

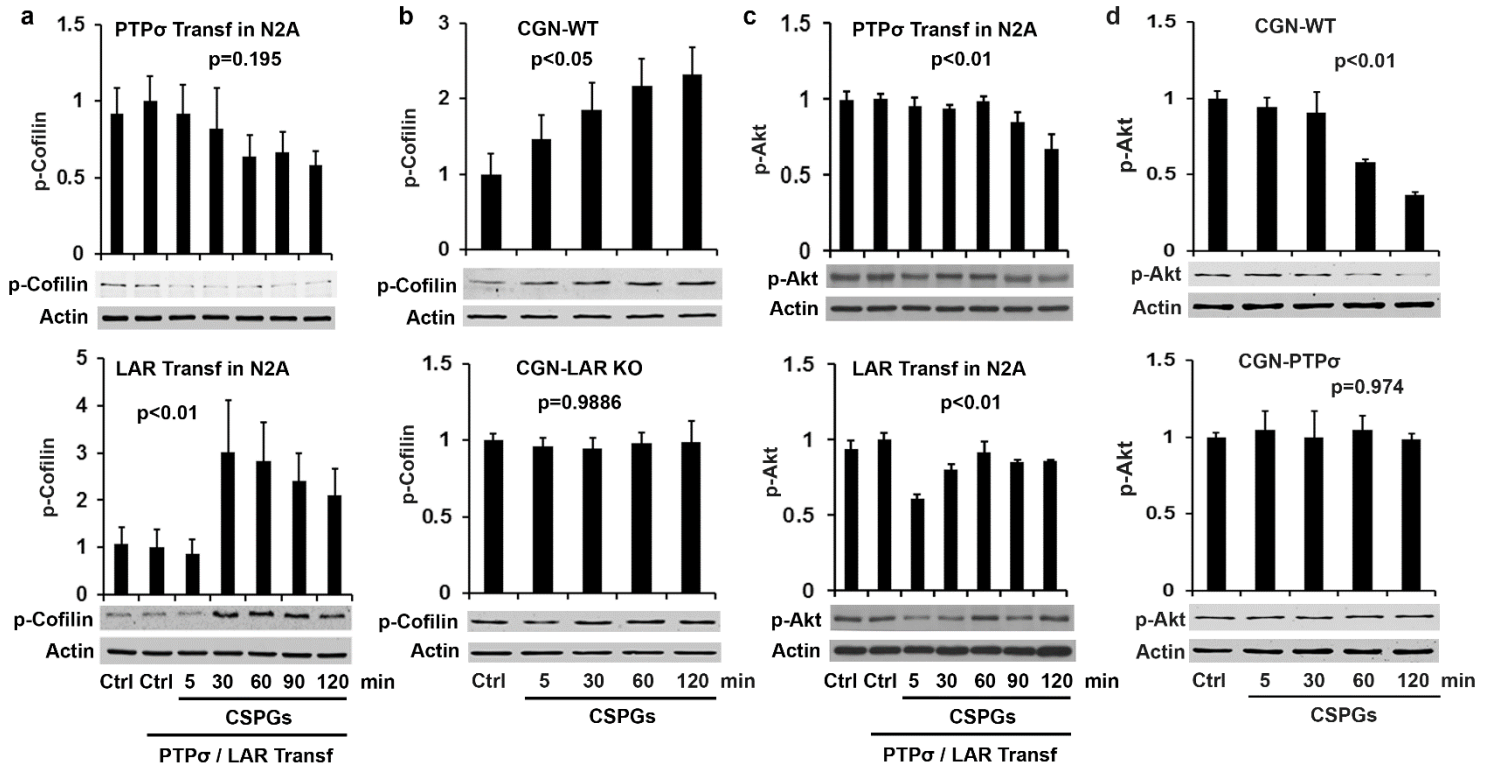
Two PTP receptors mediate CSPG inhibition by convergent and divergent signaling pathways in neurons

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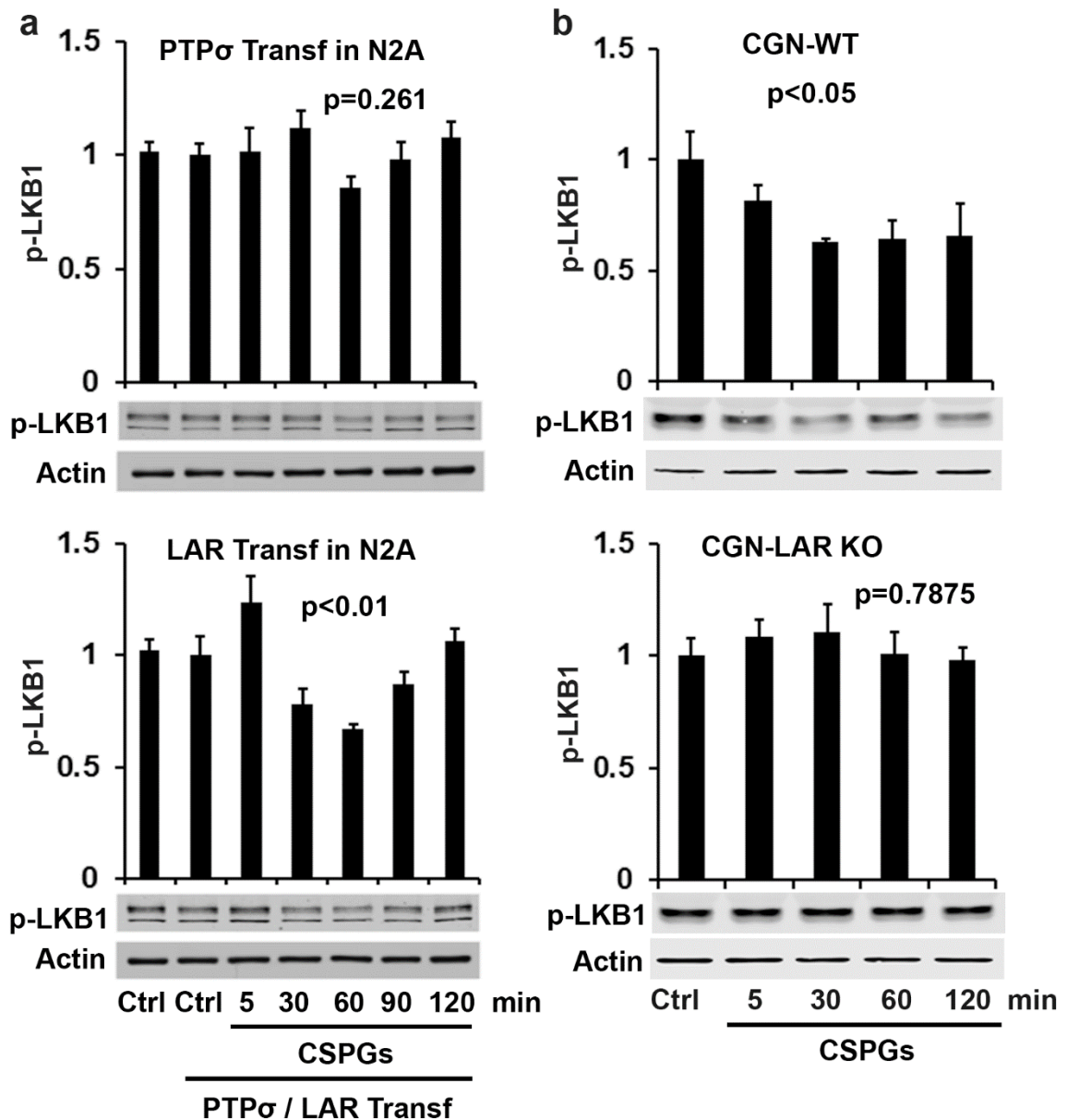
Supplemental Table 1. Comparison of the results with two loading proteins: actin and total signaling protein.

Proteins	Fig.	N2A cells		CGNs		N2A cells		CGNs		Activity
		PTP σ -Transf	PTP σ +/+	PTP σ -/-	LAR-Transf	LAR +/+	LAR -/-			
p-Cofilin/actin	S1	↔ p = 0.195	ND	ND	↑ p < 0.01	↑ p < 0.05	↔ p = 0.9886	↓ by LAR		
p-Cofilin/T-Cofilin	2	↔ p = 0.234	ND	ND	↑ p < 0.01	↑ p < 0.05	↔ p = 0.4838	↓ by LAR		
p-Akt/actin	S1	↓ p < 0.01	↓ p < 0.01	↔ p = 0.974	↓ p < 0.01	ND	ND	↓ by both		
p-Akt/T-Akt	2	↓ p < 0.01	↓ p < 0.01	↔ p = 0.879	↓ p < 0.01	↓ (Fisher et al)	↔ (Fisher et al)	↓ by both		
p-LKB1/actin	S2	↔ p = 0.261	ND	ND	↓ p < 0.01	↓ p < 0.05	↔ p = 0.7875	↓ by LAR		
p-LKB1/ T-LKB1	5	↔ p = 0.302	ND	ND	↓ p < 0.01	↓ p < 0.05	↔ p = 0.9189	↓ by LAR		

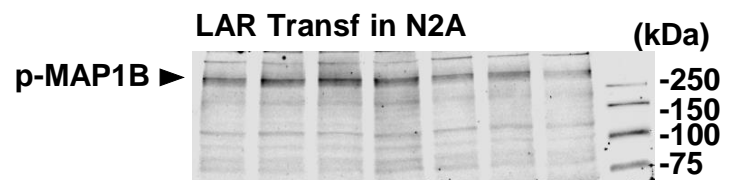
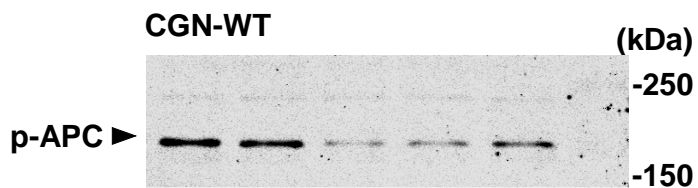
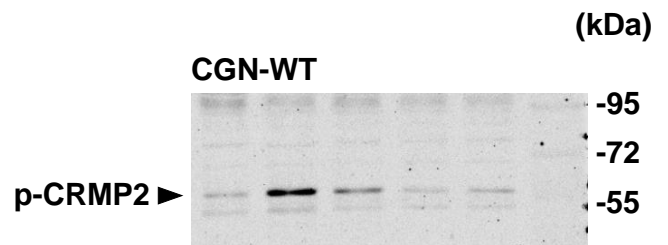
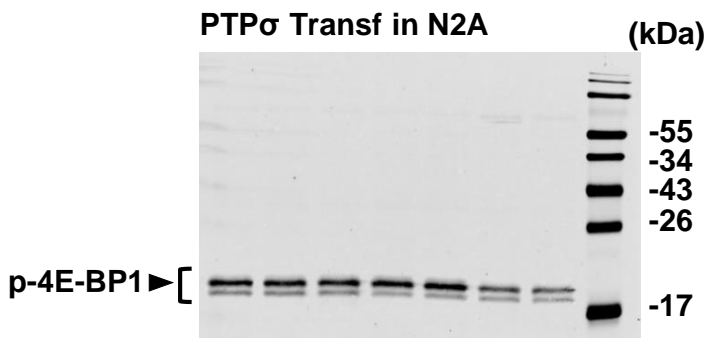
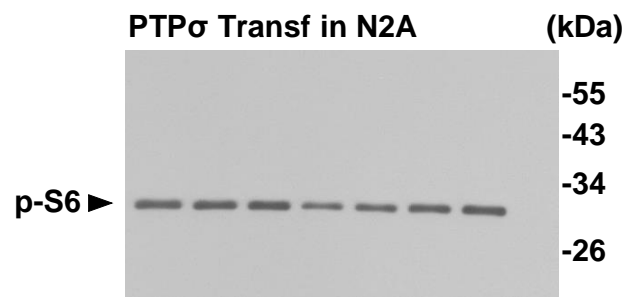
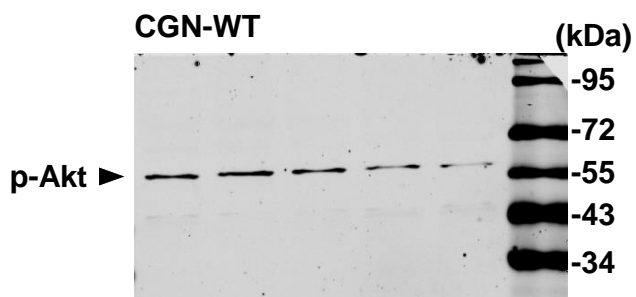
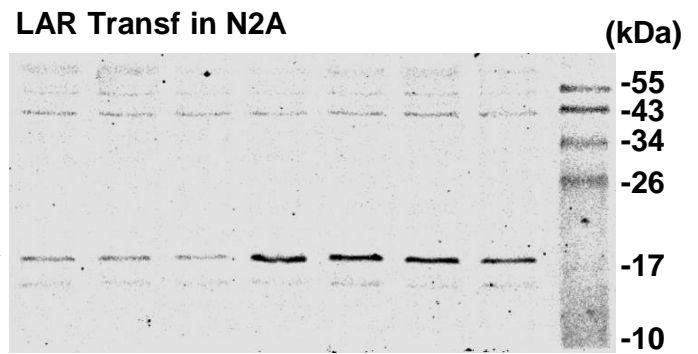
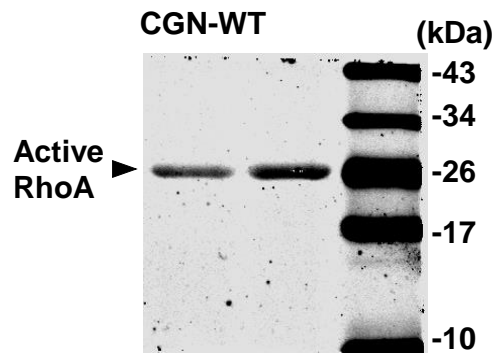
Note: ND: not determined.



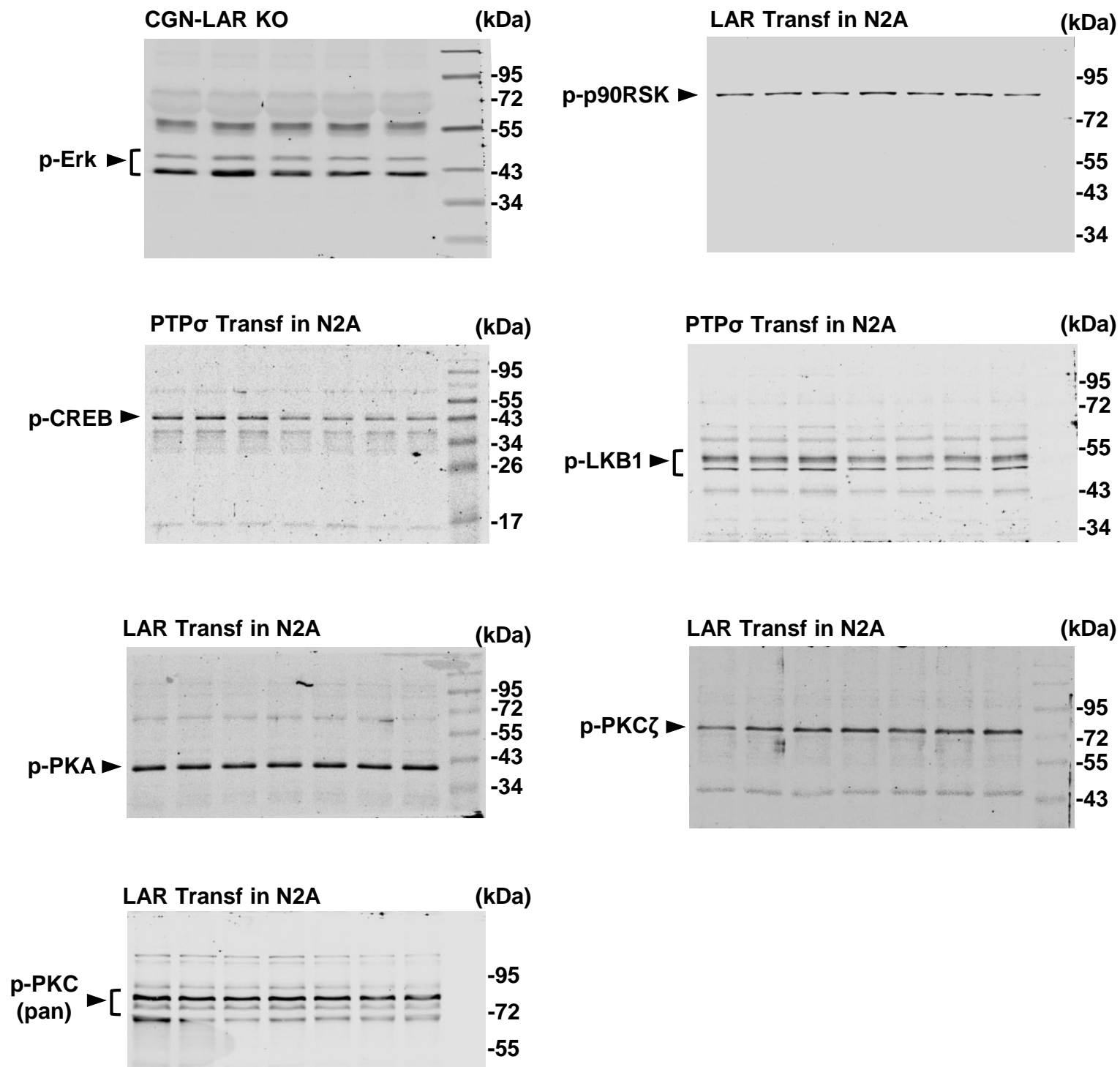
Supplemental figure 1 Effect of CSPG on activities of cofilin and Akt in N2A cells or CGNs measured with actin as a loading protein. PTP σ or LAR transfected N2A cells or CGNs derived from PTP σ or LAR KO mice were treated with purified CSPGs (1.5 μ g/ml) for different lengths of time, and the levels of phosphorylated Cofilin (p-Cofilin Ser3, inactive form, **a** and **b**) and Akt (p-Akt s473, active form, **c** and **d**) in the cell lysates were measured by Western blots. The levels of p-Cofilin and p-Akt were calibrated with levels of actin in the same samples, instead of with total Cofilin and Akt as in Fig. 2. Use of actin as a loading control indicated the same alterations of these signaling proteins as in Fig. 2.



Supplemental figure 2 Effect of CSPG application on activities of LKB1 in N2A cells or CGNs, measured with actin as a loading protein. PTP σ /LAR transfected N2A cells or CGNs derived from LAR KO mice were treated with purified CSPGs (1.5 μ g/ml) for several lengths of time and the levels of phosphorylated LKB1 (p-LKB1 Ser431, active form) in the cell lysates were measured by Western blots. The levels of p-LKB1 were calibrated with levels of actin in the same samples, instead of with total LKB1 as in Fig. 5. Use of actin as a loading control consistently showed that CSPGs stimulation decreased levels of p-LKB1 in LAR, not PTP σ , transfected N2A cells (a), and in LAR $^{+/+}$, not LAR $^{-/-}$, CGNs (b).



Supplemental figure 3 *The full-length blots are shown for active RhoA, p-Cofilin, p-Akt, p-S6, p-4E-BP, p-CRMP2, p-APC, and p-MAP1B.*



Supplemental figure 4 *The full-length blots are shown for p-Erk, p-p90RSK, p-CREB, p-LKB1, p-PKA, p-PKCζ, and p-PKC(pan).*