz-Guertin, J.M. and Jacob, T.C. (2018) <i>Dev Neurobiol</i> 78, 238-270. Hediger, M.A. (1999) <i>Am J Physiol</i> 277, F487-92. Gegelashvili, G. et al. (1996) <i>Neuroreport</i> 8, 261-5. Beschorner, R. et al. (2007) <i>Histopathology</i> 50, 897-910. Krupenko, S.A. (2009) <i>Chem Biol Interact</i> 178, 84-93. Krupenko, S.A. and Oleinik, N.V. (2002) <i>Cell Growth Differ</i> 13, 227-36. Cahoy, J.D. et al. (2019) <i>J Neurochem</i> 150, 420-440. Reed, J.C. and Reed, S.I. (1999) <i>Nat Cell Biol</i> 1, E199-200. Tong, X. et al. (2019) <i>Oncol Lett</i> 18, 359-367.
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