# Fig Sup1

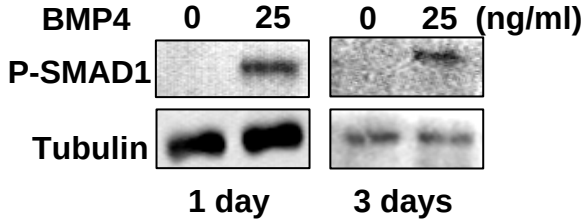



**Fig sup1. BMP4 can regulate the activation of SMAD1 and CRMP2 expression.** A) E16 primary cortical cells were cultured for 1 or 3 days and treated with 25ng/ml BMP4. Western blotting analysis of phosph-SMAD1 levels with GAPDH used as loading control. BMP4 can induce the phosphorylation of SMAD1 in primary cortical cells.

# Fig Sup2

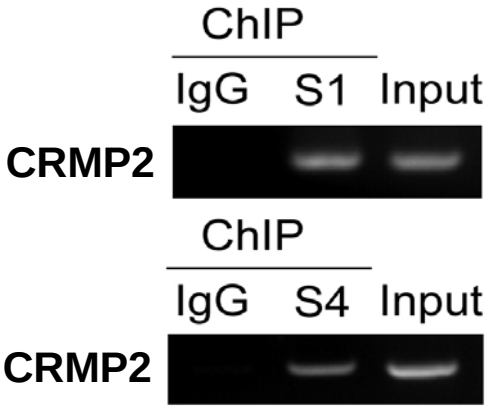



**Fig Sup2** Both SMAD1 and SMAD4 bind to CRMP2 promoter region as evaluated by ChIP analysis as described in material and methods.

# Fig Sup3

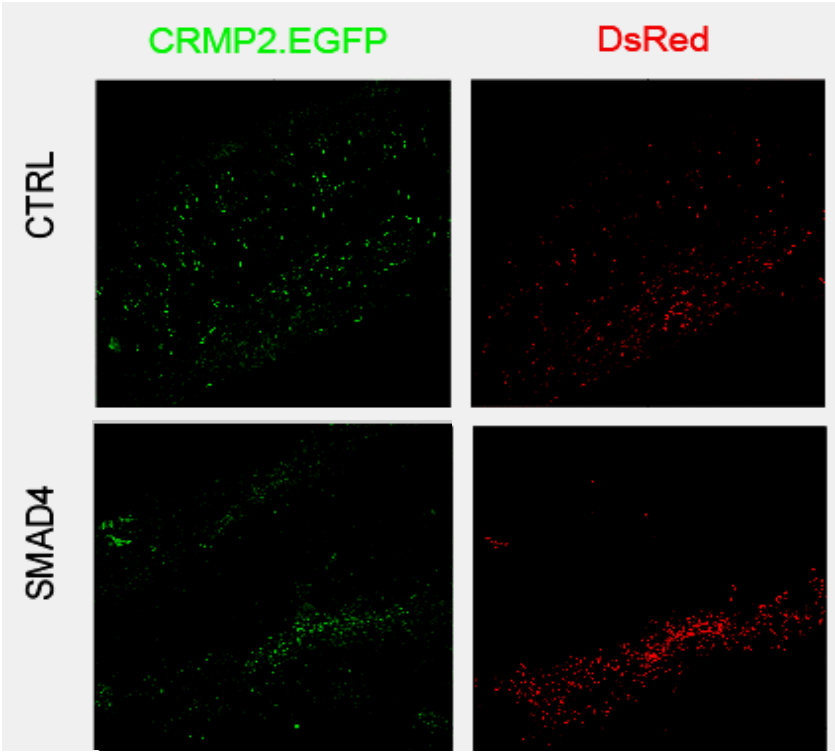



**Fig. Sup3. Ectopic expression of SMAD4 suppresses the expression CRMP2 driven EGFP.** Images of cortices co-transfected with CRMP2.EGFP (green) and DsRed (as a transfection efficiency control, red) together with control vector (upper panel), SMAD4 (lower panel) expression vector 4 days after in utero electroporation.

## Figure Sup4

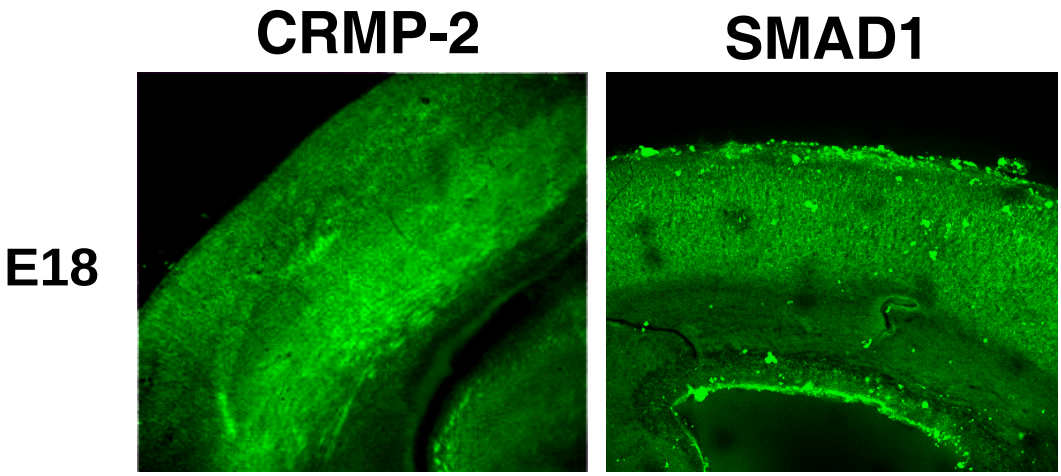



**Fig Sup4. Negative correlation between BMP2/4 and CRMP2 expression during cortex development.** The expression pattern of SMAD1 and CRMP2. Brain slices at E18 were subjected to immunofluorescent staining for SMAD1 and CRMP2.

## Figure Sup5

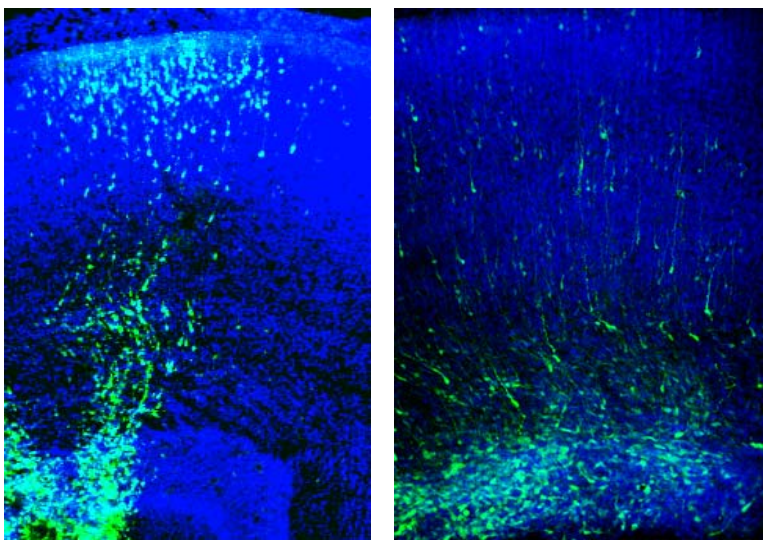



**Fig Sup5. CRMP2 plays a role in neural progenitor cell distribution in the neocortex.** CAGGS-EGFP, CAGGS-CRMP2-EGFP or CAGGS-CRMP2 C381--EGFP were introduced into neuronal progenitor cells at the ventricular zone of the rat brain by *in utero* electroporation at E16. Coronal sections of rat brain 4d after electroporation. Cells expressing CAGGS-CRMP2 C381--EGFP were largely restricted to the VZ/SVZ, although some appeared within the lower IZ at E20.

# Figure Sup6

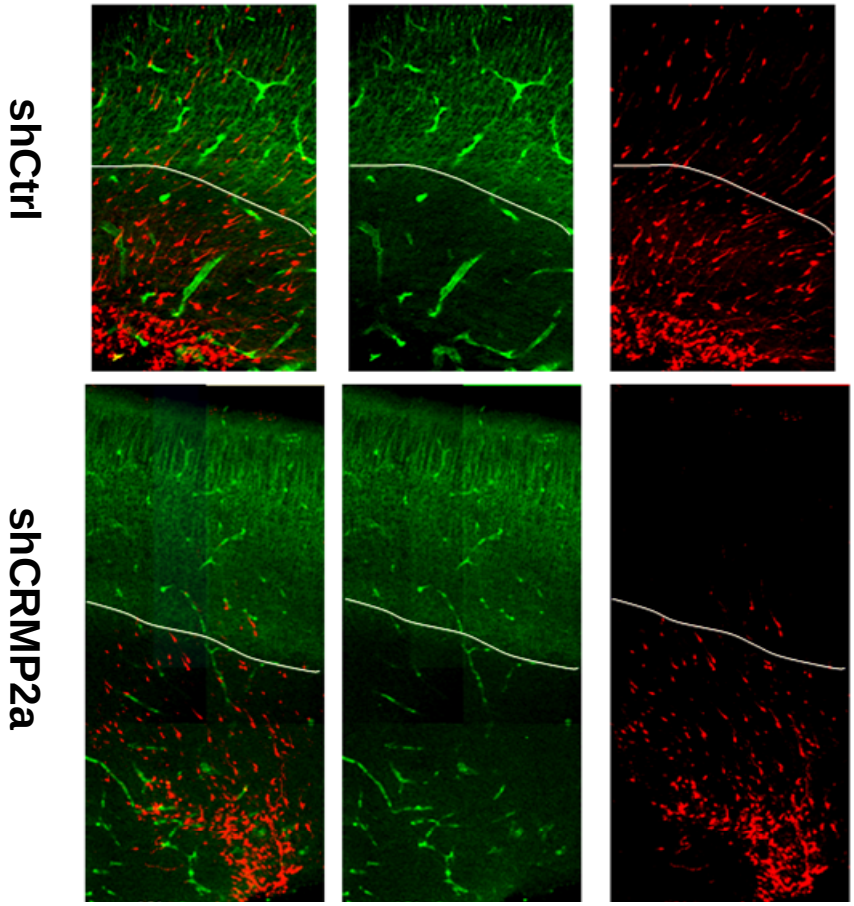



**Fig Sup6.** Disruption of cell redistribution in the neocortex by CRMP2 RNAi. Rat embryos electroporated in utero with pSIREN-RetroQ-DsRed (shCtrl), pSIREN-RetroQ-DsRed-shCRMP2a (shCRMP2a) at E16. Coronal sections of rat brain 4 d after electroporation were subjected to immunofluorescent staining for MAP2, the mature neuron marker. Down-regulation of CRMP2 caused an evident accumulation of cells in the VZ/SVZ and IZ  and only a few cells can reach the CP at E20.

# Fig. Sup7

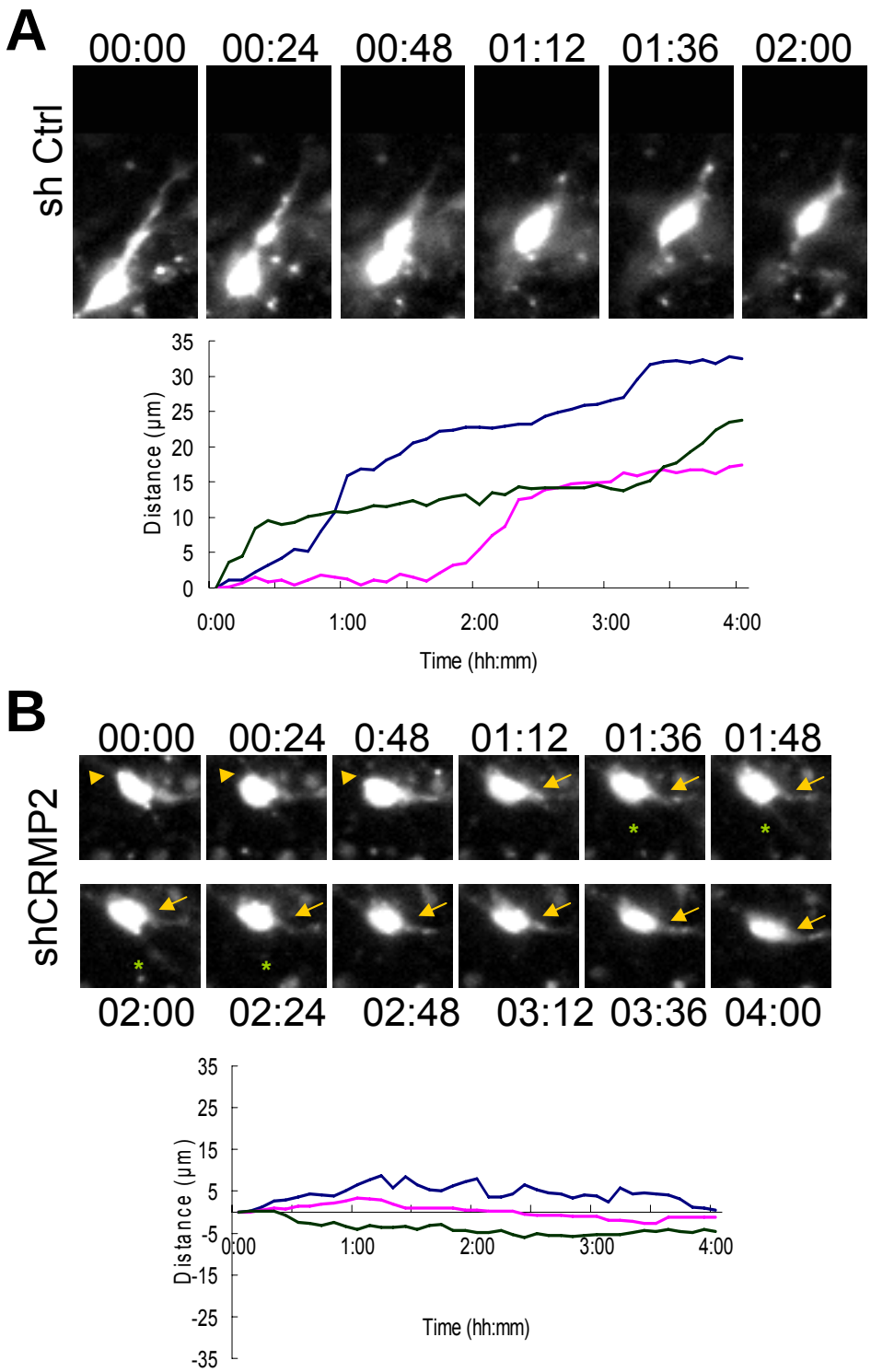



**Fig. Sup7 Live cell imaging of cell behavior within the IZ.** Rat brains were electroporated with CRMP2 (bottom) or control shRNA (top) constructs at E16, and the brains were sectioned and cultured 3 d later. (A) Images from control bipolar cells within the IZ. Cells extended a leading process toward the CP, and the cell body followed, resulting in forward locomotion. (B) Images from cells transfected with CRMP2 shRNA within the IZ. The cell body remained immobile.

# Fig. Sup8

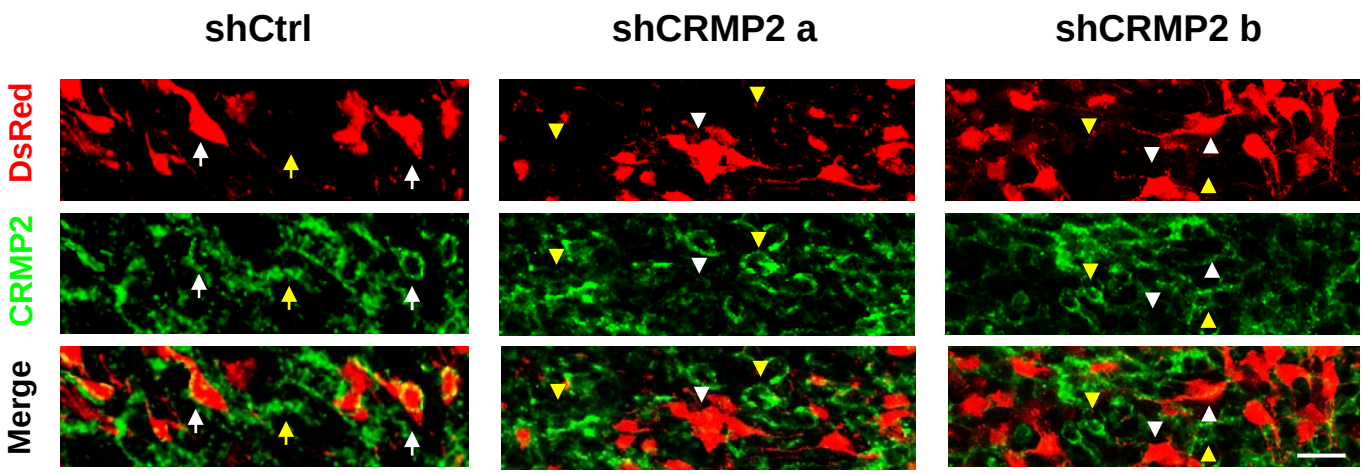
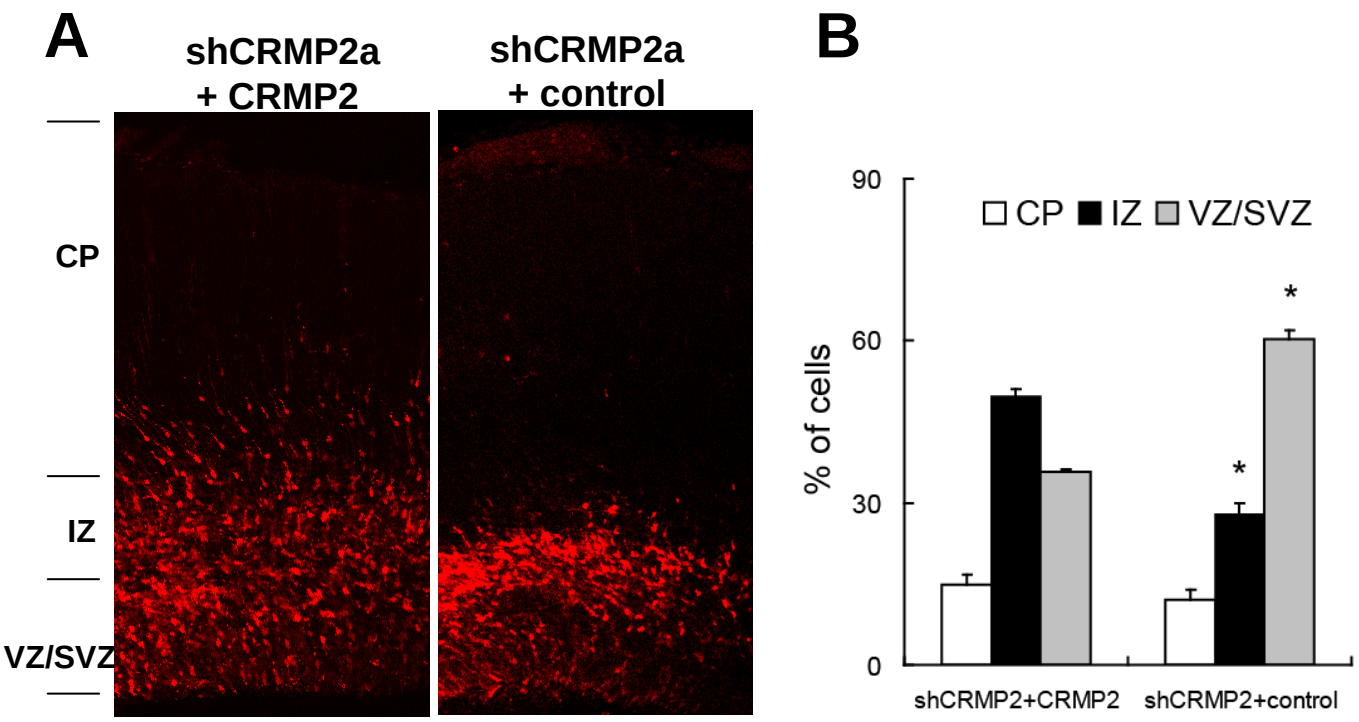



**Fig Sup8. Knock-down of CRMP2 expression by shCRMP2 *in vivo*.** Brain slices were subjected to immunofluorescent staining for CRMP2 3d after transfection at E16. There is no apparent difference between the expression of CRMP2 in shCtrl transfected cells (white arrow) and those non-transfected cells (yellow arrow). In contrast, the expression of CRMP2 in most shCRMP2a/b transfected cells (white arrowhead) is weaker than that in the non-transfected cells (yellow arrowhead).

# Fig. Sup9




**Sup Fig 9. The migration of newborn neurons inhibited by shCRMP2 can be partially rescued by wild-type CRMP2**. Rat embryos electroporated *in utero* with shCRMP2a or shCRMP2b (shCRMP2b) at E16 together with either wild-type CRMP2 expressing vector or control. (A) Coronal sections of rat brain 3 d after electroporation. Down-regulation of CRMP2 caused an evident accumulation of cells in the VZ/SVZ and IZ and cotransfection of wild-type CRMP2 can partially rescue the migration of newborn neurons. (B) Percentages of transfected cells in different regions of the neocortex in (A). Error bars represent SEM. *t* test: *, $P < 0.01$.