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

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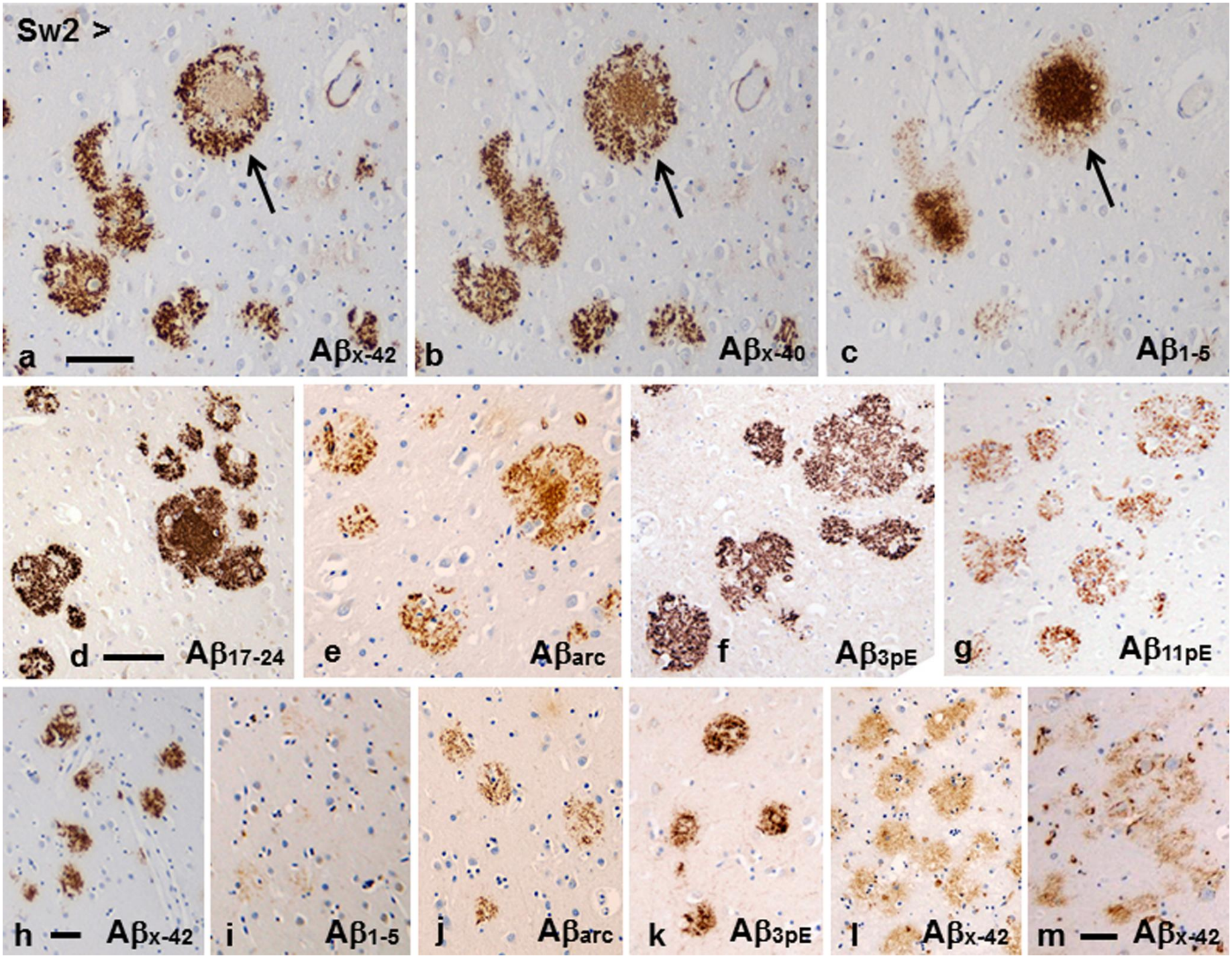
Authors: Hannu Kalimo[1], Maciej Lalowski, Nenad Bogdanovic, Ola Philipson, Thomas D. Bird , David Nochlin, Gerard D. Schellenberg, RoseMarie Brundin, Tommie Olofsson, Marc Baumann, Oliver Wirths, Thomas A. Bayer, Lars N.G. Nilsson, Hans Basun, Lars Lannfelt, Martin Ingelsson

Corresponding author: [1]Hannu Kalimo, Department of Pathology, University and University Hospital of Helsinki, Helsinki, Finland,

E-mail: hannu.kalimo@helsinki.fi


Sw2 >
a
Aβx-42
b
Aβx-40
c
Aβ1-5
d
Aβ17-24
e
Aβarc
f
Aβ3pE
g
Aβ11pE
h
Aβx-42
i
Aβ1-5
j
Aβarc
k
Aβ3pE
l
Aβx-42
m
Aβx-42

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 abAβ_{x-42} dark corona and pale centre**. b:** With abAβ_{x-40} fair staining of both centre and corona. **c:** With abAβ_{1-5} dark centre and pale corona. **d:** Mid-domain abAβ_{17-24} stains strongly both centre and corona. **e:** Specific abAβ_{arc}. gives similar pattern as abAβ_{17-24}, though with much lesser intensity. **f** and **g:** Plaques comprise of both Aβ_{3pE} and Aβ_{11pE}, 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 abAβ_{x-42}, abAβ_{arc} and abAβ_{3pE} (**h, j** and **k**) suggesting an abundance of Aβ with pyroglutamate-modified N-termini, which is consistent with the virtually negative abAβ_{1-5} 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)