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Supplementary Data

Focal Adhesion Kinase Activates NF-κB via the ERK1/2 and p38MAPK Pathways in Amyloid-$\beta_{25\text{-}35}$-Induced Apoptosis in PC12 Cells

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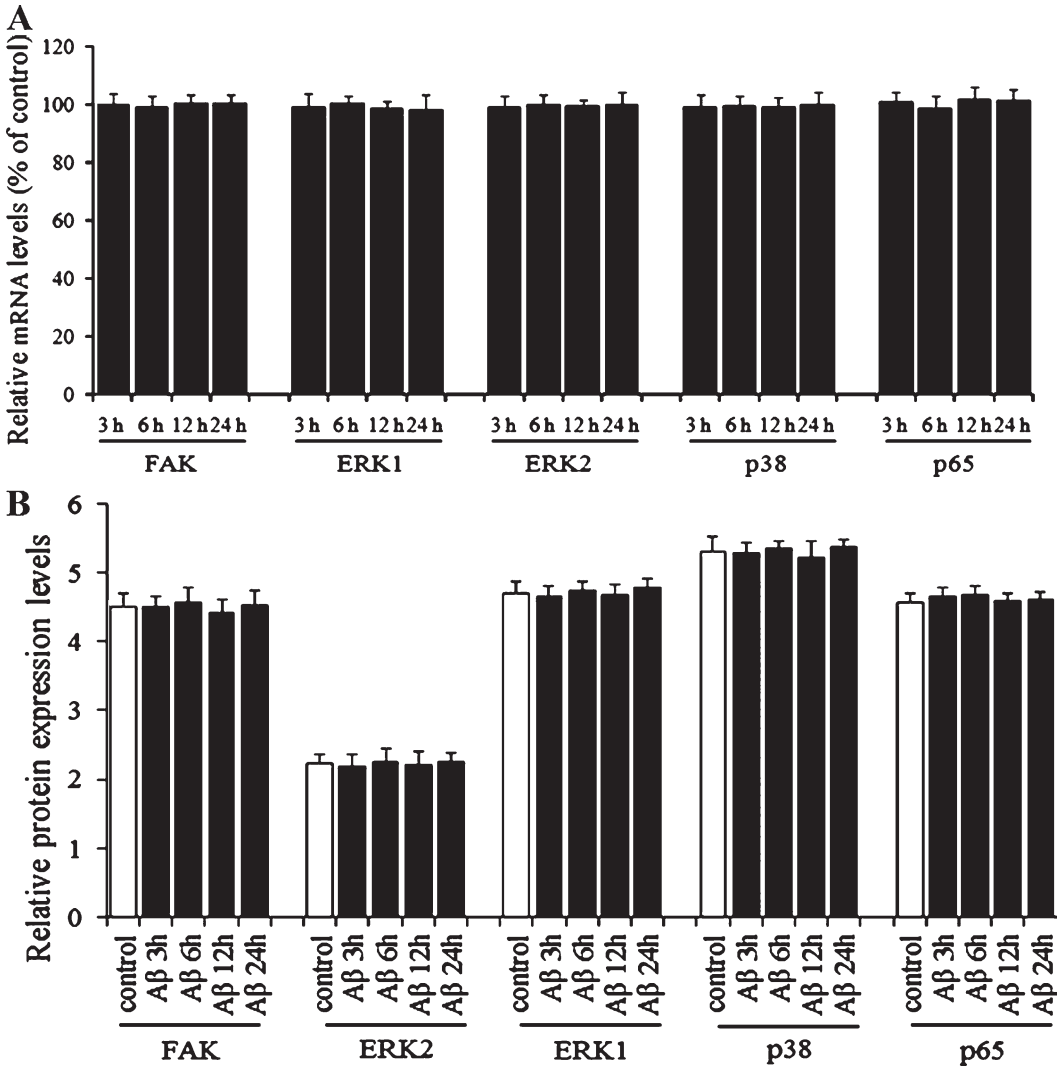

Supplementary Figure 1. Data analysis to Fig. 4C, D. A) Quantities of mRNAs of FAK, ERK1/2, p38MAPK, and NF-κB p65 were assessed by RT-PCR. GAPDH is a marker for whole mRNAs. The statistical analysis result showed that mRNA levels of FAK, ERK1/2, p38MAPK, and NF-κB p65 did not significantly change during Aβ-induced apoptosis in PC12 cells. B) Statistical analysis of the western blot data (Fig. 4D) showed that total protein levels of FAK, ERK1/2, p38MAPK, and NF-κB p65 did not significantly change during Aβ-induced apoptosis. The results of Fig 4C, D and A and B in this figure suggest that increased NF-κB activity is caused by activation of ERK1/2 and p38MAPK, but not by increases in their expression levels. Control is specimen without treatment with $A\beta_{25\text{-}35}$.