**SUPPORTING INFORMATION**

# Amyloid-β regulates gap junction protein Connexin 43 trafficking in cultured primary astrocytes

Mahua Maulik[1*], Lakshmy Vasan[1,a], Abhishek Bose[1], Saikat Dutta Chowdhury[1], Neelanjana Sengupta[1], Jayasri Das Sarma[1]

Supplementary Figures S1, S2

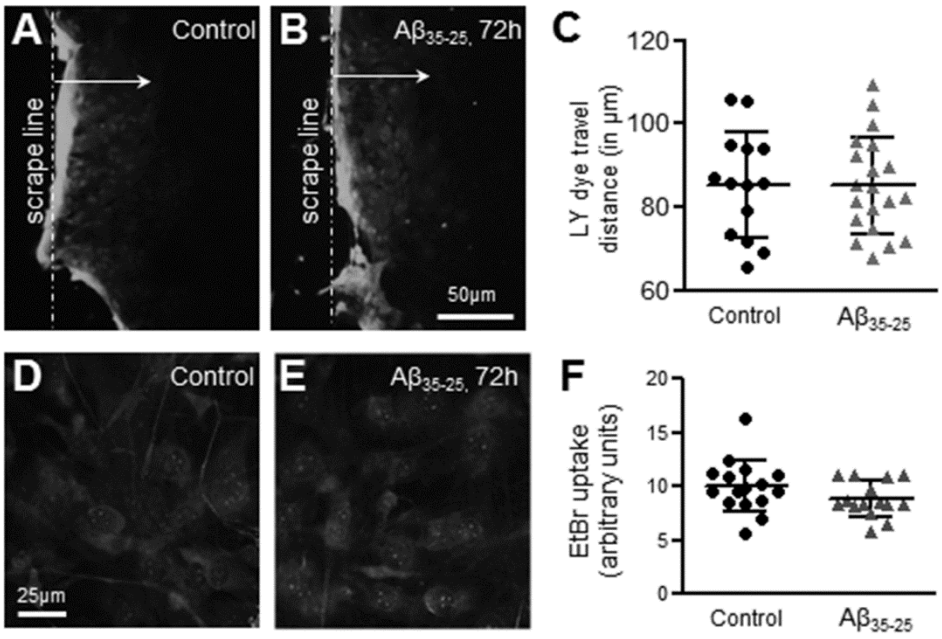


**Figure S1: Intercellular gap junction coupling and hemichannel activity in control and $A\beta_{35-25}$ reverse peptide treated mouse primary astrocytes. (A-C)** Representative photomicrographs and scatter plot showing the lucifer yellow dye spread from the scrape loading line in control (A) and $A\beta_{35-25}$ reverse peptide treated (B; 10μM, 72h) mouse primary astrocyte cultures. **(D-F)** Representative photomicrographs and scatter plot illustrating no significant difference in EtBr dye uptake between control (D) and $A\beta_{35-25}$ reverse peptide treated (F; 10μM, 72h) mouse primary astrocytes. Error bars denote the mean±SD of five to ten fields from two independent cultures.

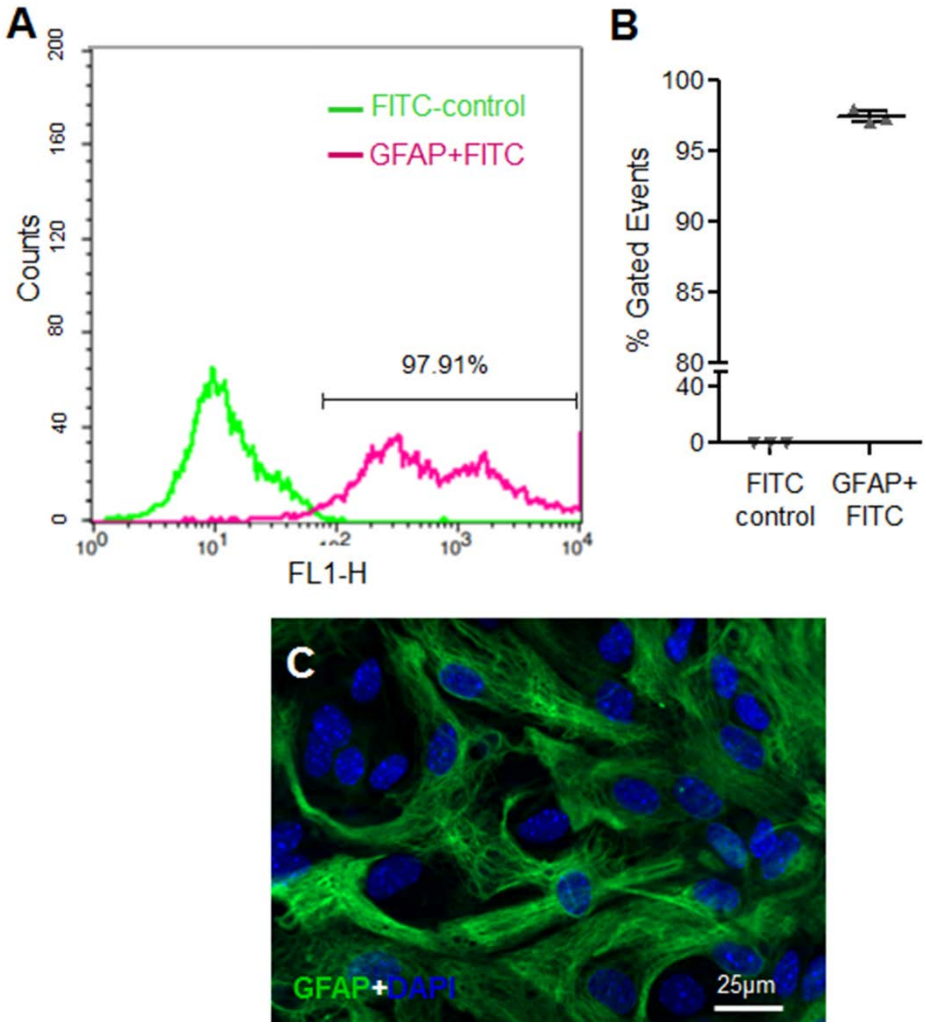


**Figure S2: Characterization of astrocyte-enriched cultures. (A,B)** Representative histogram and scatter plot of flow cytometric analysis of GFAP expressing cells in the established primary astrocyte cultures used in the present study. (C) Representative immunofluorescence image showing GFAP expression in the astrocyte enriched cultures. Values represent mean±SD from three independent experiments. ***p<0.0001.