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. 9 Immunostainings of semiconsecutive sections from a PSIΔ9 AD patient’s frontal (a-h) and temporal (i and j) cortex. The cotton wool plaques are similarly rounded structures as Arctic plaques, but the different antibodies stain them homogeneously, although with different intensities. The strong staining with abAβx-42 (a) suggests that majority of Aβ terminates at aa 42, whereas Aβx-40 (b) species are scarce. N-termini appear to be markedly variable including considerable amounts of N-terminally truncated Aβ species starting with pyroglutamate (Aβ11pE or Aβ3pE; g and h). The homogeneous staining suggests that the variably truncated Aβ species are relatively evenly distributed within the plaques. i and j: Sections from temporal cortex show accumulation of hp-tau positive NTs within Aβ plaques (six plaques marked with numbers). Note the NTs (arrows) also in the subpial Aβ–positive edging (asterisks) (bar in a 100 μm for a-h; bar in i 150 μm for i and j)