Abundance of the longer A beta 42 in neocortical and cerebrovascular amyloid beta deposits in Swedish familial Alzheimer's disease and Down's syndrome.

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Abstract: Recent studies have demonstrated the deposition of amyloid beta (A beta) protein with carboxyl- and aminoterminal heterogeneity in cortical and cerebrovascular deposits of Alzheimer's disease (AD). Using carboxyl end-terminal specific antibodies to A beta peptides, we examined the immunocytochemical distribution of A beta 40 and A beta 42 species in brain tissue from a Swedish subject with familial AD (FAD) bearing the double mutation at codons 670/671 in the amyloid beta precursor protein (A beta PP), and from subjects with Down's syndrome and sporadic AD. In the Swedish subject, we found profound parenchymal A beta deposits and cerebral amyloid angiopathy in all four cortical lobes and cerebellum. A beta 42 was evident in almost all parenchymal deposits as well as many vascular deposits. Although A beta 40 was present in meningeal and intraparenchymal vessels, deposits containing this shorter peptide reactivity were sparse. Surprisingly, our observations in Swedish FAD showing a remarkable abundance of A beta 42 in both parenchymal and vascular deposits were qualitatively similar to the Down's syndrome and most sporadic AD cases, and to previously published A beta PP717 FAD. While previous transfection studies in different cell cultures indicate substantially increased soluble A beta production and A beta 40 species to be predominant, it would appear that the double A beta PP mutations in Swedish FAD largely result in the deposition of the longer A beta 42 in vivo.

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