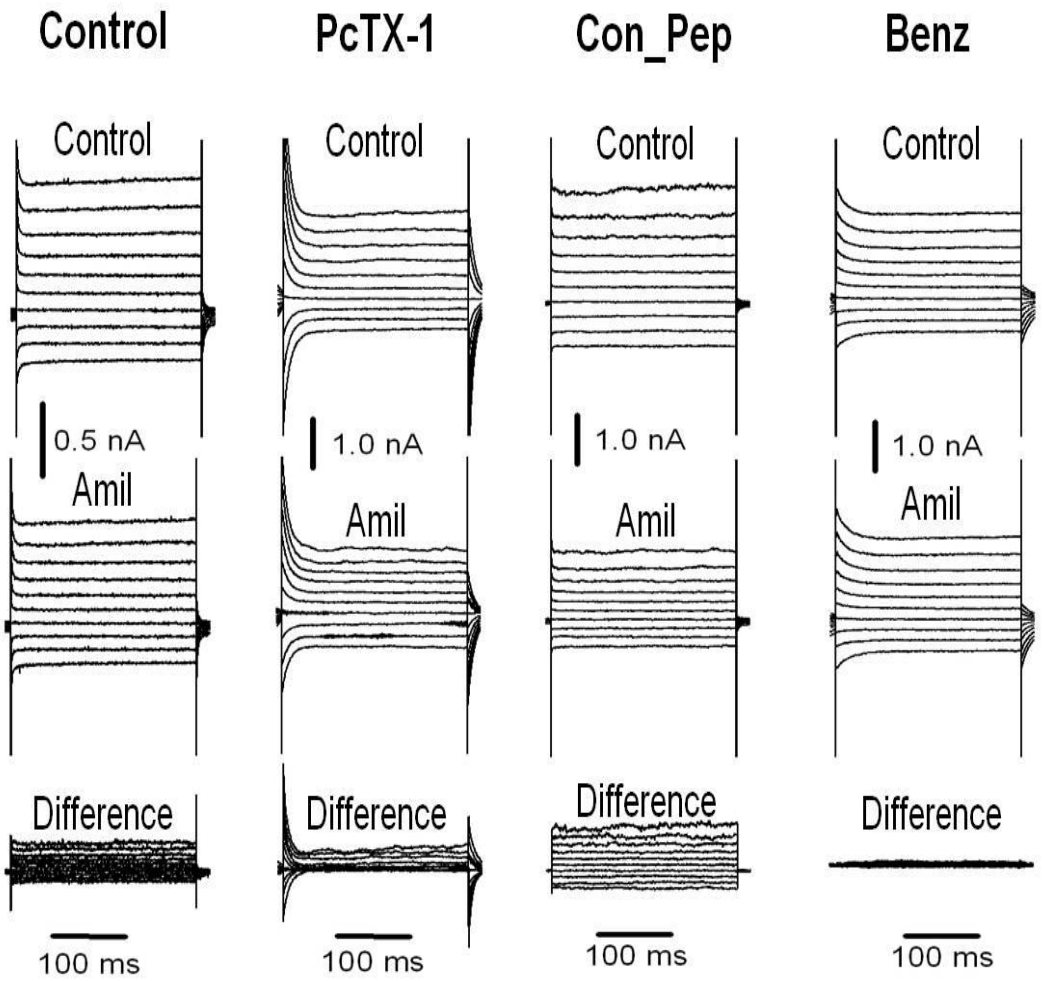
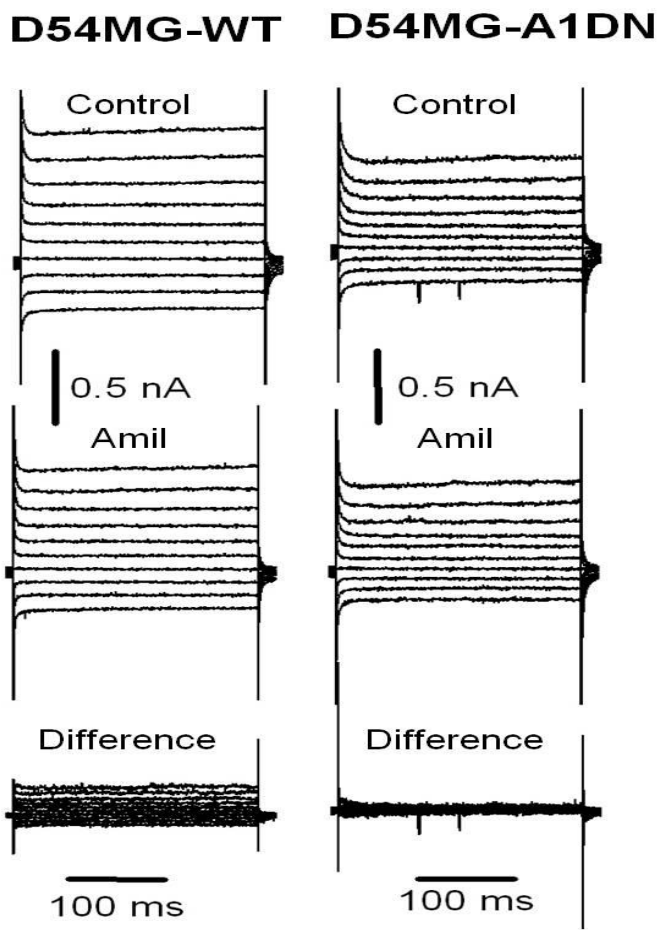
SUPPLEMENTARY DATA

Figure S1



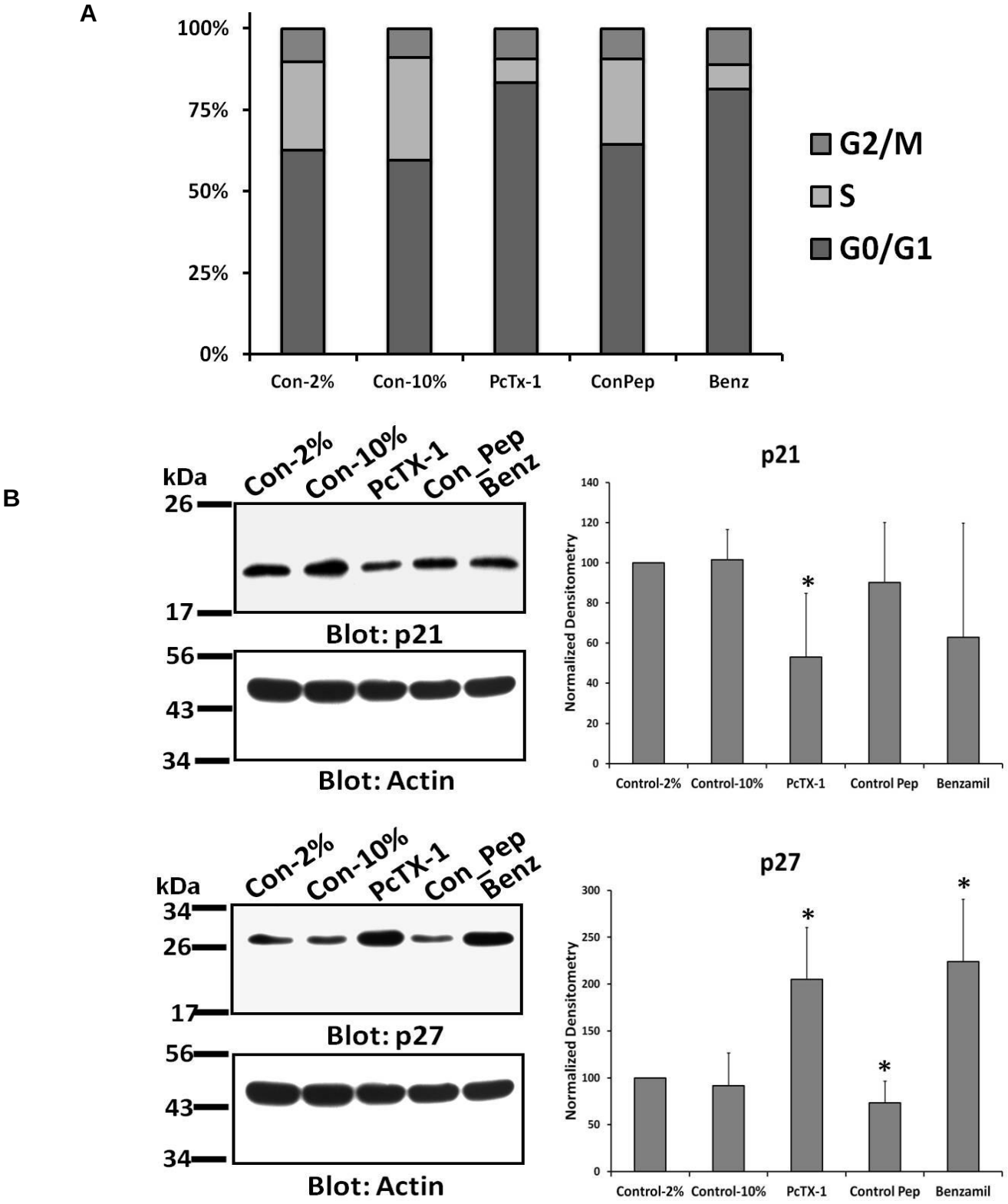
Whole cell patch clamp recording of D54-MG cells treated with PcTX-1, control peptide, and benzamil for 24h. Representative traces of whole cell patch clamp recording of D54-MG cells showing the basally active whole cell current (top traces), current after perfusion of 100µM amiloride to the bath solution (middle traces), amiloride sensitive difference current (bottom traces). Current-voltage relationships were obtained using a pulse protocol in which cells were stepped from a -40 mV holding potential from -100 to +80 mV in 20 mV increments for 250 ms. There was a significant difference in amiloride sensitive current in control cells or control peptide pretreated cells as compared to the currents recorded from cells pretreated with PcTx-1 and benzamil.

Figure S2



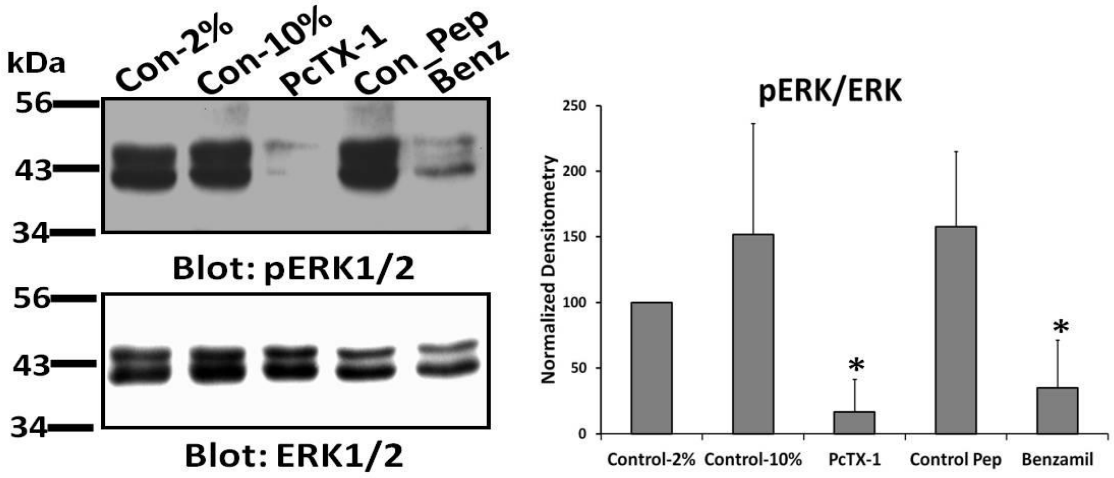
Representative whole cell patch clamp recordings of wild type D54MG (D54MG-WT) and D54-MG cells stably transfected with DN eGFP-ASIC1 cDNA (D54MG-A1DN) showing the basally active whole cell current (top traces), current after perfusion of 100μM amiloride to the bath solution (middle traces), amiloride sensitive difference current (bottom traces). Current-voltage relationships were obtained using a pulse protocol in which cells were stepped from a -40 mV holding potential from -100 to +80 mV in 20 mV increments for 250 ms.

Figure S3



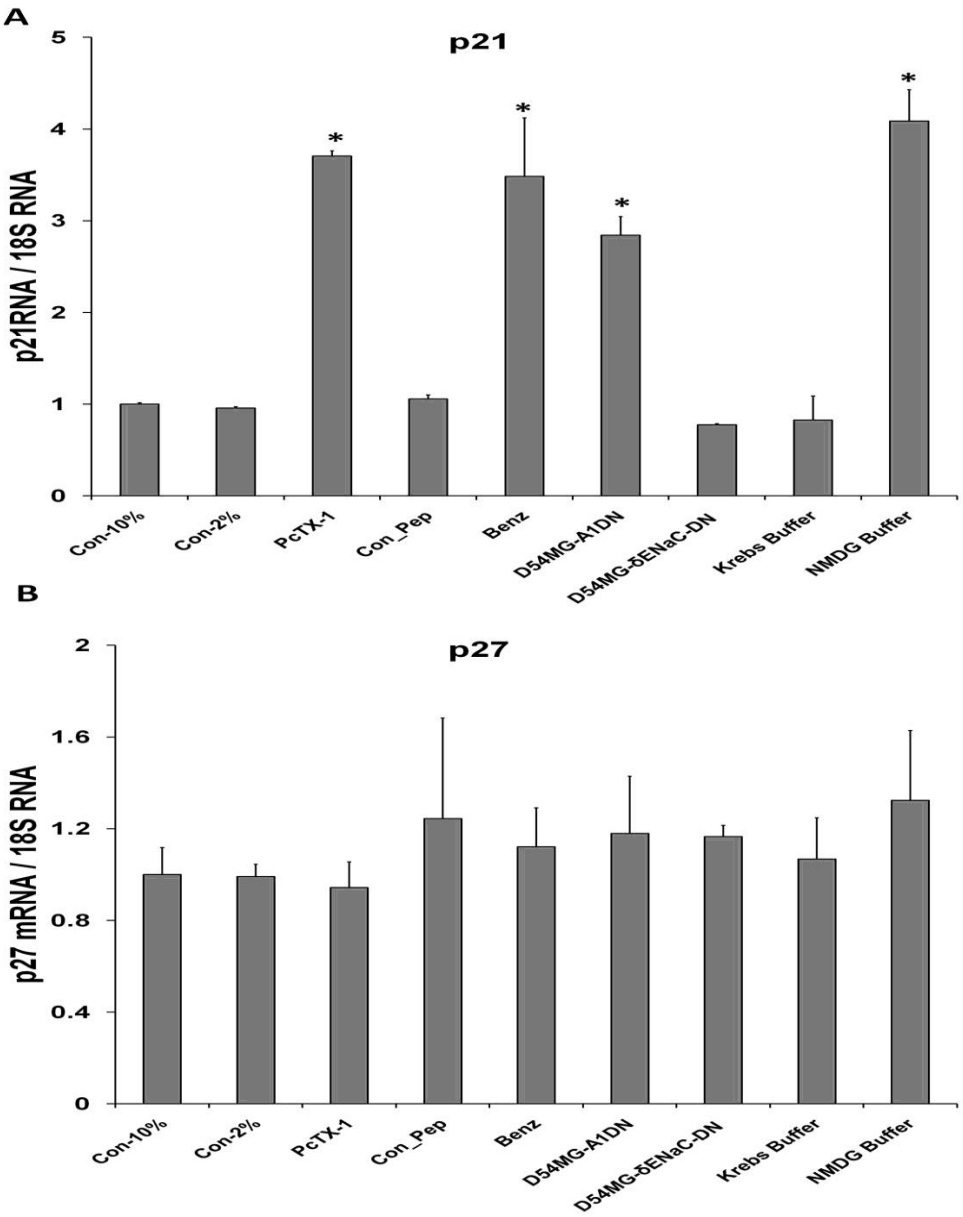
Inhibition of glioma conductance caused cell cycle arrest in U87MG cells A. The stacked bar graph shows the percentage of U87MG cells in each cell cycle phase for different experimental conditions. Values are mean ± S. D. (n=5). **B.** PcTx1 and benzamil increased expression of $p27^{kip1}$ following 24h incubation, but had no effect on the expression of $p21^{cip1}$ (n=4). The blots were stripped and reprobed with β-actin for loading control. Each bar represents the normalized density compared to control with 2% FBS.

Figure S4



Inhibition of glioma conductance inhibited ERK1/2 phosphorylation in U87MG cells. The phosphorylation of ERK1/2 was significantly reduced in PcTX-1 (by 83 ± 25%, n=4) or benzamil (by 65 ± 36%, n=4) treated U87MG cells. Each bar represents the normalized density compared to control with 2% FBS.

Figure. S5



Real-time PCR of $p21^{Cip1}$ and $p27^{Kip1}$ from D54-MG cells. This figure shows average relative mRNA expression for different conditions proportional to 18S rRNA in D54-MG cells. Expression for $p21^{Cip1}$ (***A***) is significantly higher (n=4) in cells treated with either PcTX-1 (100nM), or benzamil (100μM) or incubated in low [Na^+] buffer for 24h as compared to D54-MG cells. The expression of $p21^{Cip1}$ was also increased by knock-down of ASIC-1, but not the δENaC subunit. The results are the averages of four different samples run in duplicate. The mRNA level for $p27^{Kip1}$ (***B***) were unchanged by the same experimental maneuvers (n=4).