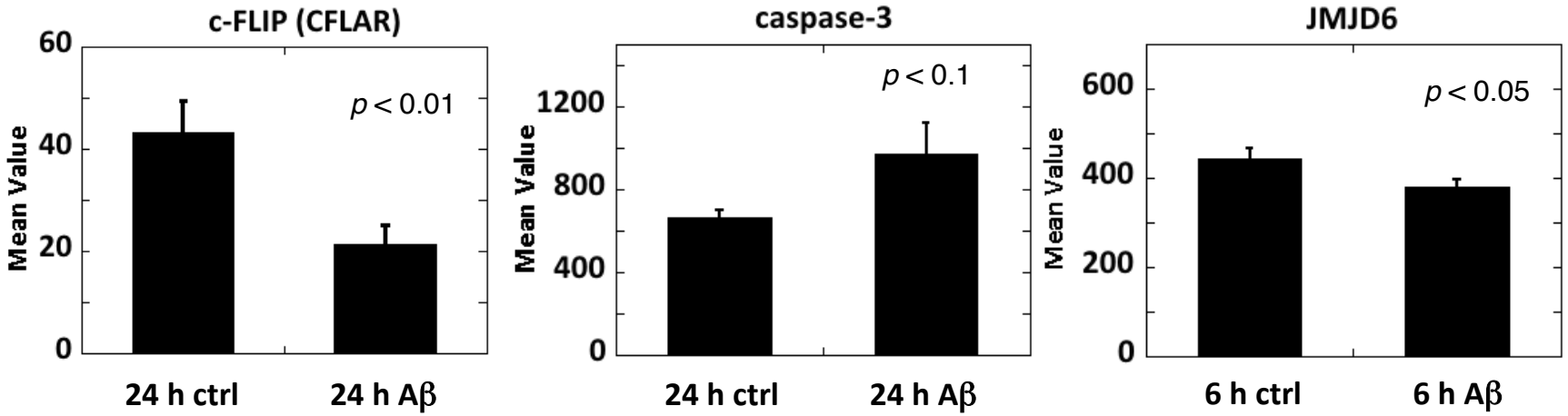
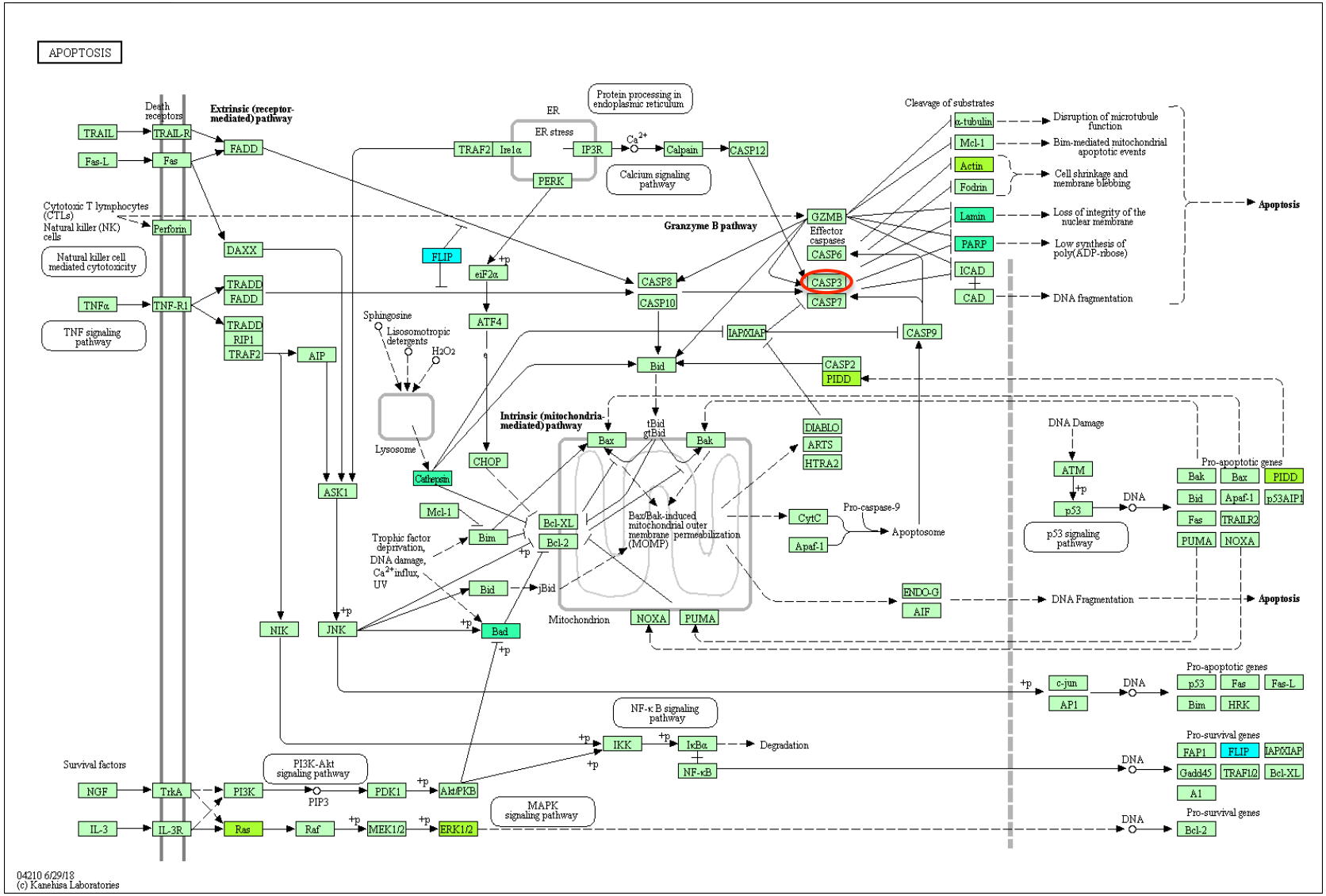
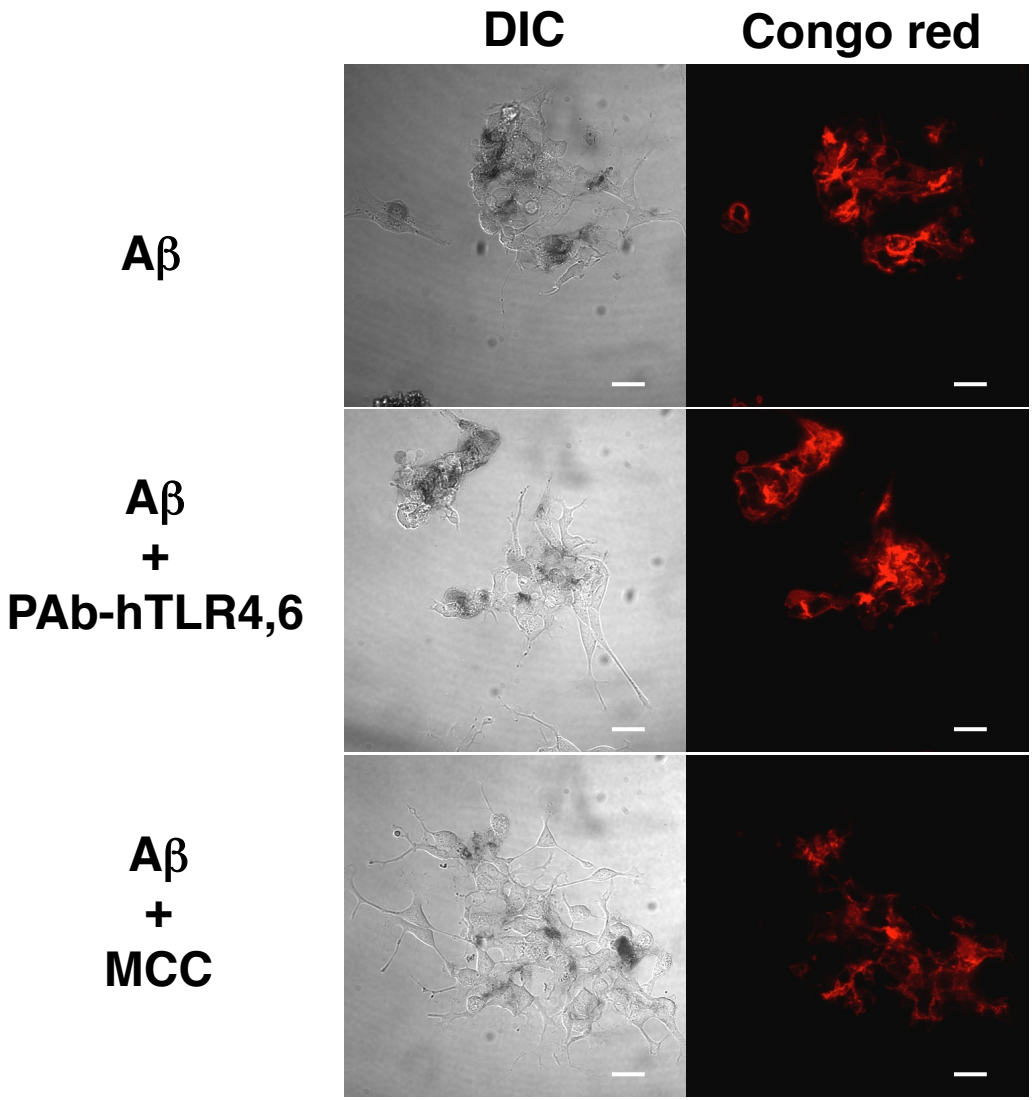


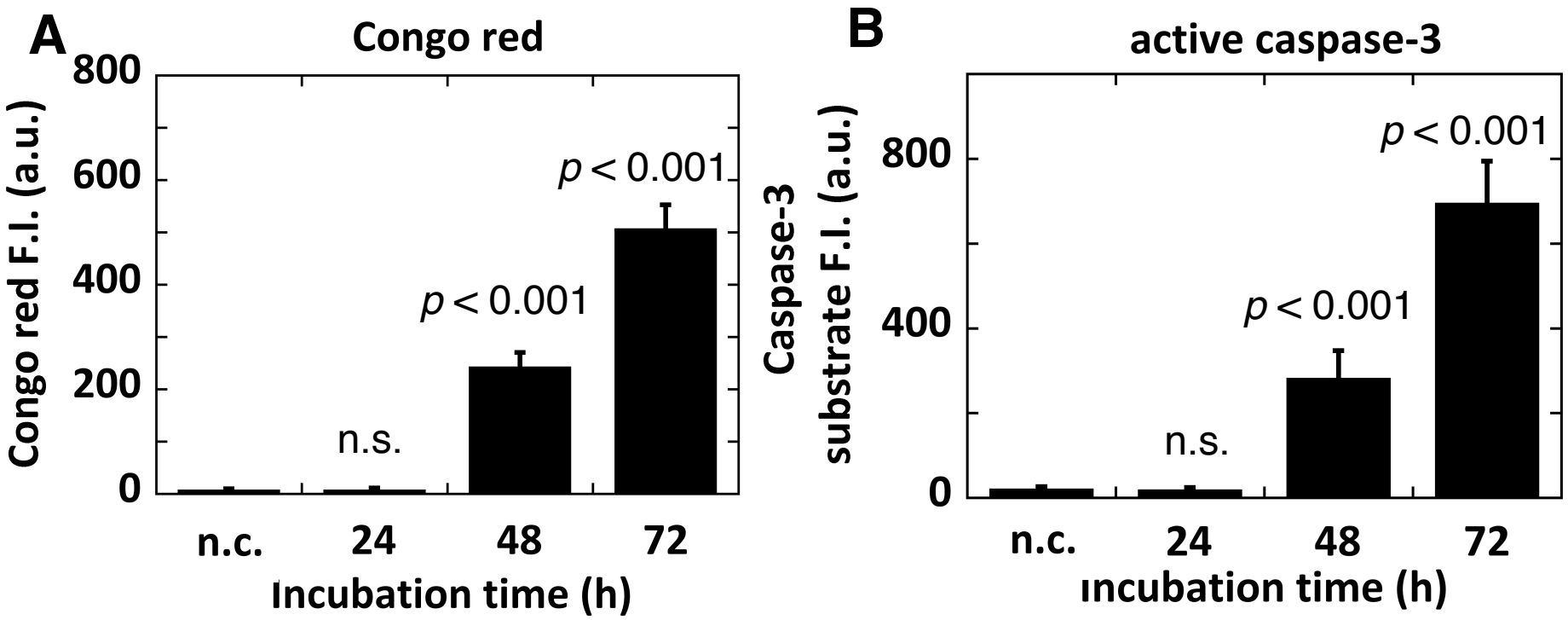
Supporting Figure S1 Selected examples of alterations in gene expression induced by Aβ. Aβ-(1–42) (5 μM) was incubated with the cells for 24 h (c-FLIP (Probe Set ID: 210564_x_at) and caspase-3 (Probe Set ID: 202763_at)) or 6 h (JMJD6 (Probe Set ID: 212723_at)). Differences from the control without Aβ were evaluated by the two-tailed Student's *t*-test.



Supporting Figure S2 KEGG pathway map related to apoptosis. Blue color indicates significant decrease of gene expression compared with the control ($p < 0.01$). Caspase-3 is highlighted by the red circle.



Supporting Figure S3 Fibril formation under treatment with inflammation inhibitors.
Aβ fibrils were detected with Congo red (n = 5, representative images are presented). Upper: Aβ only (5 μM, 24h); Middle: Aβ plus neutralizing antibodies for TLR4 and TLR6 (5 μg mL^{-1} each); Lower: Aβ plus MCC950 (10 μM).



Supporting Figure S4 Correlation between fibril formation of Aβ-(1–40) and apoptosis. Time-dependent increases in Congo red staining (A) and active caspase-3 (B) upon treatment with Aβ-(1–40) . SH-SY5Y cells were treated with 50 μM monomeric Aβ-(1–40) for the indicated periods. The fluorescence intensity of cells was quantified from fluorescence images obtained with a confocal microscope (mean ± S. E. (n = 100)). Differences from the negative control (n.c.) were evaluated by the two-tailed Student's t-test. No significant increase in caspase activity was observed after 24-h incubation, whereas significant caspase activities were observed after 48- and 72-h incubations concomitant with amyloid deposition.