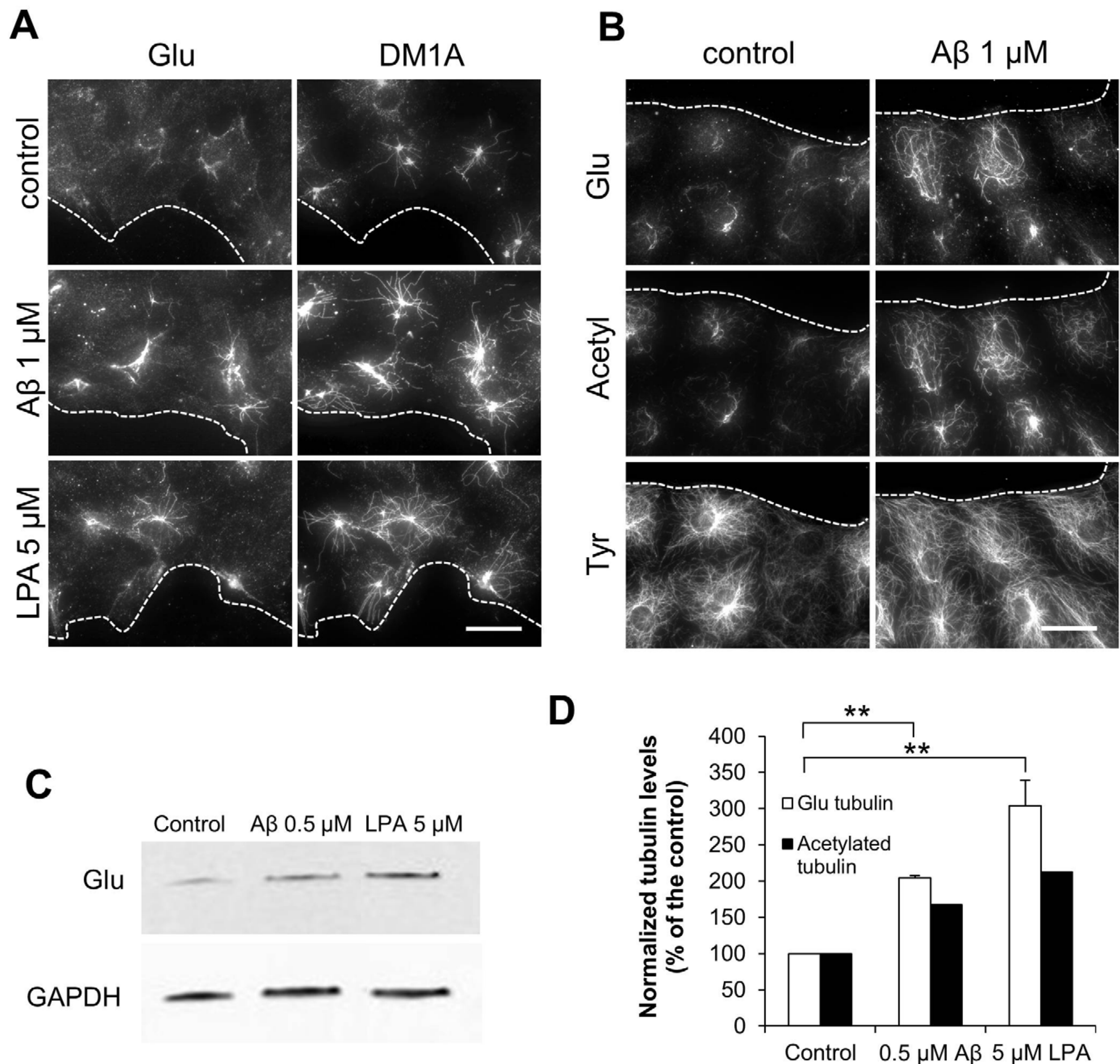
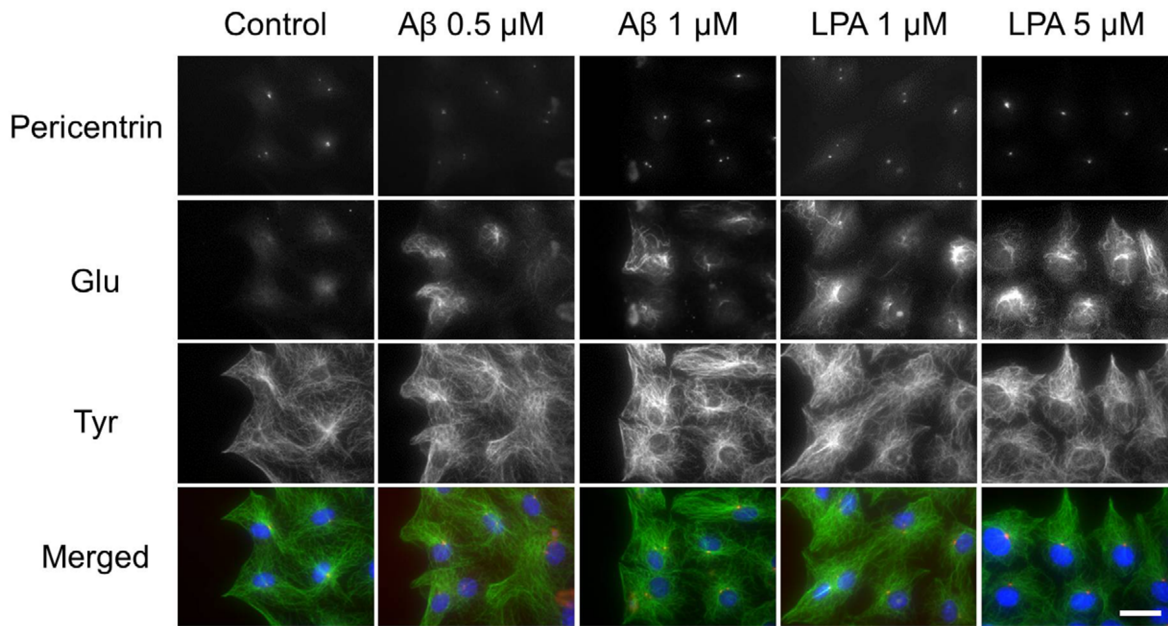


Fig. S1. Glu MTs induced by Aβ are stable. (A) Immunostaining of detyrosinated (Glu) and pan-tubulin (DM1A) in serum-starved cells at the wound treated with Aβ (2 h) and Nocodazole (1 h) prior to brief permeabilization with saponin before fixation. (B) Immunostaining of detyrosinated (Glu), tyrosinated (Tyr) and acetylated (Acetyl) tubulin in cells at the wound treated with Aβ for 2 h. (C) Western blot analysis of whole cell lysates from serum-starved NIH3T3 cells treated with Aβ or LPA prior to harvesting. Detyrosinated tubulin was detected by the Glu antibody and an antibody specific for GAPDH was used as a loading marker. (D) Quantification of the levels of detyrosinated (Glu) or acetylated tubulin normalized against GAPDH levels. In the case of detyrosinated tubulin, the data are mean $\pm$ SEM from three independent experiments. ** $p<0.01$ by t-test. Bars, 20 mm.



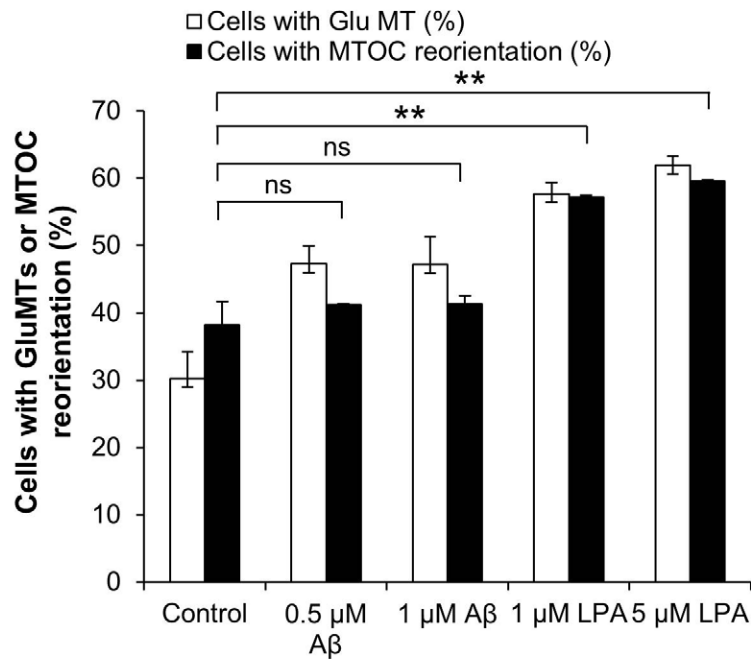


Fig. S2. Aβ does not induce reorientation of MTOC in wounded monolayers. (A) Immunostaining of detyrosinated (Glu), tyrosinated (Tyr) tubulin and pericentrin to localize the MTOC in starved cells at the wound treated with either Aβ or LPA at the indicated concentrations. (B) Quantification of the percentage of cells treated as in (A) with detyrosinated (Glu) MTs or reoriented MTOC. Data are mean ± SEM from three independent experiments (n>100 per sample in each experiment).** $p<0.01$ by Chi square test. Bar, 20 mm.

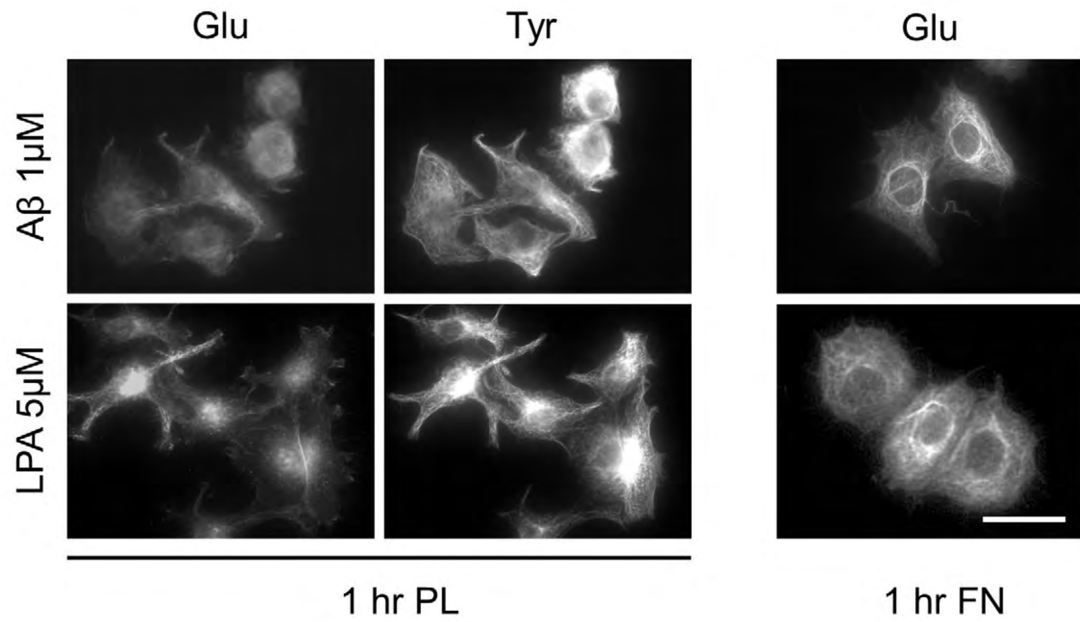


Fig. S3. Immunostaining of detyrosinated (Glu) and tyrosinated (Tyr) MTs in cells spreading on polylysine (PL) or fibronectin (FN) for 1 h while treated with Aβ or LPA. Bar, 20 mm.

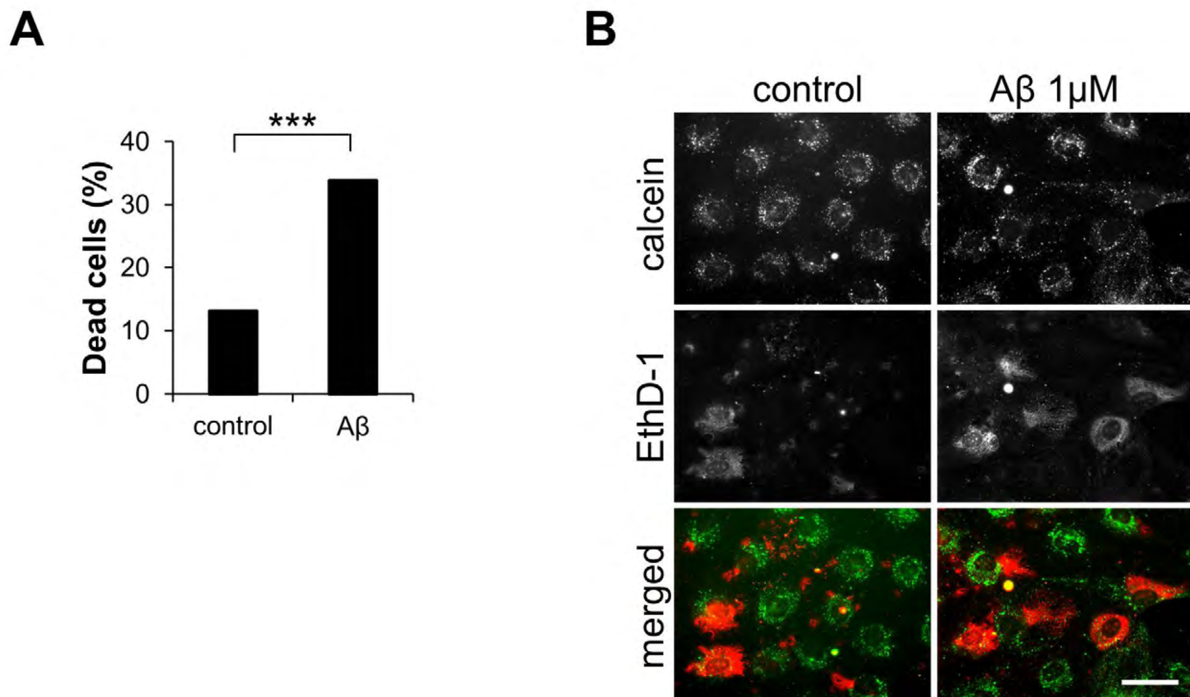


Fig. S4. Aβ induces cell death in NIH3T3 cells. (A) Quantification of the percentage of dead cells by calcein/EthD-1 fluorescence in cells treated with Aβ (1 μM) for 24 h prior to live-labeling and fixation. (B) Fluorescence images of cells treated as in (A). *** $p<0.001$ by Chi square test. Bar, 20 mm.