PKC epsilon phosphorylation of NaV1.8 increases sodium channel function and produces mechanical hyperalgesia in mice

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Supplemental Table 1. Voltage dependence of $Na_V1.8$ activation and inactivation.

	Activation (V_h = -70 mV)			Steady-state inactivation		
	$V_{1/2}$	*k*	n	$V_{1/2}$	*K*	n
Prkce$^{+/+}$						
Control	-21.14±1.78	6.33±1.17	7	No test		
ψεRACK	-32.42±1.07*	4.42±0.78	5	-35.40±0.51†	6.14±0.46	12
Scr-ψεRACK	-21.14±1.80	5.96±1.21	5	-38.12±0.36$^{\#}$	6.24±0.33	13
Prkce$^{-/-}$						
Control	-24.37±0.82	5.05±0.62	5	No test		
ψεRACK	-22.76±0.89	5.42±0.62	7	-41.19±0.54	6.61±0.49	10
Scr-ψεRACK	-21.06±1.22	5.12±0.85	7	-40.56±0.30	5.81±0.27	13

*$p < 0.001$ compared with control and scrambled ψεRACK-treated *Prkce*$^{+/+}$ cells and with ψεRACK-treated *Prkce*$^{-/-}$ cells; †p < 0.001 compared with scrambled ψεRACK-treated *Prkce*$^{+/+}$cells and with ψεRACK-treated *Prkce*$^{-/-}$ cells; #p<0.001 compared with scrambled ψεRACK-treated *Prkce*$^{-/-}$ neurons.

Supplemental Figure 1. $Na_v1.8$ activation or inactivation kinetics.

Administration of ψεRACK or a scrambled ψεRACK peptide (Scrambled) did not alter the kinetics of activation (A) or inactivation (B) in *Prkce* $^{+/+}$ (WT) or *Prkce* $^{-/-}$ (KO) DRG neurons. CTRL: untreated control neurons.