BCytokine2

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positive (TKO) cells were plated in 1B. Cytokine-2 was detected by Western blot analysis and Western blotting with an anti-mouse TKO cell (H4D) and anti-mouse TKO-LnF cells (H4D) (Fig. S1 C). The Ki-37 content of the KiB-tagged cytokine-2 was detected by western blot analysis. The KiB-tagged cytokine-2 was detected by Western blot analysis by Western blotting (Fig. S1 D). The KiB-tagged cytokine-2 was detected by Western blot analysis by western blotting (Fig. S1 E). Discussion Cytokine-2 and Cytokine-3 are important targets of TPA, with the potential to target specific TPA receptors (1). TPA is a reactive, pro-inflammatory, pro-inflammatory cytokine, which is associated with a progressive increase in the number of reactive molecules in the brain, which is associated with increased neurotoxicity and neuronal injury (2, 3). Next, we investigated the effect of TPA on TPA-induced neurotoxicity by rat slices. As previously reported (4), rat slices were used as a control and TPA-induced neurotoxicity was observed (5). The experiments showed that TPA treatment resulted in much greater neurotoxicity than that of TPA alone (Fig. S1C). The concentration of TPA in rat slices significantly decreased (P ¡ 0.01) TPA-induced neurotoxicity (P ¡ 0.005) and (P ¡ 0.01) compared to TPA alone (Fig. S1D). TPA-induced neurotoxicity was observed in rat slices from 6 months to 3 years of age (Fig. S1E). As by-products of TPA were also observed in rat slices from 3 to 7 years of age (Fig. S1F). The induction of neurotoxicity in TPA-treated rats was accompanied by the induction of TPA-induced neurotoxicity in TPA-induced rats (Fig. S1G). TPA-induced neurotoxicity was accompanied by TPA-induced neurotoxicity in TPA-induced rats (Fig. S1H). TPA-induced neurotoxicity was accompanied by the induction of TPA-induced neurotoxicity in TPA-induced rats (Fig. S1I). TPA-induced neurotoxicity was accompanied by the induction of TPA-induced neurotoxicity in TPA-induced rats (Fig. S1J). TPA-induced neurotoxicity was accompanied by the induction of TPA-induced neurotoxicity in TPA-induced rats (Fig. S1K). The induction of TPA-induced neurotoxicity in TPA-treated rats was associated with induction of TPA-induced neurotoxicity in TPA-induced rats (Fig. S1L). TPA-induced neurotoxicity was accompanied by TPA-induced neurotoxicity in TPA-induced rats (Fig. S1M). TPA-induced neurotoxicity was accompanied by TPA-induced neurotoxicity in TPA-induced rats (Fig. S1N). TPA-induced neurotoxicity was accompanied by the induction of TPA-induced neurotoxicity in TPA-induced rats (Fig. S1O). TPA-induced neurotoxicity was accompanied by TPA-induced neurotoxicity in TPA-induced rats (Fig. S1P). TPA-induced neurotoxicity was accompanied by the induction of TPA-induced neurotoxicity in TPA-induced rats (Fig. S1R). The induction of TPA-induced neurotoxicity in TPA-induced rats was associated with induction of TPA-induced neurotoxicity in TPA-induced rats (Fig. S1S). TPA-induced neurotoxicity was accompanied by the induction of TPA-induced neurotoxicity in TPA-induced rats (Fig. S1T). TPA-induced neurotoxicity was accompanied by the induction of TPA-induced neurotoxicity in TPA-induced rats (Fig. S1U). TPA-induced neurotoxicity was accompanied by TPA-induced neurotoxicity in TPA-induced rats (Fig. S1V). TPA-induced neurotoxicity was accompanied by the induction of TPA-induced neurotoxicity in TPA-induced rats (Fig. S1