Amyloid deposits, also called plaques, of Alzheimer's patients consist of several protein components like the amyloid beta-peptides (Abeta, Aβ) 1-40/42 and additional C- and N-terminally truncated and modified fragments. Very abundant are the isoaspartate (isoAsp)-Abeta and pyroglutamyl (pGlu)-Abeta peptides. The latter are formed by cyclization of the N-terminal glutamate at position 3 or 11 catalyzed by glutaminyl cyclase (QC) resulting in very amyloidogenic and neurotoxic variants of Abeta; Abeta-pE3 and Abeta-pE11.

In contrast to extracellular plaques that do not perfectly correlate with Alzheimer’s disease intraneuronal Abeta accumulation and vascular Abeta deposits have gained more and more evidence to be among the crucial factors responsible for progressive neuron loss.

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