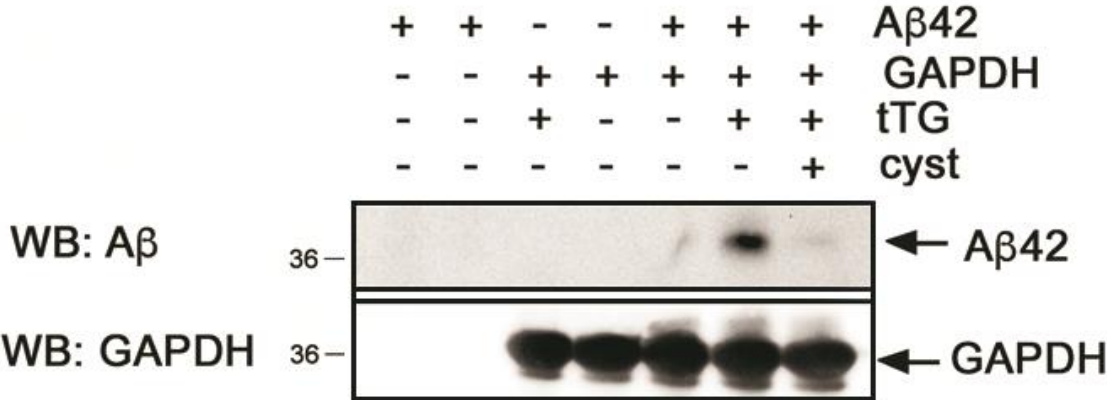


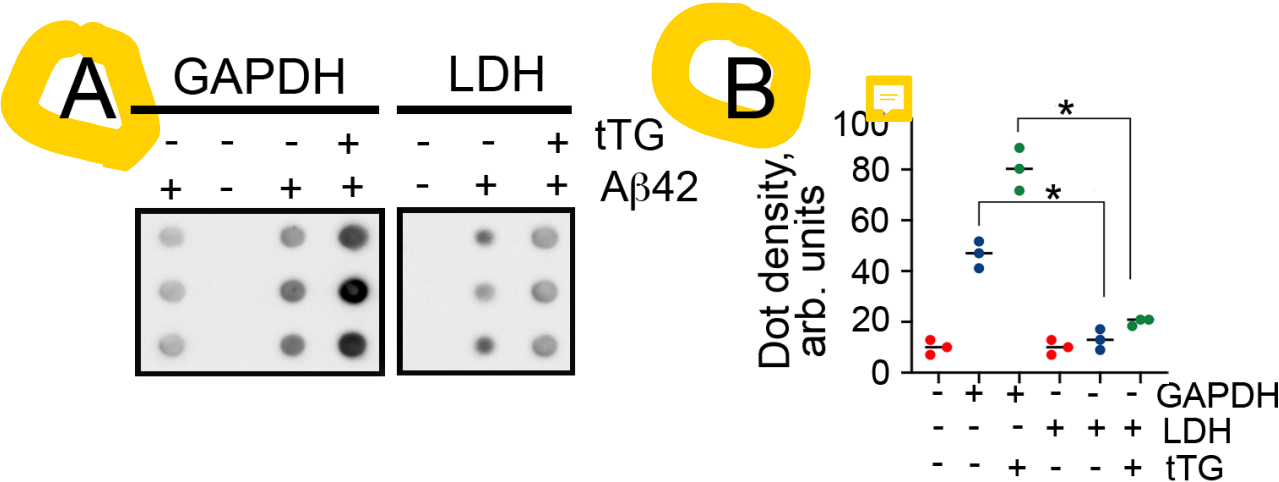
Extracellular GAPDH Promotes Alzheimer Disease Progression by Enhancing Amyloid- β Aggregation and Cytotoxicity

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SUPPLEMENTARY DATA

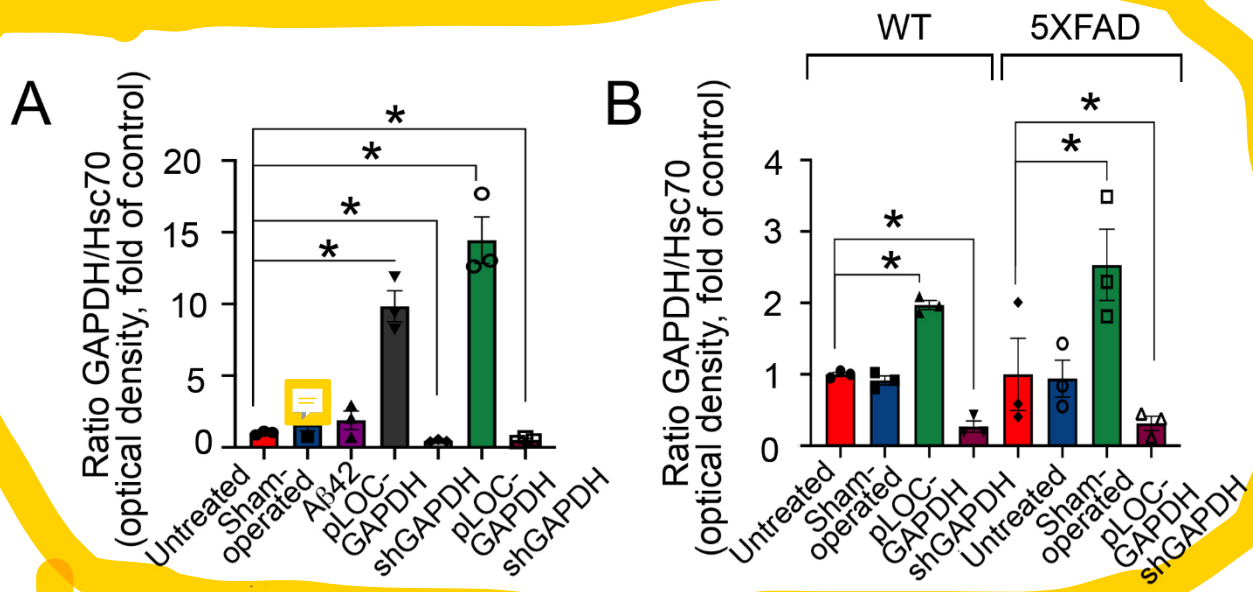


Supplementary Figure 1. GAPDH and Aβ42 form SDS insoluble complex in the presence of tTG.



Supplementary Figure 2. An arbitrary cytosolic protein (LDH) released from dying cells does not form insoluble aggregates with Aβ.

SUPPLEMENTARY DATA



Supplementary Figure 3. GAPDH accumulation in hippocampus promotes neurodegeneration in murine models of AD.

Supplementary Table 1. Cohort of MCI and AD patients used for GAPDH detection in CSF.

#	Stage of Disease	Number of patients in group	Average age	MMSE, range
1	MCI	n=22; 12 males, 10 females	69,4±2,5	26-30
2	Mild AD	n=41; 22 males, 19 females	75,5±1,1	21-25
3	Moderate AD	n=49; 26 males, 23 females	75,6±0,9	12-20
4	Severe AD	n=51; 31 males; 20 females	74,8±3,5	0-11

Supplementary Table 2. Cohort of MCI and AD disease patients used for detecting Aβ-GAPDH aggregates in CSF.

#	Stage of Disease	Number of patients in group	Average age	MMSE, range
1	MCI	n=6; 4 males, 2 females	71,5±2,5	26-28
2	Mild AD	n=8; 6 males, 2 females	76±2,2	21-25
3	Moderate AD	n=7; 3 males, 4 females	78,3±2,4	12-20
4	Severe AD	n=6; 3 males; 3 females	74,8±3,5	0-11