Title: The Arctic APP mutation leads to Alzheimer’s disease pathology with highly variable topographic deposition of differentially truncated Aβ

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Suppl. Fig. 6 a-i: Immunostainings of Am1 patient’s cerebellum demonstrates the marked inter-individual variation despite the same genetic defect (cf. Fig. 5). Only abAβ_{x-42} and abAβ_{17-24} are clearly positive (a and c), whereas abAβ_{x-40} (b) and the more N-terminal abAβ_{8-17}, abAβ_{5-10} and abAβ_{1-5} (d-f) give virtually no parenchymal staining, even though the blood vessels are strongly positive. Weak staining with abAβ_{arc} (g) is consistent with most parenchymal deposits being composed of wild-type Aβ. A fair proportion of the deposited Aβ appears to have pyroglutamate N-termini (h and i). (bar in a 150 μm for all panels)