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

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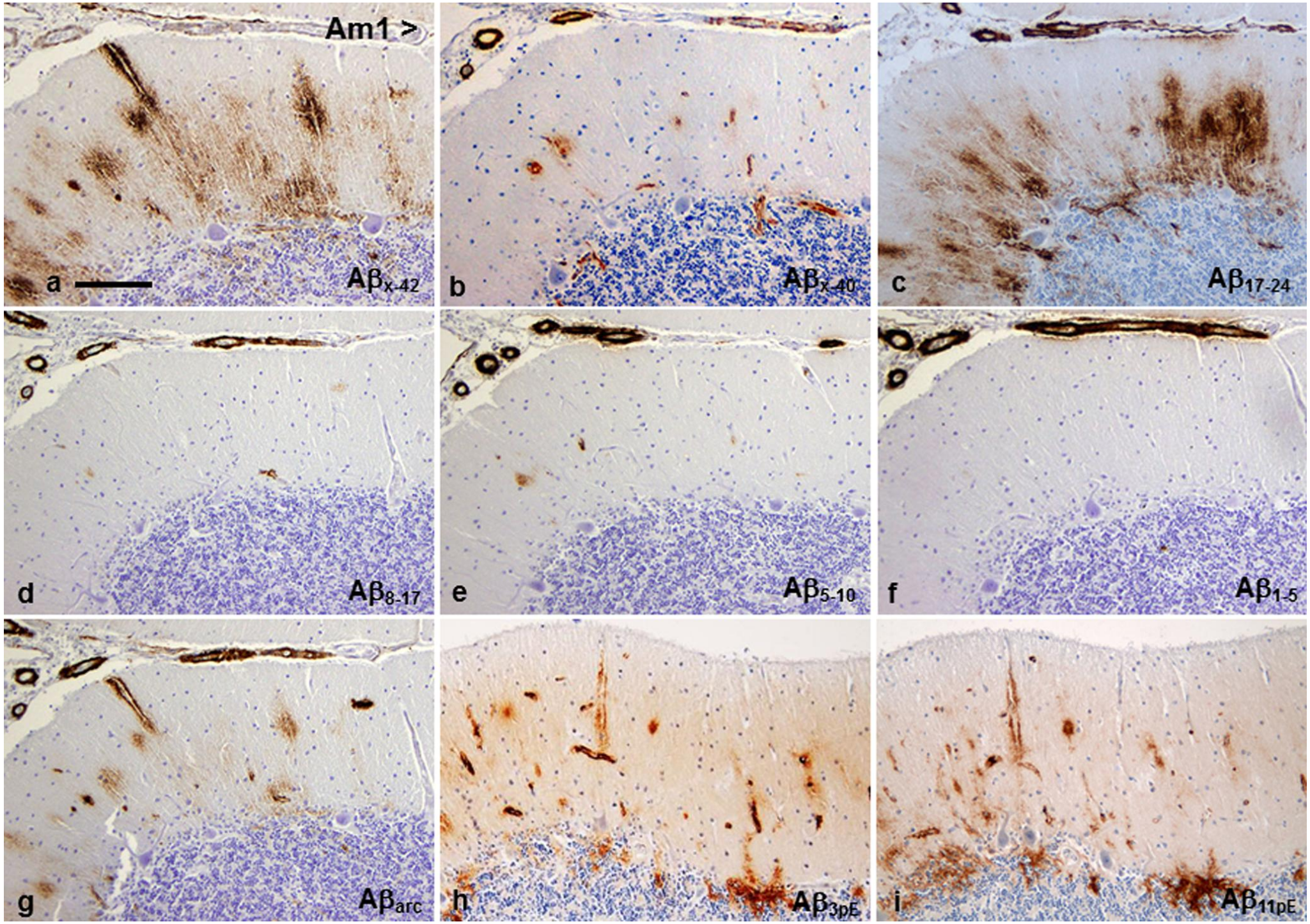
Am1 >
a
Aβx-42
b
Aβx-40
c
Aβ17-24
d
Aβ8-17
e
Aβ5-10
f
Aβ1-5
g
Aβarc
h
Aβ3pE
i
Aβ11pE

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)