SUPPLEMENTARY DATA

TAK1 Improves Cognitive Function via Suppressing RIPK1-Driven Neuronal Apoptosis and Necroptosis in Rats with Chronic Hypertension

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Supplementary Figure 1. The expression of p-p65, p-JNK and p-p38 in different groups. (A) Co-immunostaining of mCherry-fluorescence (red) and GFAP (green) in the cerebral cortex. Scale bar: 50 μm. (B, C) Western blot shows the expression of p-p65, p-JNK, and p-p38 in the cerebral cortex and hippocampus of AAV-siScramble (n=4), AAV-siTAK1 (n=5), AAV-Scramble (n=4) and AAV-TAK1 (n=5) groups. (D-G) Quantitative analysis of p-p65, p-JNK, and p-p38 levels (Protein levels were normalized to its corresponding total protein). Data are expressed as mean ± SEM.
Non-parametric Mann-Whitney U test was used (*p < 0.05, **p < 0.01). (H, I) Western blot shows the expressions of p-p65, p-JNK, and p-p38 in the cerebral cortex and hippocampus of sham+AAVsiScramble and sham+AAV-siTAK1 groups (n=4 per group). (J, K) Quantitative analysis of p-p65, p-JNK, and p-p38 levels (Protein levels were normalized to its corresponding total protein). Data are expressed as mean ± SEM. Non-parametric Mann-Whitney U test was used (*p < 0.05, **p < 0.01). (L) Western blot shows the expression of p-p65 in HT-22 cells with or without indicated TNFα, 5Z-7-Oxozaenol, Nec-1s and JSH-23 treatment. (M) Quantitative analysis of p-p65 levels (Protein levels were normalized to p65). n=3 independent cell culture experiments. Data are expressed as mean ± SEM. Non-parametric Kruskal-Wallis test was used (*p < 0.05).