

anti-APP Δ C31 (specific to C-terminal APP Δ 31) antibody, rabbit serum (SAC)

74-110 100ul

The Alzheimer **amyloid precursor protein (APP)** is a transmembrane protein whose abnormal processing is associated with the pathogenesis of Alzheimer's disease. APP695 lacking the protease inhibitor domain is the predominant form in neuronal tissues. APP695 is cleaved by caspases into the 664-residue amino (N)-terminal fragment that lacks the carboxyl C-terminal 31-residues (**APP Δ C31**) and the 31-residues C-terminal fragment (APP-C31). **APP Δ C31** potentially plays pathophysiological roles in neuronal death (ref.3). An antibody (named SAC) against the C-terminus of caspase 3-cleaved human APP695 (APP Δ C31) was raised in rabbit.

Applications:

1. Western blotting (dilution: 1/3,000-1/1,000)
2. Immunocytochemistry (dilution: 1/1,000-1/500)
3. ELISA

Other applications have not been tested.

Immunogen: Synthetic peptide corresponding to the C-terminus of the caspase 3-cleaved human APP (aa 658-664 of human APP695).

Specificity: Specific to the C-terminal end of **APP Δ C31** of human, mouse and rat.

Form: Antiserum added with 0.05% sodium azide.

Storage: Shipped at 4°C and stored at -20°C

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

References: This antibody was used in ref.3 and 4.

1. Kang HG *et al.* (1987) "The precursor of Alzheimer's disease amyloid A4 protein resembles a cell-surface receptor." *Nature* **325**: 33-736 PMID: [2881207](#)
2. Selkoe DJ (1994) "Normal and abnormal biology of the beta-amyloid precursor protein." *Annu. Rev. Neurosci.* **17**: 489-517 PMID: [8210185](#)
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](#)

Related products: #74-102 anti-Activated Caspase3 antibody, #74-104 anti-APP (C-terminal) antibody, #74-106 anti-APP (N-terminal) antibody, #74-108 anti-APP (C-terminal of the caspase 3- cleaved APP) antibody,

To be continued.

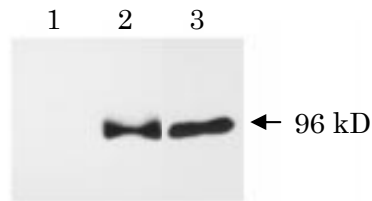


Fig.1 Western blot analysis of APP Δ C31 (ref.3).

Human NT2 neurons (neurally differentiated human NT2 embryonic carcinoma cells) were infected with adenovirus vector expressing β -galactosidase (lane 1), wild-type APP (lane 2) or APP Δ C31 (lane3). Cell lysates were prepared 48 h after infection, and proteins were analyzed by Western blotting using this antibody (SAC). Neurons overexpressing wild-type APP contained a 96 kD SAC-immunoreactive fragment which was also detected in APP Δ C31-overexpressing neurons.

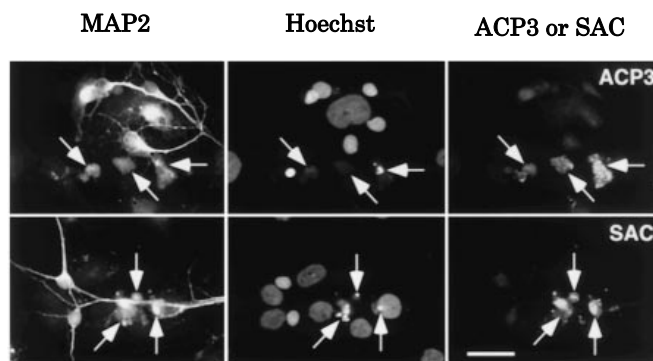


Fig.2 Immunocytochemical analysis of APP Δ C31: Caspase-3 activation and generation of the caspase-cleaved fragment APP Δ C31 within neurons induced by serum deprivation (ref.3).

Neurally differentiated NT2 cells were cultured for 96 h in the absence of fetal calf serum. Cells were triply labeled for MAP2, the neuronal marker microtubule-associated protein 2 (MAP2), chromosomal DNA (Hoechst), and activated caspase-3 (ACP3; upper panel) or APP Δ C31 (SAC; lower panel). MAP2-immunopositive neurons with apoptotic nuclei (arrows) are intensively immunostained with ACP3 and SAC.