Supplementary Table S1: Transgenic mice models of Amyloid Pathology

Model	Mutation	Model Background	Aβ Pathology	Disadvantages	References
Tg2576	APP KM670/671NL (Swedish)	C57BL/6 x SJL	Plaques by 11-13 months	Overexpression of multiple APP fragments Random integration of transgenes Sudden death may occur (Tg2576) Aβ accumulation is preceded by cognitive impairment Genetic backgrounds mixed in some cases	27, 33-35, 168
PDAPP	APP V717F (Indiana)	C57BL6 x DBA2	Plaques at 6 months		27,28,29,31,168
J20	APP K670_M671delinsNL (Swedish), APP V717F (Indiana)	C57BL/6	Plaques by 5-7 months		26, 45-48,168
TgCRND8	APP K670_M671delinsNL (Swedish), APP V717F (Indiana)	Hybrid C3H/He-C57BL/6	Plaques by 3 months		49-52, 168

5xFAD (B6SJL)	APP KM670/671NL (Swedish), APP I716V (Florida), APP V717I (London), PSEN1 M146L, PSEN1 L286V	C57BL/6 x SJL	Plaques by 1.5 months	Overexpression of multiple APP fragments No perfect negative control Multiple mutations (APP+PS1) Occurrence of non-specific ER stress No NFTs Cross breeding may be complicated in some cases Random integration of transgenes	26,27,168
5xFAD (C57BL6)	APP KM670/671NL (Swedish), APP I716V (Florida), APP V717I (London), PSEN1 M146L, PSEN1 L286V	C57BL6	Plaques by 1.5-2 months		59,60,62,58
APPxPS1	APP K670_M671delinsNL (Swedish) and PSEN1 L166P	C57BL/6	Plaques by 1.5 months (cortex) and 3-4 months (hippocampus)		63-67
TREM2- BAC x 5xFAD	APP KM670/671NL (Swedish), APP I716V (Florida), APP V717I (London), PSEN1 M146L, PSEN1 L286V	TREM2-BAC: FVB/NJ; 5xFAD: C57BL/6 x SJL	Plaques by 8.5 months	No NFT or neuronal loss	26,69-72
AppNL-F	APP KM670/671NL (Swedish), APP I716F (Iberian)	C57BL/6	Plaques by 6 months	No NFT or neuronal loss Multiple FAD mutations of APP Overproduction of CTF-β Genomic homozygosity	26,68,168
APPV717I	APP V717I (London)	C57BL/6 x FVB/N	Plaques by 10 months	No NFT observed	43,44

Tg-SwDI	APP K670_M671delinsNL (Swedish), APP E693Q (Dutch), APP D694N (Iowa)	C57BL/6	Plaques by 3 months	No NFT observed	26,53,54

Abbreviations: Aβ (amyloid-beta); NFT (Neurofibrillary Tangles); APP (Amyloid Precursor Protein); FAD (familial Alzheimer's Disease); CTF-β (C-terminal fragment beta); ER (Endoplasmic Reticulum)

Supplementary Table S2: Transgenic mice model of Tau pathology

Model	Mutation	Model Background	Tau Pathology	Disadvantages	References
JNPL3	MAPT P301L	C57BL/6, DBA/2, SW mixed background	NFTs develop from 4.5-6.5 months	Absence of amyloid pathology	82-85
hTau	M.A.P.T.: knock out; M.A.P.T.: Transgenic	Cross between SW and B6D2F1	Hyperphosphorylated tau at 6 months	Absence of amyloid pathology	88-90,232
rTg (tau P301L)	MAPT P301L	Produced by crossing 129S6 (activator) to F.V.B. (responder)	Pretangles at 2.5 months. Argyrophilic tangle-like inclusions in the cortex by 4 months and in the hippocampus by 5.5 months	No association found between genetic mutation and tau pathology in AD pateints Overexpression of mutant human tau may attribute to	26,88,89,97,232

				behavioral phenotypes and neuropathological deficits. No amyloid pathology	
PS19 (tau P301S)-	MAPT P301S	(C57BL/6 x C3H)F1	NFTs develop at 6 months	No association found between genetic mutation and tau pathology in AD pateints No amyloid pathology	26,100,232

Abbreviations: NFT (Neurofibrillary Tangles); MAPT (Microtubule Associated Protein Tau); AD (Alzheimer's Disease)