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Product datasheet for AP31802PU-N

Amyloid beta (N-term) Chicken Polyclonal Antibody

Product data:

Product Type:	Primary Antibodies
Applications:	IF, IHC
Recommended Dilution:	**Immunocytochemistry.** **Immunohistochemistry.** *Recommended Dilutions:* 1/2000-1/5000. **Quality Control:** Antibodies were analyzed by Immunohistochemistry (at a concentration of 3 µg/ml) using Fluorescein-labeled Goat anti-Chicken IgY (1/500 dilution, Cat.-No AP31795FC-N) or HRP-labeled Goat anti-Chicken IgY (1/2000 dilution, Cat.-No AP31795HR-N) as the secondary reagent.
Reactivity:	Human, Mouse
Host:	Chicken
Isotype:	IgY
Clonality:	Polyclonal
Immunogen:	Hens were immunized with a synthetic peptide KLH conjugated corresponding to DAE FRH DSG YEV HHQ KL, residues 1-17 of the Amyloid beta-peptide (Amyloid Precursor Protein residues #672-688). After repeated injections, immune eggs were collected, and the IgY fractions were purified from the yolks.
Specificity:	Recognizes Beta-Amyloid Peptide (N-term).
Formulation:	10mM PBS, pH 7.2 containing 0.02% Sodium Azide as preservative. State: Aff - Purified State: Liquid purified (filter sterilized) IgY fraction.
Concentration:	lot specific
Purification:	Affinity Chromatography using a peptide column.
Conjugation:	Unconjugated
Storage:	Store the antibody undiluted in the dark at 2-8°C.
Stability:	Shelf life: one year from despatch.

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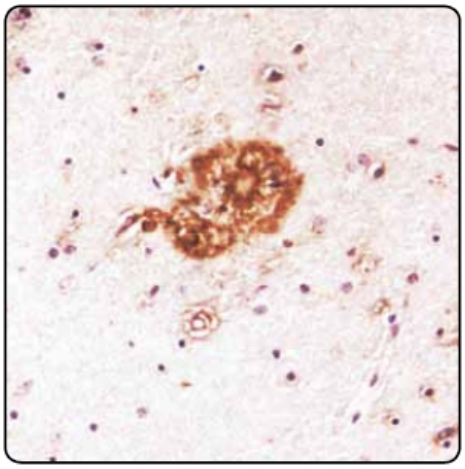
Background:

Beta-Amyloid peptide is a 40- or 42-amino acid fragment of the Human Beta Amyloid Precursor Protein (770 amino acids) produced by the proteolytic actions of Beta and Gamma-secretases. Both forms of Beta-amyloid peptide are rather insoluble and tend to self-aggregate into distinctive extracellular “plaques.” These plaques are evident in brains from patients with Alzheimer’s disease, as well as in brains from individuals with a history of traumatic head injuries. In the case of Alzheimer’s disease, it has been suggested that these extracellular Beta-amyloid peptide plaques are themselves cytotoxic (rather than simply being markers of brain pathology), and are responsible for the dendritic pruning and other neurodegenerative changes seen.

Product images:



Beta-Amyloid-positive neuritic plaque in cerebral cortex as seen in a post-mortum specimen taken from an Alzheimer's disease patient. Picture courtesy of Dr. Randy Woltjer, Oregon Health & Sciences University.