Supplementary information

Figure S1. Generation of Ube3a-maternal deficient AD mice. Ube3a-maternal deficient AD mice (AD/Ube3a (m-/p+) were generated by crossing Ube3a-maternal deficient females Ube3a(m-/p+) with AD transgenic males (APPswe/PSEN1δE9). Genotypes were confirmed by PCR using primer specific for human APPswe/PSEN1δE9 and mouse *Ube3a* gene (A). B) Immunoblot analysis of Ube3a and transgenic human APP/PS1. Cortical samples from 4 months old mice of various genotypes were used for immunoblot analysis. Note the absence of Ube3a expression in maternally Ube3a-null wild-type WT/Ube3a(m-/p+) or AD mice (AD/Ube3a(m-/p+) and presence of APPswe/PSEN1δE9 transgenes in AD and AD/Ube3a(m-/p+) mice.

Figure S2. A) Ube3a-deficient AD mice exhibits significant decrease in brain weight at their 12 months of age. Values are mean ± SD of 5 animals in each genotypes. *$p < 0.05$ when compared to wild type, Ube3a-maternal deficient and AD mice groups (one-way ANOVA). B) Representative immunohistochemical staining of GFAP. C) TUNEL staining. DNase1 treated section was used as positive control. Brain sections obtained from 3 different mice in each genotypes at their age of 12 months were used for both staining. Cortical region was shown in both the staining.

Figure S3. Representative immunohistochemical staining of Aβ plaques in the brain section obtained from AD and Ube3a-maternal deficient AD mice of different age. Brain sections were stained with 6E10 antibody. Scale bar; 500μm.

Figure S1

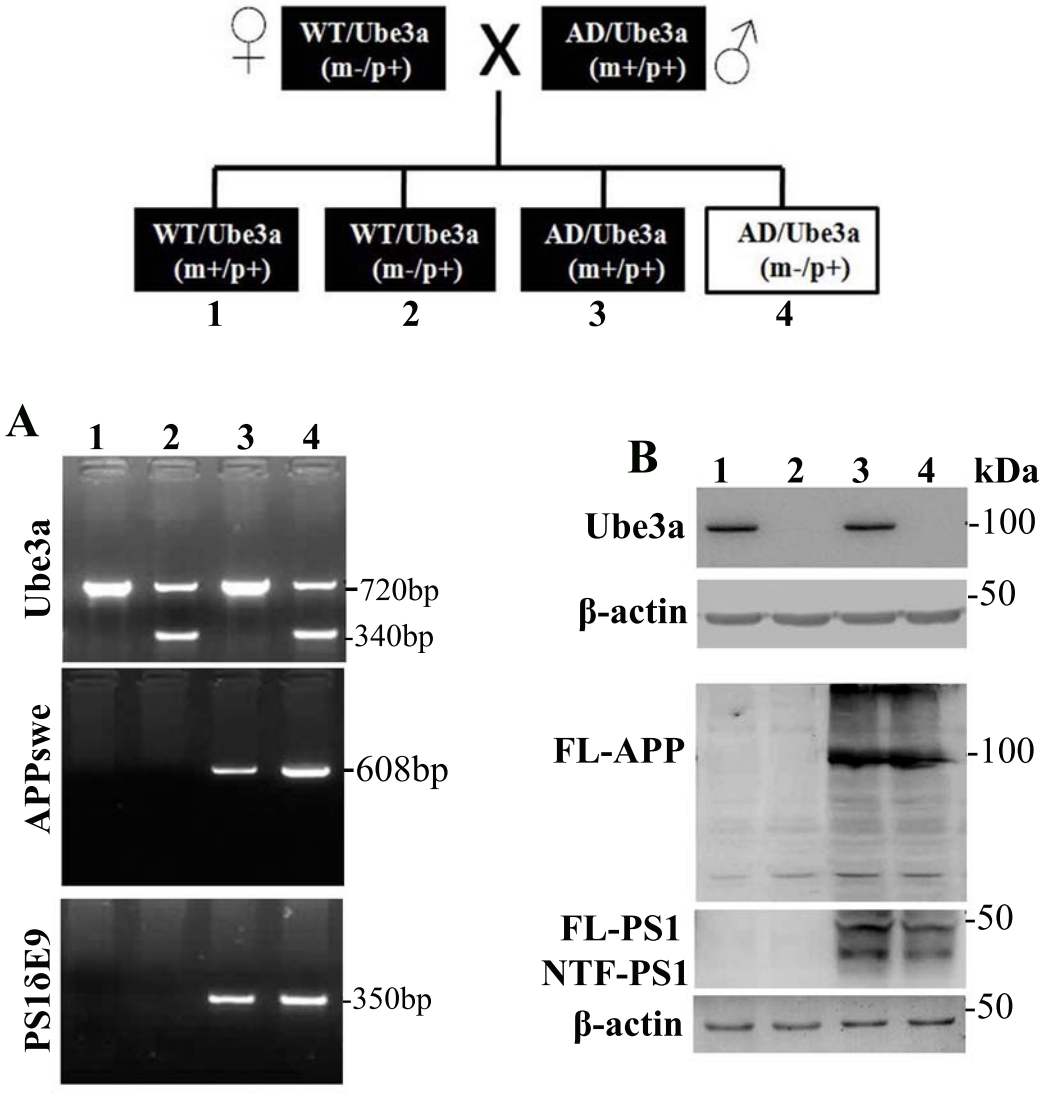
♀
WT/Ube3a
(m-/p+)
X
AD/Ube3a
(m+/p+)
♂
WT/Ube3a
(m+/p+)
1
WT/Ube3a
(m-/p+)
2
AD/Ube3a
(m+/p+)
3
AD/Ube3a
(m-/p+)
4
A
1 2 3 4
Ube3a
720bp
340bp
APPswe
608bp
PS1δE9
350bp
B
1 2 3 4 kDa
Ube3a
100
β-actin
50
FL-APP
100
FL-PS1
NTF-PS1
50
β-actin
50

Figure S2

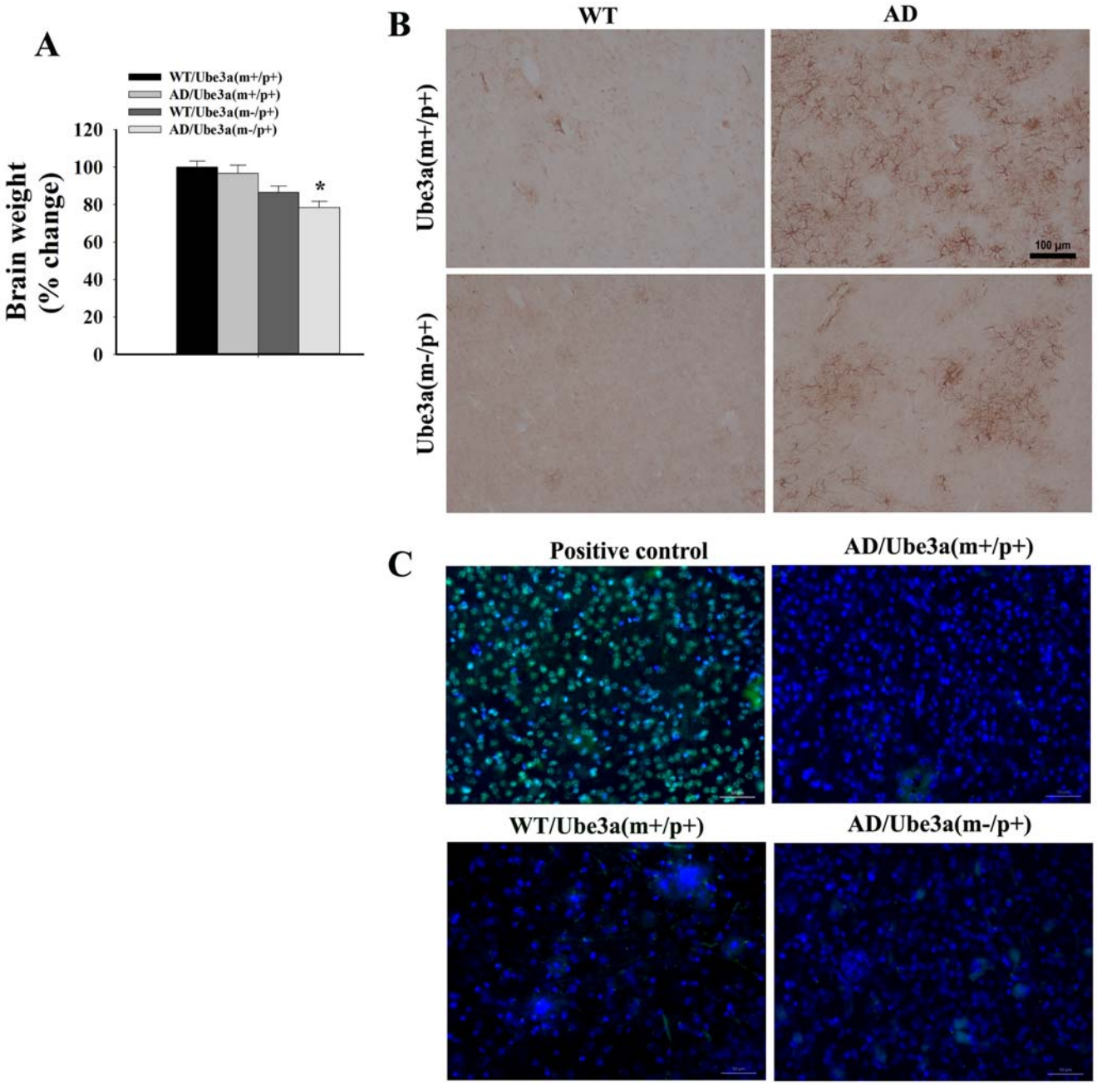
A
WT/Ube3a(m+/p+)
AD/Ube3a(m+/p+)
WT/Ube3a(m-/p+)
AD/Ube3a(m-/p+)
Brain weight (% change)
120
100
80
60
40
20
0
*
B
WT
AD
Ube3a(m+/p+)
Ube3a(m-/p+)
100 μm
C
Positive control
AD/Ube3a(m+/p+)
WT/Ube3a(m+/p+)
AD/Ube3a(m-/p+)

Figure S3