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. 4 a-g: Aβ-plaques in Swe2 patient’s claustrum show similar targetoid pattern as in neocortex (a-c consecutive sections). a: With abAβx-42 dark corona and pale centre. b: With abAβx-40 fair staining of both centre and corona. c: With abAβx-1,5 dark centre and pale corona. d: Mid-domain abAβ17-24 stains strongly both centre and corona. e: Specific abAβarc gives similar pattern as abAβ17-24, though with much lesser intensity. f and g: Plaques comprise of both Aβ3pE and Aβ11pE, though less of the latter. h-k: Plaques in Sw2 patient’s putamen are small and diffusely stained. The most intense stainings are seen with abAβx-42, abAβarc and abAβ3pE (h, j and k) suggesting an abundance of Aβ with pyroglutamate-modified N-termini, which is consistent with the virtually negative abAβ1-5 staining (i). l: In Sw2 patient’s amygdala plaques are similar as in putamen but more numerous. m: In Sw2 patient’s thalamus the plaques are ragged and weakly stained. (bar in a 100 μm for a-c; bar in d 100 μm for d-g; bar in h 50 μm for h-l; bar in m 50 μm)