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Editorial

# Amyloid Precursor Protein Processing and Alzheimer's Disease

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## Abstract

Alzheimer's disease (AD), the leading cause of dementia worldwide, is characterized by the accumulation of the β-amyloid peptide (Aβ) within the brain along with hyperphosphorylated and cleaved forms. Accumulation of cerebral amyloid-beta peptide (Abeta) is essential for developing synaptic and cognitive deficits in Alzheimer's disease.

**Keywords:** Neurodegeneration; Dementia; BACE1; α-Secretase; γ-Secretase; Aging

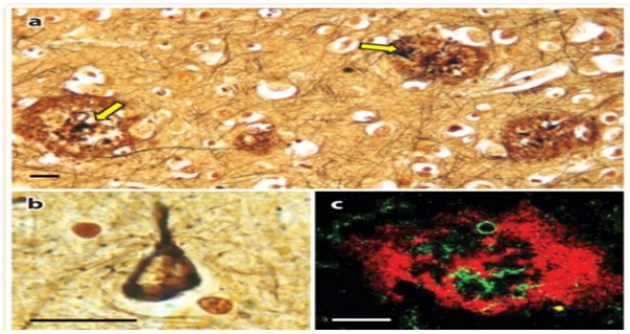


**Figure 1:** Dementia patients reports are taken in the given figures.

Pathology of Alzheimer's disease. (a, b) Brain cut sections of a patient with dementia [1] are stained with silver dye, revealed neuritic plaques observed in panel a andneurofibrillary tangle observed in panel b. The plaques in panel a consist of an amorphous reddish protein (Aβ) with dystrophic neurites (yellow arrows, dark black [2] material). (c) An Aβ plaque observed with an anti-Aβ antibody (red) showing infiltrating microglia stained with IBA1 antibody ( green) [3].

**Figure 2**

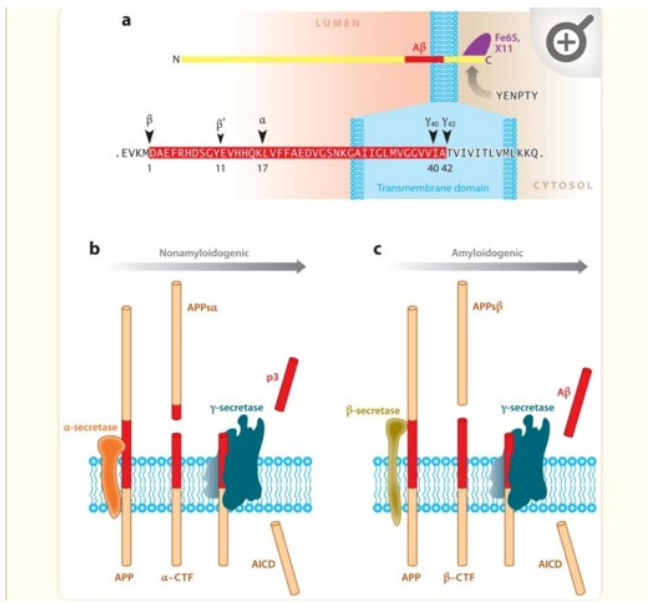


**Figure 2**

(a) The APP family of proteins are large, biologically active, N-terminal ectodomains and a shorter C-terminus that consist of a crucial Tyrosine–Glutamic Acid-Asparagine-Proline-Threonine-Tyrosine (YENPTY) protein-sorting domain to the adaptor proteins X11 and Fe65 bound. The Aβ peptide started within the [4] ectodomain and continued into the transmembrane region (red ). (b) Nonamyloidogenic processing of APP involved α-secretase followed by γ [5]-secretase is shown. (c) Amyloidogenic processing of APP involving BACE1 followed by γ-secretase is shown. Both processes generate soluble ectodomains (sAPPα and sAPPβ) and identical intracellular C-terminal fragments (AICD) [6].

**Figure 3**

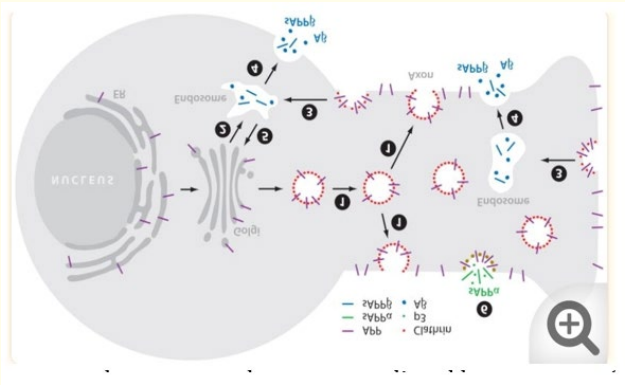

**Figure 3**

APP trafficking in neurons. Newly synthesize APP (purple) is transported from the Golgi down the axon (1) or into a cell body endosomal compartment (2). After inserting into the cell surface, some APP is cleaved by α-secretase (6) generated the sAPPα fragment, which diffused away (green), and some is reinternalize into endosomes (3), where Aβ is [7] generates (blue). Following proteolysis, the endosome recycles to the cell surface (4), releasing Aβ(blue) and sAPPβ. Transport from endosomes to the Golgi prior to APP cleavage can also occur, mediated by retromers (5).

## Discussion

In this research we discussed about the amylotropic protein synthesis and histological study and pathological study and also serum studies and neurological study and neurochemical studies

## Conclusion

Alzheimer's disease (AD), the leading cause of dementia worldwide, is characterized by the accumulation of the β-amyloid peptide (Aβ) within the brain along with hyperphosphorylated and cleaved forms.

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