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

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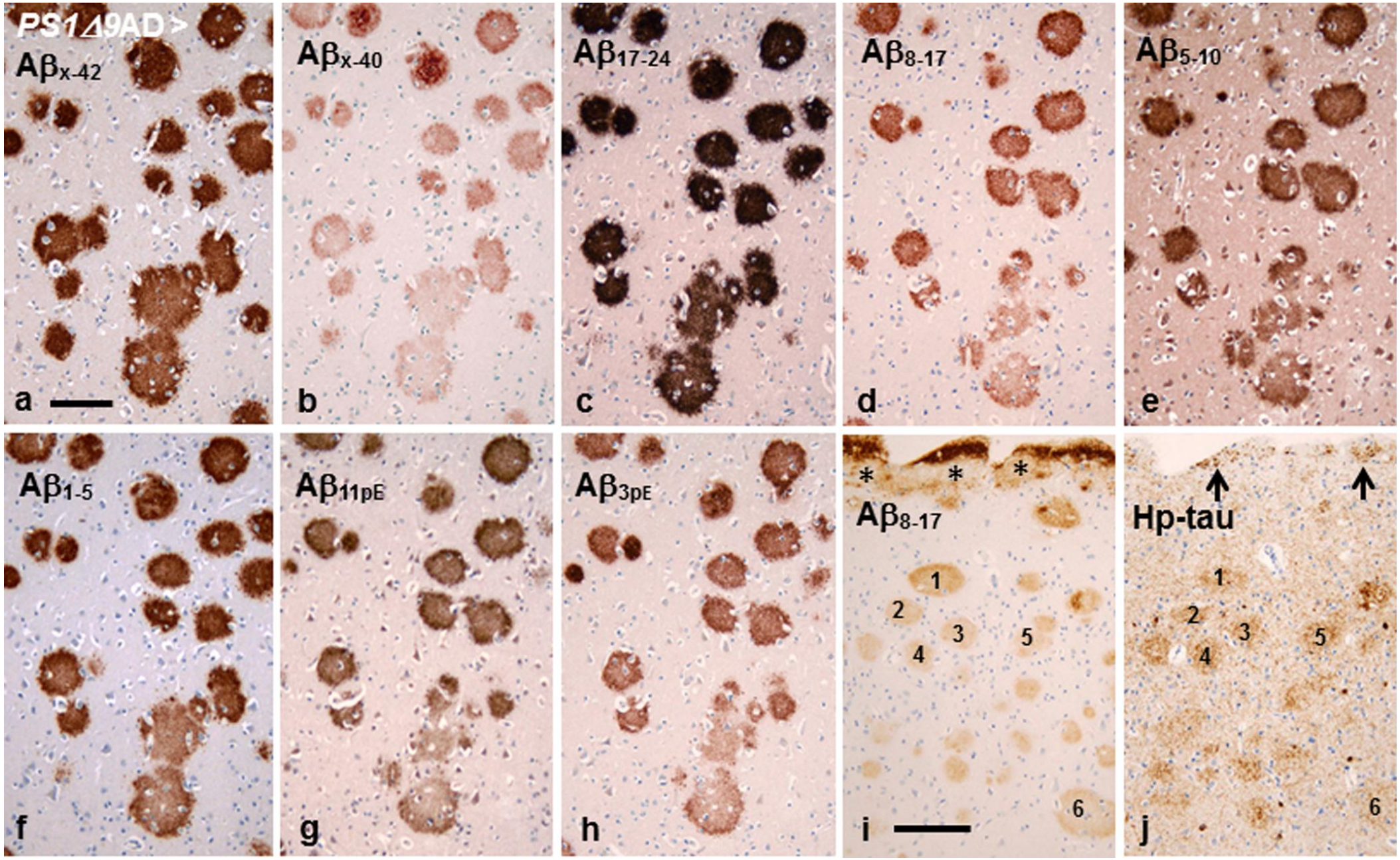
PS1Δ9AD >
Aβx-42
a
Aβx-40
b
Aβ17-24
c
Aβ8-17
d
Aβ5-10
e
Aβ1-5
f
Aβ11pE
g
Aβ3pE
h
*
*
*
Aβ8-17
1
2
3
4
5
6
i
Hp-tau
1
2
3
4
5
6
j

Suppl. Fig. 9 Immunostainings of semiconsecutive sections from a *PS1*Δ9 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 ab$A\beta_{x-42}$ (**a**) suggests that majority of Aβ terminates at aa 42, whereas $A\beta_{x-40}$ (**b**) species are scarce. N-termini appear to be markedly variable including considerable amounts of N-terminally truncated Aβ species starting with pyroglutamate ($A\beta_{11pE}$ or $A\beta_{3pE}$; **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**)