

anti-Amyloid Precursor Protein (APP C-terminus) antibody, rabbit serum (AC1)

74-104 100 µl

Shipping and Storage: Shipped at 4°C or -20°C, and upon arrival, aliquot and store at -20°C.

Immunogen: Synthetic peptide corresponding to the C-terminus (aa 671-695) of human APP695.

Form: Rabbit antiserum added with 0.05% sodium azide.

Reactivity: Specific to human, mouse and rat APP.

Applications:

1. Western blotting (dilution: 1/3,000)
2. Immunocytochemistry (dilution: 1/1,000)
3. Immunohistochemistry (dilution: 1/500) Other applications have not been tested.

Background: The Alzheimer amyloid precursor protein (APP) is an integral membrane protein expressed in many tissues and concentrated in the synapses of neurons. Its primary function is not known, though it has been implicated as a regulator of synapse formation and neural plasticity. APP is best known and most commonly studied as the precursor molecule whose proteolysis generates amyloid beta ($A\beta$), a 39- to 42-amino acid peptide whose amyloid fibrillar form is the primary component of amyloid plaques found in the brains of Alzheimer's disease patients. Isoform APP695 lacking the protease inhibitor domain is the predominant form in neuronal tissues. An antibody (named AC1) against the C-terminus of human APP695 (aa 671-695) was raised in rabbit (2).

Data Link: Swiss-Prot [P05067](#)

References: This antibody has been used in ref.2, 3, 4 and 5.

1. Kang HG et al. (1987) "The precursor of Alzheimer's disease amyloid A4 protein resembles a cell-surface receptor." *Nature* 325: 733-736 PMID: [2881207](#)
2. Yoshikawa K et al. (2002) "Degeneration in vitro of post-mitotic neurons overexpressing the Alzheimer amyloid protein precursor." *Nature* 359: 64-67 (1992) PMID: [1301020](#)
3. Nishimura I et al. (2002) "Cell death induced by a caspase-cleaved transmembrane fragment of the Alzheimer amyloid precursor protein." *Cell Death Differ.* 9: 199-208 PMID: [11840170](#)
4. Nishimura I et al. (2003) "Upregulation and antiapoptotic role of endogenous Alzheimer amyloid precursor protein in dorsal root ganglion neurons." *Exp. Cell Res.* 286: 241-251 PMID: [12749853](#)
5. Nishimura I et al. (1998) "Degeneration in vivo of rat hippocampal neurons by wild-type Alzheimer amyloid precursor protein overexpressed by adenovirus-mediated gene transfer." *J. Neurosci.* 18: 2387-2396 PMID: [9502800](#)

Related products: #74-102 anti-Activated Caspase3 antibody, #74-106 anti-APP (N-terminal) antibody, #74-108 anti-APP (C-terminal of the caspase 3- cleaved APP) antibody, #74-110 anti-APP Δ 31 (specific to C-terminal APP Δ 31) antibody

To be continued.....

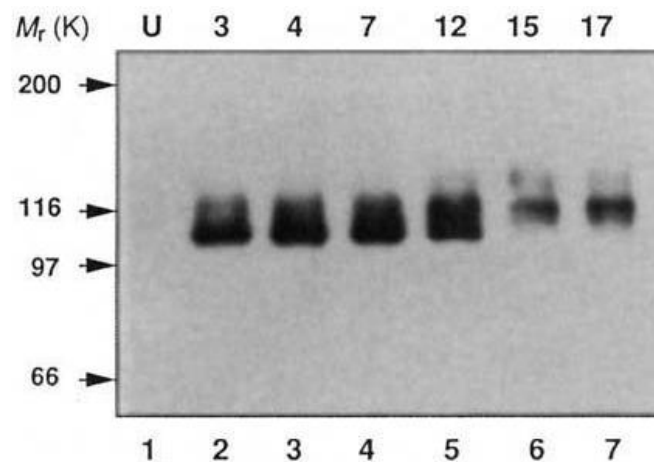


Fig.1 Endogenous expression of APP in mouse P19 cells during neural differentiation was analyzed by Western blotting using this antibody (ref.2).

lane1, undifferentiated P19 cells (U); lane2, day 3; lane3, day4; lane4, day7; lane5, day12; lane6, day15; lane7, day17.

APP species with 105-120K markedly increased during days 3-12, but declined thereafter. On the other hand, APP species with 115-130K were detected on day 15 and 17.

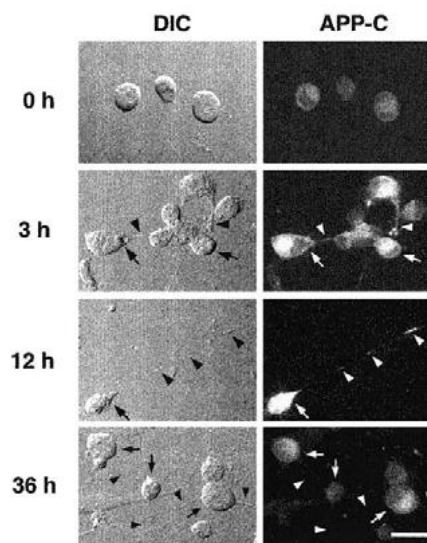


Fig.2 Immunocytochemistry for APP (ref 4).

Mouse dorsal root ganglion neurons were cultured in the presence of nerve growth factor (NGF), fixed at indicated time points, and immunostained for the C-terminus of APP with this antibody. Left panels are differential interference contrast images of the same fields. APP immunoreactivity was very low at 0 h but increased in neuronal somata (arrows), neurites (arrowheads) 3-12 h after NGF treatment, a period when neurites showed a marked outgrowth. APP immunoreactivity decreased at 36 h.