Figure 5A-figure supplement 5: ERK phosphorylation is dose- and time-dependent and can be inhibited.



**Gαq/PKC and Gαs/PKA pathways involved in TRP/TRPR-induced ERK1/2 phosphorylation.** Dose- and time-response analyses of TRP/TRPR-induced ERK1/2 phosphorylation in HEK293 cells (**A**) and Sf21 cells (**B**). Cells expressing TRPR were serum-starved then incubated either with an increasing dose of TRP2, (from 0.1 pM to 1 μM) for 10 min or with 100 nM TRP2 for different times (from 0 to 90 min), then harvested to quantify ERK1/2 phosphorylation. Effects of Gαi inhibitor pertussis toxin (PTX), MEK inhibitor U0126, PKA inhibitor H89, and PKC inhibitor Go6983 on TRP2-induced ERK1/2 phosphorylation in HEK293 cells (**C**) and Sf21 cells (**D**). The cells were pretreated with or without inhibitors for 2 hours then stimulated with ddH2O (control) or TRP2 (10 nM or 1 μM) for 10 min. The phosphorylated ERK was normalized to a loading control (total ERK). All data are presented as mean ± s.e.m. from three independent replicates, and blots shown are representative of these experiments. One-way ANOVAs followed by Tukey’s post-hoc tests were used for multi-group comparisons, and significant differences (*p* < 0.05) are denoted by letters.