Supplemental Information

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against pericyte loss and cognitive
deficits in Alzheimer's disease

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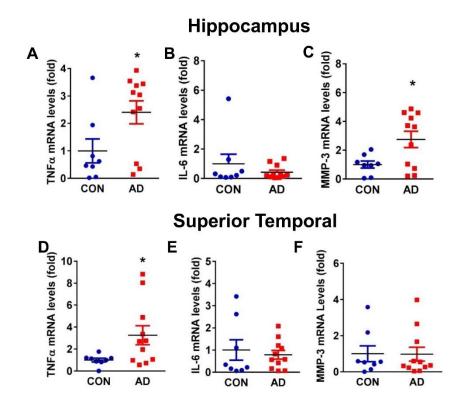


Figure S1. Expression of $TNF\alpha$, IL-6 and MMP3 in AD patients. RNAs were isolated from 100 mg brain hippocampal tissue or superior temporal tissue of human donors with AD (N = 11) and cognitively normal controls (N = 8). The expression levels of $TNF\alpha$, IL-6 and MMP3 were determined by RT-PCR in the (A-C) hippocampus and (D-F) superior temporal. Data are expressed as mean \pm standard error of the mean. *P < 0.05 compared to CON group. CON: control; AD: Alzheimer's disease.

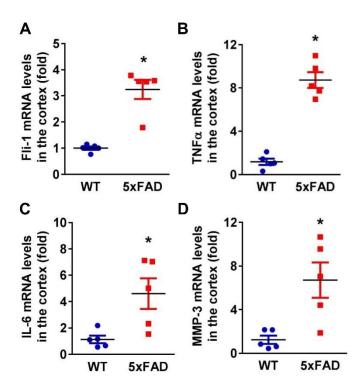


Figure S2. Increased *Fli-1*, *TNFa*, *IL-6* and *MMP3* in the cortex of 5xFAD mice. The cortex was isolated from WT and 5xFAD mice at 6.5 months of age. (A) *Fli-1* mRNA levels were determined by RT-PCR. N=5 mice/group. Expression levels of (B) TNFa, (C) IL-6 and (D) MMP3 were determined by RT-PCR. N=5 mice/group. Data are expressed as mean \pm standard error of the mean. *P < 0.05 compared to WT group.

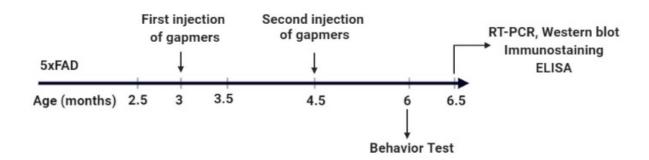


Figure S3. The experimental design *in vivo*. The *in vivo* experimental design including the timeline of intrahippocampal injection of Control or Fli-1 Gapmers, behavioral test, RT-PCR, western blot, immunostaining and ELISA.

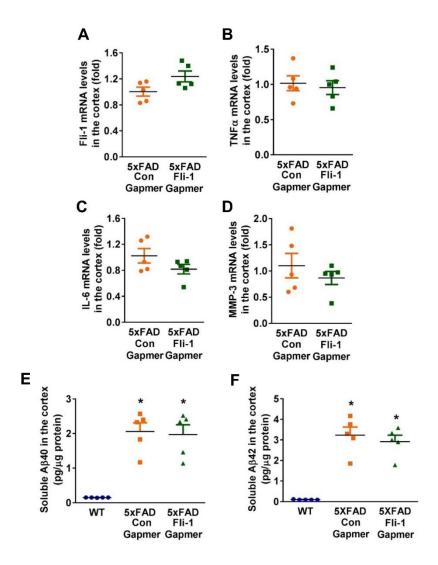


Figure. S4. Intrahippocampal injection of Fli-1 Gapmer did not affect expression levels of *Fli-1*, *TNFα*, *IL-6* and *MMP3*, and Aβ levels in the cortex of 5xFAD mice. Fli-1 or control Gapmers were injected into both sides of the hippocampus of 5xFAD mice at 3 and 4.5 months of age. The cortex was isolated from WT and 5xFAD mice treated with control or Fli-1 gapmers at 6.5 months of age. (A) *Fli-1*, (B) *TNFα*, (C) *IL-6* and (D) *MMP3* levels were determined by RT-PCR. (E-F) Soluble human Aβ40 and Aβ42 levels in the cortex was detected by ELISA. N=5 mice/group. Data are expressed as mean \pm standard error of the mean. *P < 0.05 compared to WT group.