Ginsenoside Rd attenuates tau protein phosphorylation via the PI3K/AKT/GSK-3β pathway after transient forebrain ischemia

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Fig. S1 Effects of Rd on p-tau expression in ischemic rat brain 7 d and 14 d after MCAO. Western blotting analysis was performed using antibodies against S199/202 (a: 7 d and b: 14 d) or PHF-1 (c: 7 d and d: 14 d) or tau-5 (e: 7 d and f: 14 d) as described in Methods. Data are expressed as mean ± SEM (n = 6).
GAPDH was used as internal control. Mean values in vehicle-treated (Sham) groups were scaled to 100%. * p <0.05 vs. MCAO group, SA: saline

Fig. S2 Effects of Rd on AKT and GSK-3β phosphorylation in ischemic rat brain after MCAO. Western blotting analysis was performed using antibodies against p-GSK-3β/GSK-3β (a: 7 d and b: 14 d) or p-AKT/AKT (c: 7 d and d: 14 d) as described in Methods. Data are expressed as mean ± SEM (n = 6). Mean values in vehicle-treated (Sham) groups were scaled to 100%. * p < 0.05 vs. MCAO+Rd group, SA: saline
Fig. S3 PI3K/AKT/GSK-3β pathway is involved in the effect of Rd on tau phosphorylation in vitro. Western blotting analysis of proteins extracted from cultured neurons 24 h after OGD was performed using antibodies against S199/202 (a) or PHF-1 (b) or tau-5 (c) or p-GSK-3β (d) as described in Methods. LY294002 (LY, 5 μM) was pretreated 12 h before OGD. Data are expressed as mean ± SEM (n = 6). GAPDH was used as internal control. Mean values in sham-treated groups were scaled to 100%. * p < 0.05 vs. OGD+Rd group, SA:saline