SUPPLEMENTAL FIGURE LEGENDS

Supplemental Figure 1. 3 μM $A\beta_{40}$ was incubated with equimolar concentration of lipid-free human ApoA-I for 5 days as described in the text and $A\beta_{40}$ aggregation examined by WBs for Aβ. WB was performed with 6E10 antibody.

Supplemental Figure 2. ApoA-I decreases Aβ-induced apoptosis. A. Primary neurons were treated for 48 hr with 25 μM Aβ42 plus/minus 2.5 μM human ApoA-I (10:1 ratio of Aβ:ApoA-I) and cell death determined by Hoechst staining. Prior to the treatment Aβ42 was pre-incubated with human ApoA-I or vehicle for 72 hours. Arrows point to apoptotic nuclei. **B.** The graph shows the quantification of the results. The data are the result of two experiments in triplicate. Analysis by *t*-test.

Supplemental Figure 3. WB for total soluble Aβ was performed on 4-12% NU PAGE gels followed by WB with 6E10 antibody. APP/PS1/wt, n=11; APP/PS1/ko, n=10. Note: on the upper pannel the samples from APP/PS1 mice were separated by a sample from non-transgenic mouse as a negative control.

Supplemental Figure 4. The deletion of ApoA-I does not affect protein levels of Abca1 and ApoE in wt mice. **A.** WB for Abca1 was performed on RIPA extracted brain proteins as described in the text. The asterisk in Abca1ko WB points to a non-specific band. **B.** WB for ApoE was performed on TBS extracted brain proteins. wt/Abca1ko and wt/ApoEko mice were used as controls. **C.** Graphical representation of the difference in intensity/amount of ApoE. N=5-6 per group.

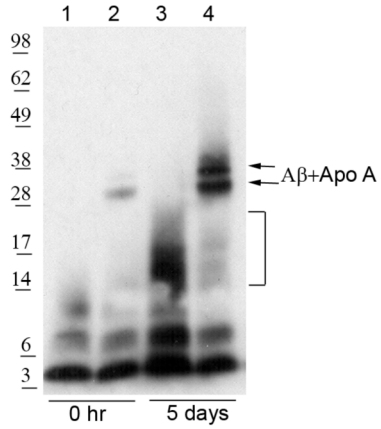
Supplemental Figure 5. Lack of ApoA-I does not affect amyloid plaque load regardless of the gender. **A** and **B**, Brain sections were stained with X-34 to visualize fibrillar amyloid plaques from 12 mo old APP/PS/wt and APP/PS/ko mice. Graphical representation of % area of hippocampus (**A**) and cortex (**B**) covered by X-34 positive deposits (% X-34 load). APP/PS1/wt, n=8 males and 6 females; APP/PS1/ko, n=7 males and 5 females.

Supplemental Figure 6. Lack of ApoA-I does not affect astrogliosis in APP/PS1 mice. Brain sections were stained with anti GFAP antibody to visualize astrocytes in the brains of 12 mo old APP/PS/wt and APP/PS/ko mice. Graphical representation of % area of hippocampus (**A**) and cortex (**B**) covered by GFAP positive deposits (% area). APP/PS1/wt, n=15; APP/PS1/ko, n=13. Note that there was no gender-dependent difference.

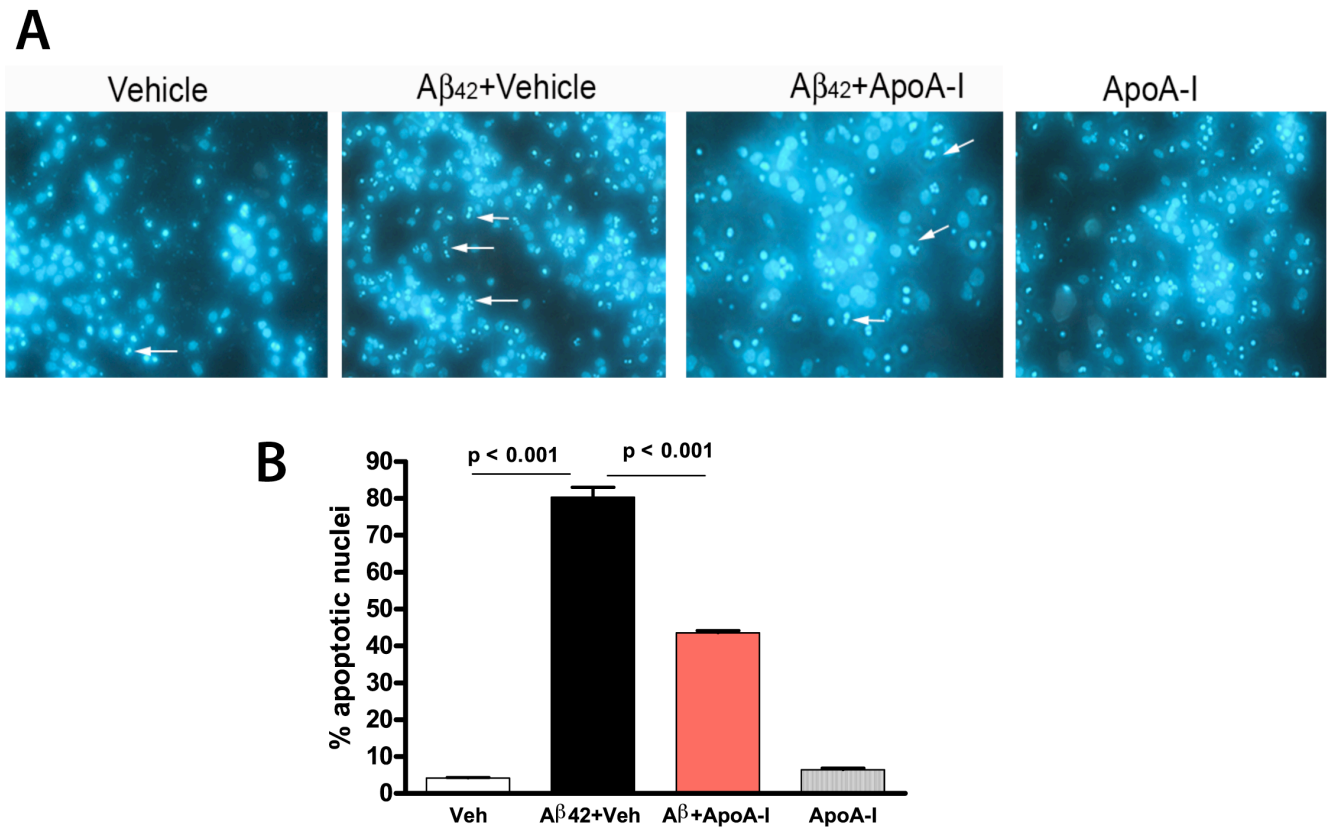
Supplemental Figure 7. Lack of gender-dependent effect on CAA in APP/PS1 mice. **A** and **B**, Insoluble Aβ was extracted from cortices and hippocampi of 12 mo old APP/PS1/wt and APP/PS1/ko mice and measured by ELISA as shown on Figure 8. APP/PS1/wt n=4 males and females; APP/PS1/ko, n=5 males and females. **C.** Amyloid deposits in cerebral blood vessels (cortex and hippocampus) were evaluated using X-34 staining as described for Figure 8D. APP/PS1/wt, n=8 males and 6 females; APP/PS1/ko n=7 males and 5 females.

Supplemental Figure 8. Lack of ApoA-I increases insoluble Aβtotal level in 6 and 16 month old mice. Blood vessels were isolated from cortices and hippocampi of APP/PS/ko and APP/PS/wt mice and insoluble Aβ extracted using formic acid. The level of Aβtotal represents the sum of $A\beta_{40}$ and $A\beta_{42}$ levels as measured by ELISA. The data are presented as fold of Aβtotal levels in APP/PS1/wt mice. Note that the difference between 16 months old is statistically insignificant (N.S.). For 6 moth old mice, N=6 mice per group and for 16 month old mice, N=3-6 mice per group.

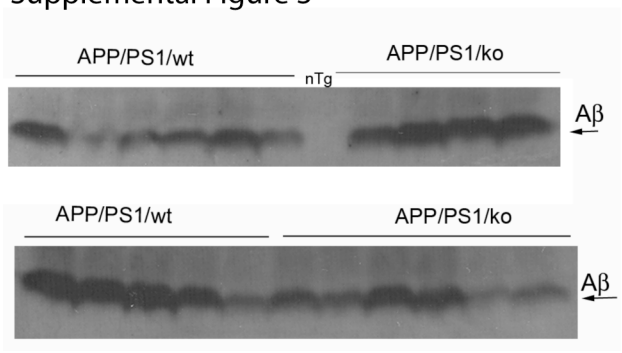
Supplemental Figure 1



Supplemental Figure 2



Supplemental Figure 3



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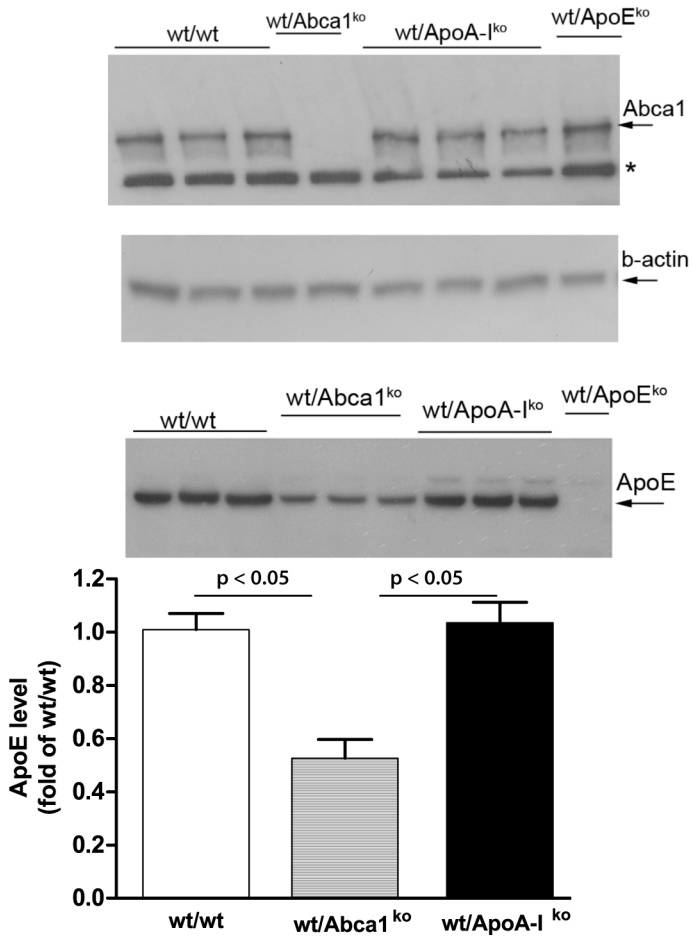

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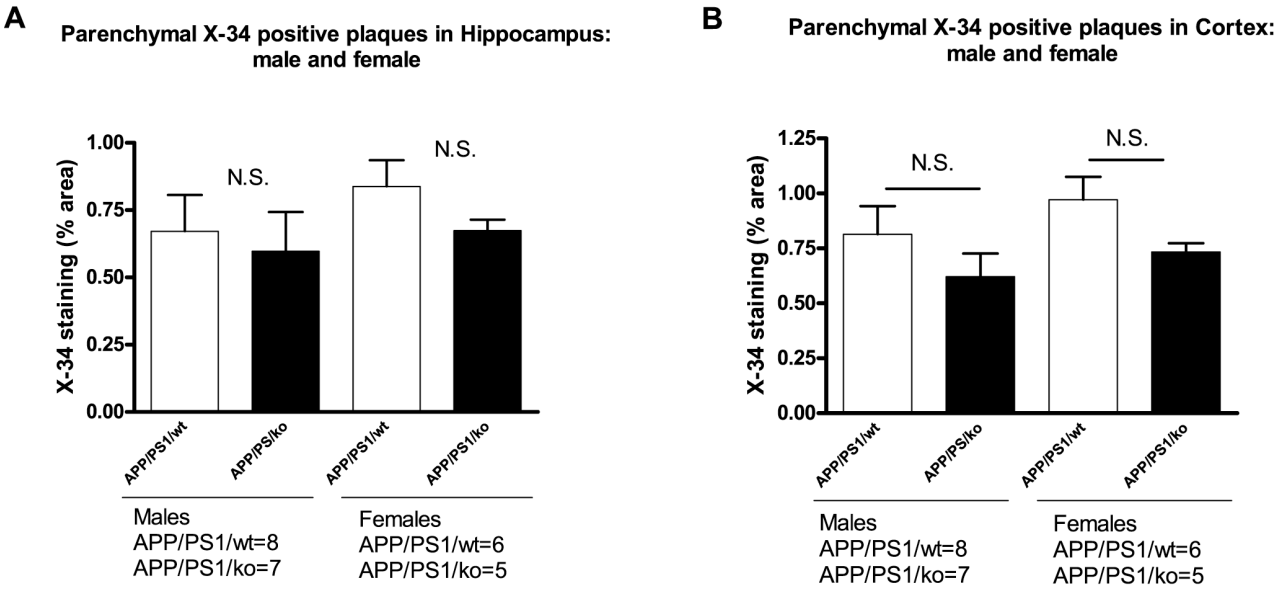

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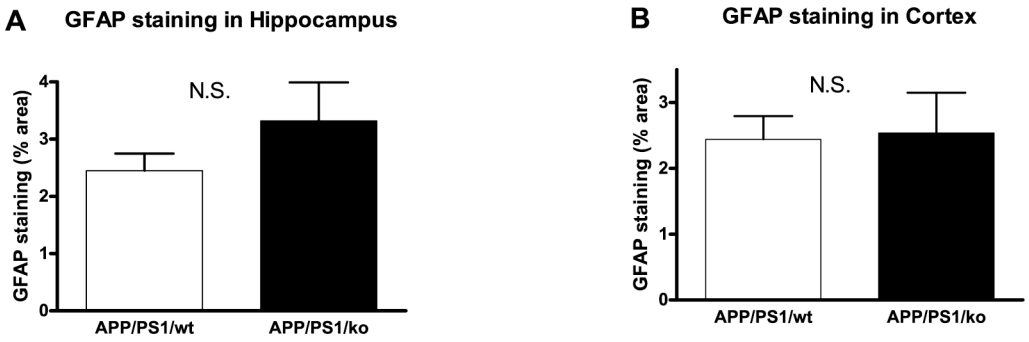
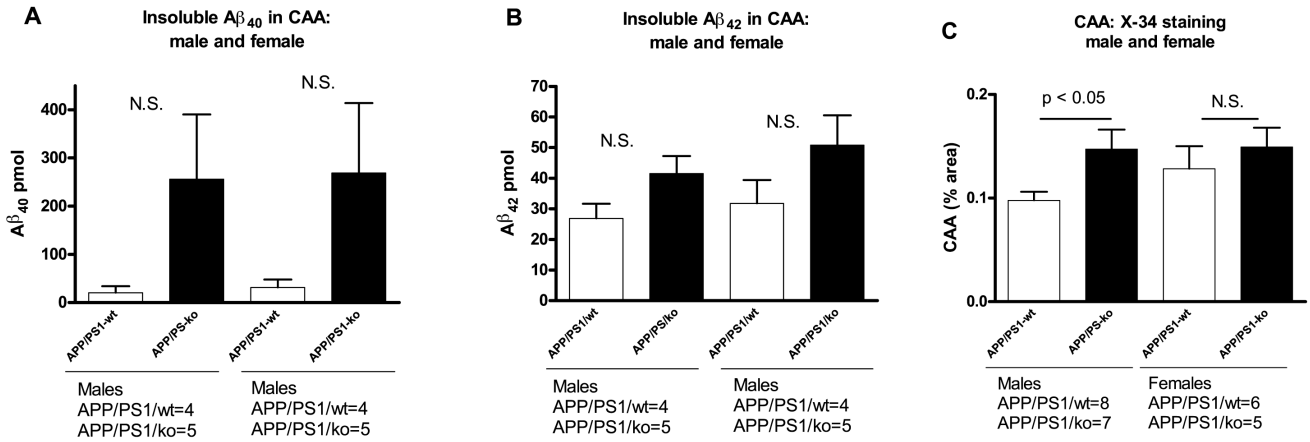


Supplemental Figure 7



Supplemental Figure 8