Muslimov et al., http://www.jcb.org/cgi/content/full/jcb.201310045/DC1

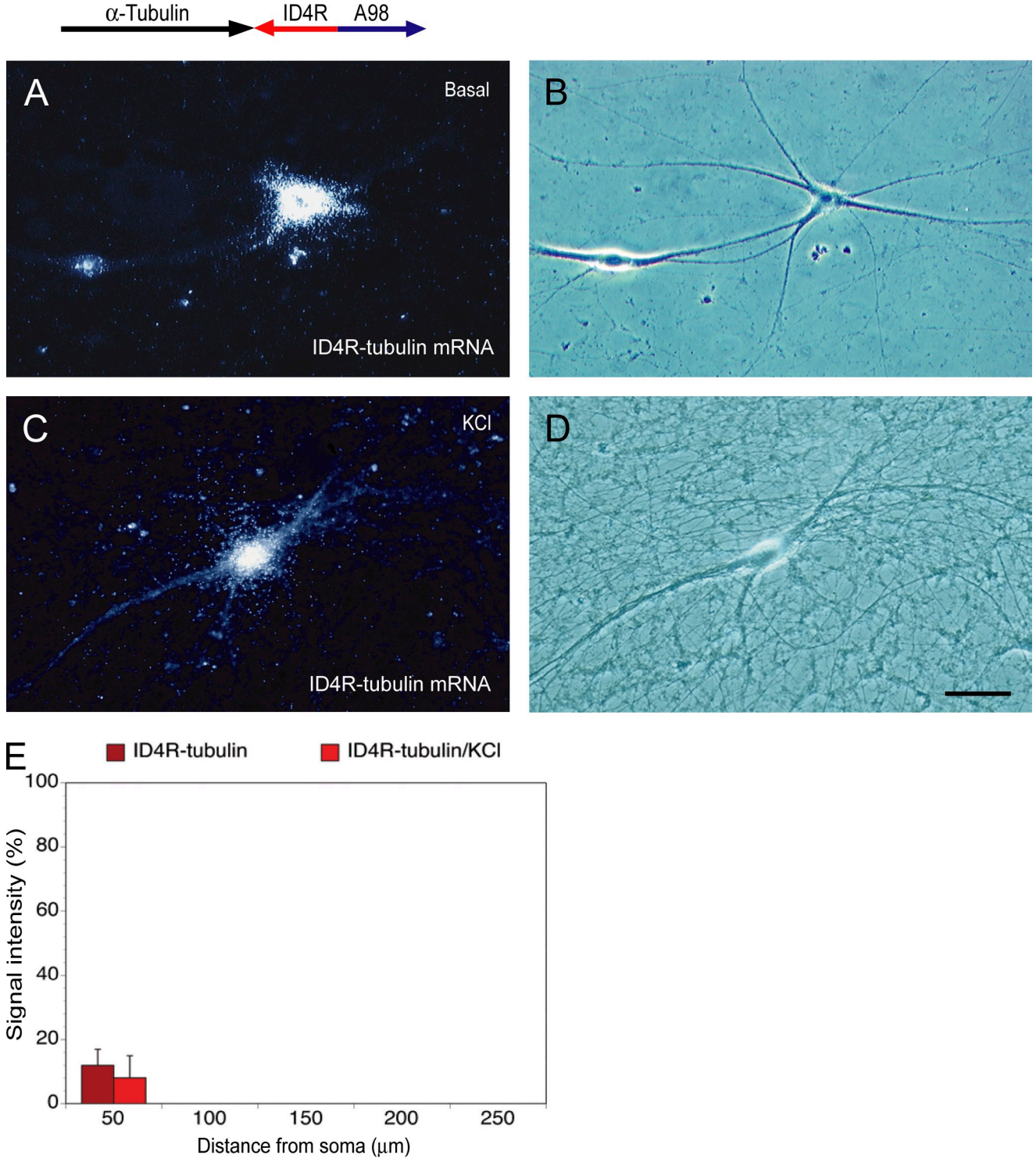

Figure S1. **Somatic retention of reverse complementary ID4-chimeric mRNA.** (A–D) Microinjected ID4R-chimeric α-tubulin mRNA (see sketch) did not substantially exit neuronal somata of sympathetic neurons in culture either under basal conditions (A and B) or under conditions of K^+ depolarization (C and D). Number of cells analyzed: (A and B) 16 neurons, 59 dendrites; (C and D) 13 neurons, 49 dendrites. Bar, 50 µm. (E) Quantitative analysis: one-way ANOVA, Dunnett's post hoc analysis (comparison of RNA levels in the basal state [A and B] with RNA levels after K^+ depolarization [C and D]): $P > 0.9$ for interval points 50 µm. Error bars indicate SEM.

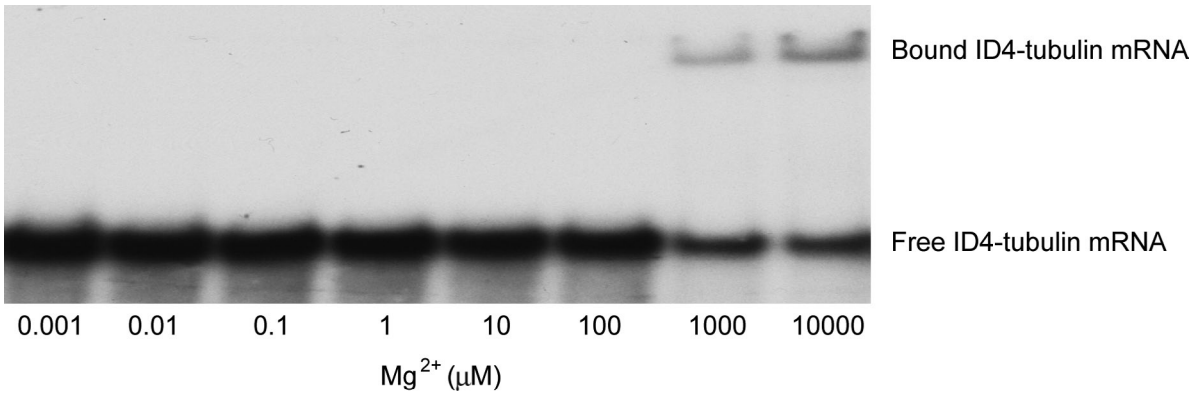

Figure S2. **Mg^{2+} ions promote binding of ID4 mRNA to hnRNP A2 in the millimolar concentration range but not in the micromolar or nanomolar concentration ranges.** In EMSA experiments, mobility shifts indicating binding of ID4-chimeric α-tubulin mRNA to hnRNP A2 were not observed at Mg^{2+} concentrations <1 mM.

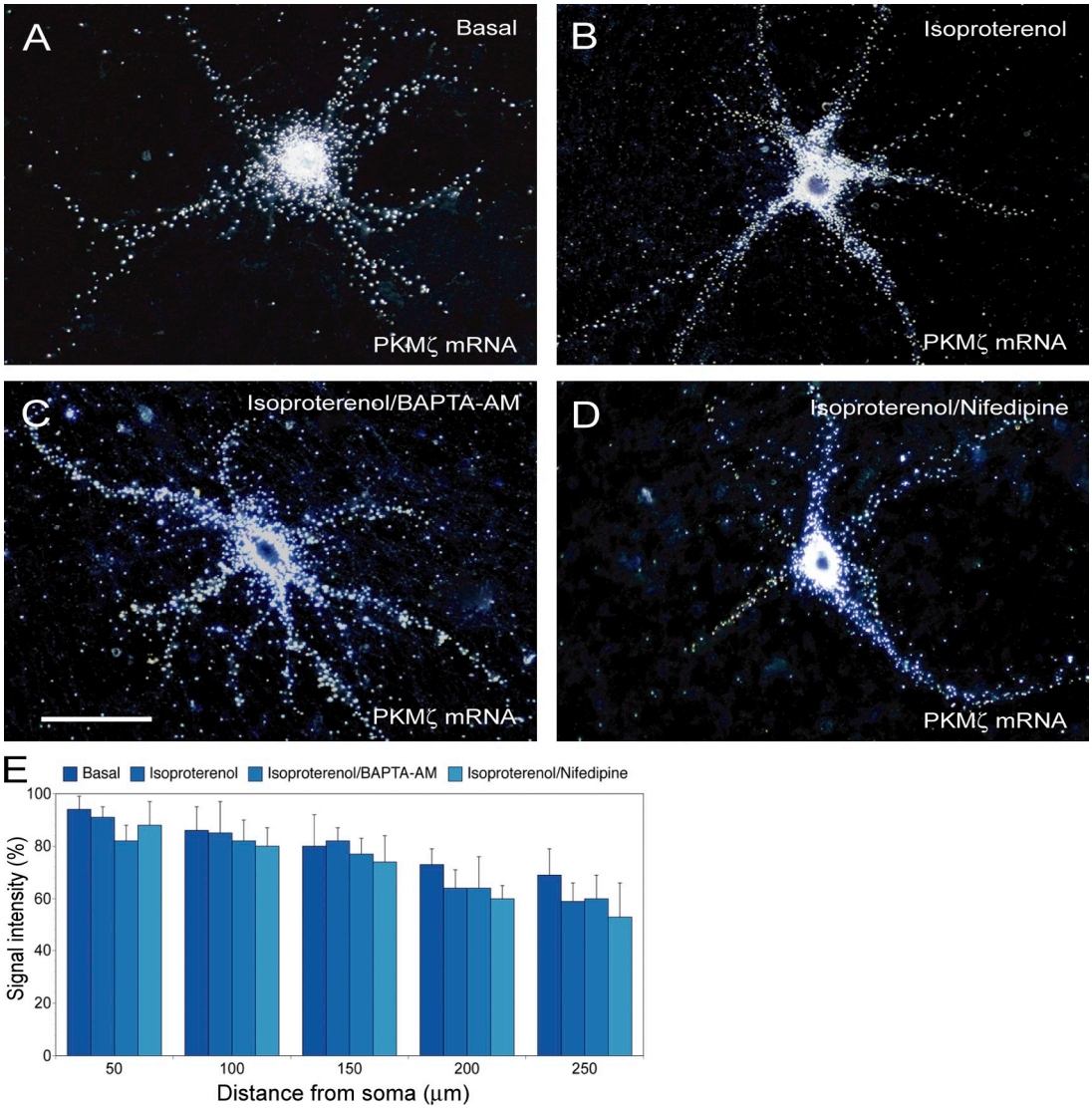

Figure S3. **Dendritic transport of PKMζ mRNA is constitutive and remains unaltered under conditions of Ca^{2+} chelation or L-type VDCC blockade.** (A) PKMζ mRNA was delivered along dendrites after microinjection into sympathetic neurons in culture. (B–D) No changes were observed in extent or pattern of dendritic labeling after application of β-AR agonist isoproterenol (B), application of isoproterenol after preincubation with intracellular Ca^{2+} chelator BAPTA-AM (C), or application of isoproterenol after preincubation with L-type VDCC blocker nifedipine (D) or nimodipine (not depicted). Number of cells analyzed: (A) 9 cells, 31 dendrites; (B) 9 cells, 33 dendrites; (C) 10 cells, 34 dendrites; (D) 11 cells, 37 dendrites. Bar, 50 μm. (E) Quantitative analysis. One-way ANOVA, Dunnett's post hoc analysis (comparison of RNA levels in the basal state with RNA levels after β-adrenergic activation and after β-adrenergic activation in the presence of BAPTA-AM or nifedipine): comparison with isoproterenol (B), $P > 0.6$ for all interval points; comparison with isoproterenol/BAPTA-AM (C), $P > 0.7$ for all interval points; comparison with isoproterenol/nifedipine (D), $P > 0.7$ for all interval points. Error bars indicate SEM.

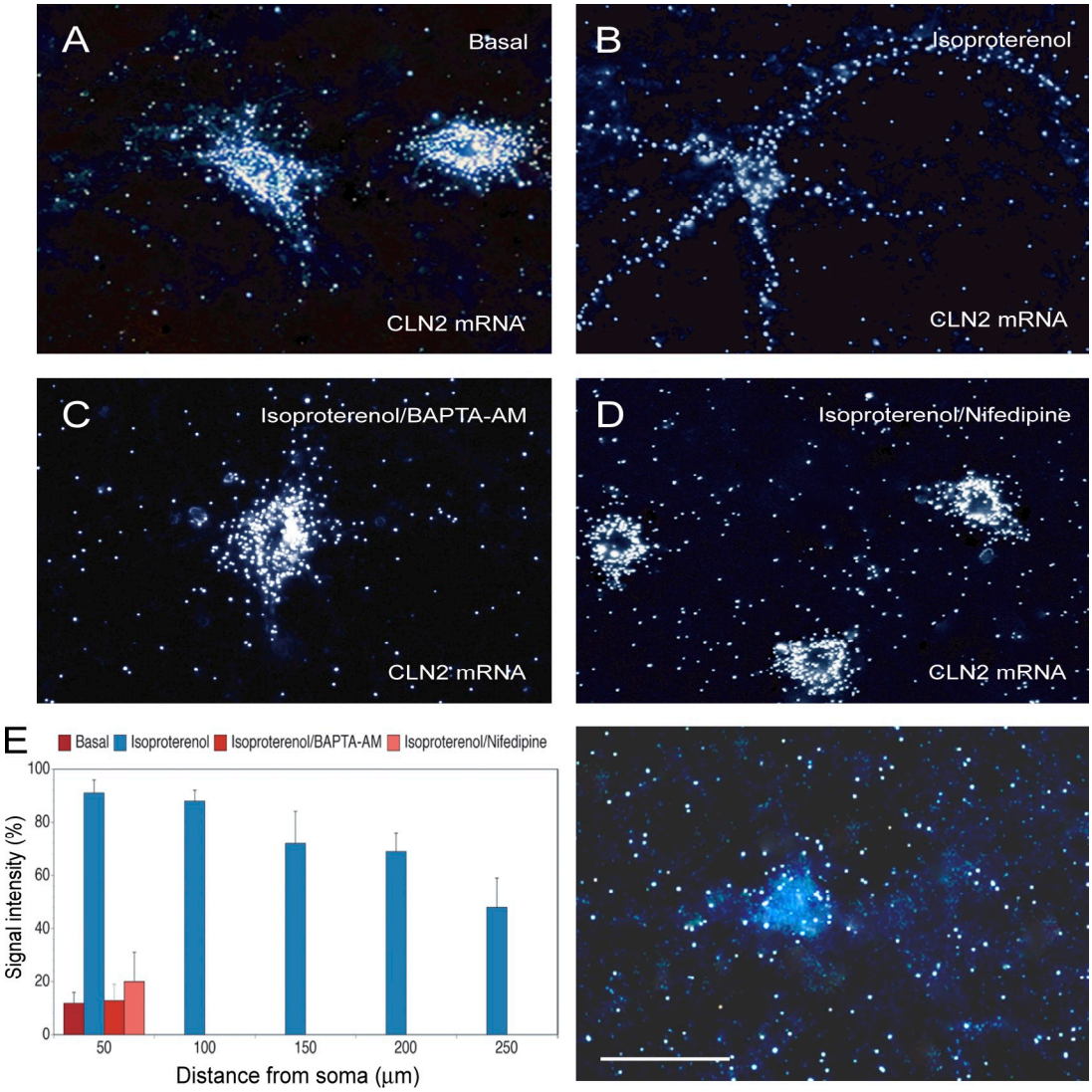

Figure S4. **Endogenous CLN2 mRNA is targeted to dendrites after β-adrenergic activation in sympathetic neurons.** In situ hybridization was performed with a probe specific for rat CLN2 mRNA. (A and B) Localization of endogenous CLN2 mRNA remained somatic under basal conditions (A) but was dendritic after β-adrenergic activation (B). (C and D) Isoproterenol-induced dendritic localization was prevented by preincubation with intracellular Ca^{2+} chelator BAPTA-AM (C) and by preincubation with L-type VDCC blocker nifedipine (D) or nimodipine (not depicted). (F) Only background labeling was apparent when in situ hybridization was performed with a CLN2 mRNA "sense strand" control probe. Number of cells analyzed: (A) 12 neurons, 49 dendrites; (B) 13 neurons, 48 dendrites; (C) 10 neurons, 33 dendrites; (D) 11 neurons, 37 dendrites. Bar, 50 μm. (E) Quantitative analysis. One-way ANOVA, Dunnett's post hoc analysis (comparison of RNA levels in the basal state with RNA levels after β-adrenergic activation and after β-adrenergic activation in the presence of BAPTA-AM or nifedipine): comparison with isoproterenol (B), $P < 0.001$ for all interval points; comparison with isoproterenol/BAPTA-AM (C), $P > 0.7$ for interval points at 50 μm; comparison with isoproterenol/nifedipine (D), $P > 0.8$ for interval points at 50 μm. Error bars indicate SEM.