

Supporting Materials

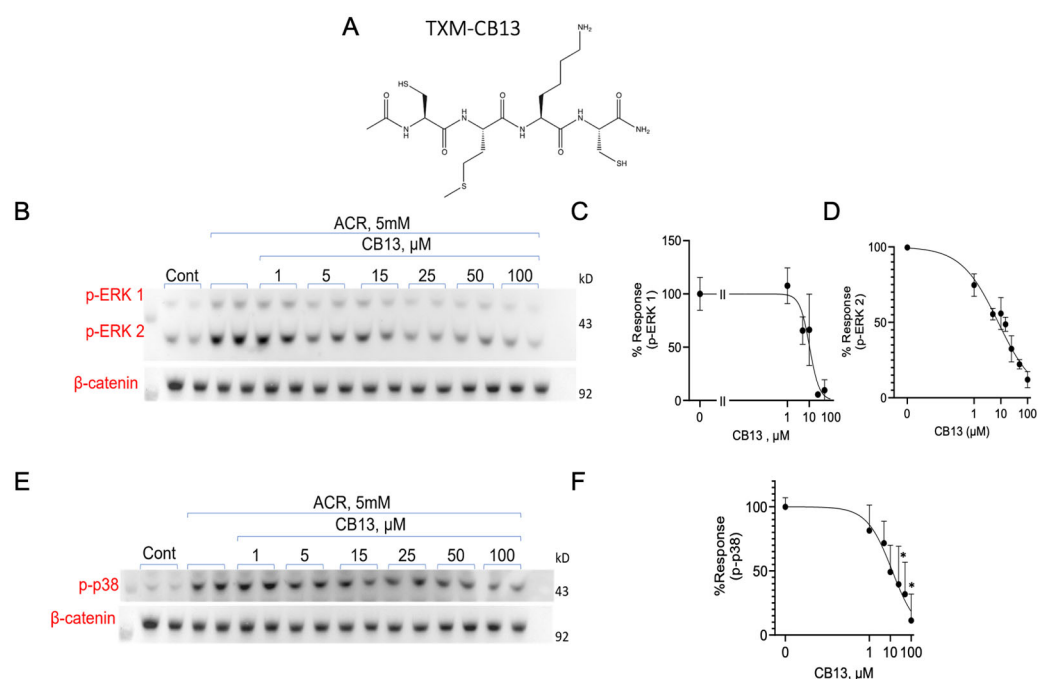


Figure S1. TXM-CB13 reverses ACR-induced activation of MAPKs. (A) The structure of TXM-CB13 (B) PC12 cells were treated with 5mM ACR for 2h, washed, and incubated with increasing concentrations of TXM-CB13. Cell lysate proteins were separated on 10% SDS-PAGE and inhibition of ACR-induced phosphorylation of (C) ERK1 and (D) ERK2 was calculated and quantified by normalization to β -catenin (E,F) p38^{MAPK} phosphorylation was visualized using the corresponding anti-phospho p38^{MAPK} antibodies and quantified by normalization to β -catenin. The values shown are averages ($\pm$ SEM) of three independent experiments normalized to β -catenin the phosphorylation state of cells treated with ACR after 3 h. Student's *t*-test (two populations) was performed for ACR treated cells * $p < 0.05$.

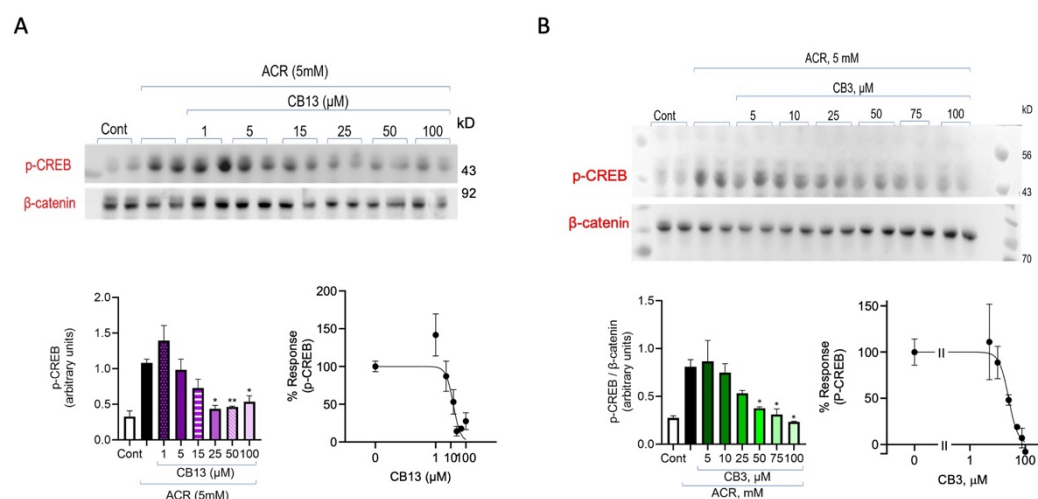


Figure S2. Inhibition of ACR-induced phosphorylation of CREB. PC12 cells were treated with 5mM ACR for 2h, washed, and with increasing concentrations of (A) TXM-CB13 and (B) TXM-CB3 for an additional 1 h. Cell lysate proteins were loaded in equal amounts and separated on 10% SDS-PAGE. Inhibition of ACR-induced phosphorylation of CREB was quantified and the values shown are averages ($\pm$ SEM) of two independent experiments normalized to β -catenin. Student's *t*-test (two populations) was performed for ACR treated cells * $p < 0.05$; ** $p < 0.01$.