

Figure S1. Related to Figure 3. Dimer and Tetramer Models of RPRD1B:CTD Complex Share Similar Features. Comparison of dimeric and tetrameric models of RPRD1B:CTD complex.

(A) Dimer (left) and tetramer (right) models from crystal structures.

(B) Electrostatic potential surface for dimer (left) and tetramer (right) models.

(C) Dimer model CTD peptide fragment superimposed with the electrostatic potential surface around the corresponding binding site.

(D) Tetramer model CTD peptide fragment superimposed with the electrostatic potential surface around the corresponding binding site.

(E) Recognition elements around first K7ac and first S2p in the CTD peptide fragment in the tetramer model.

(F) Recognition elements around second K7ac and second S2p in the CTD peptide fragment in the tetramer model.

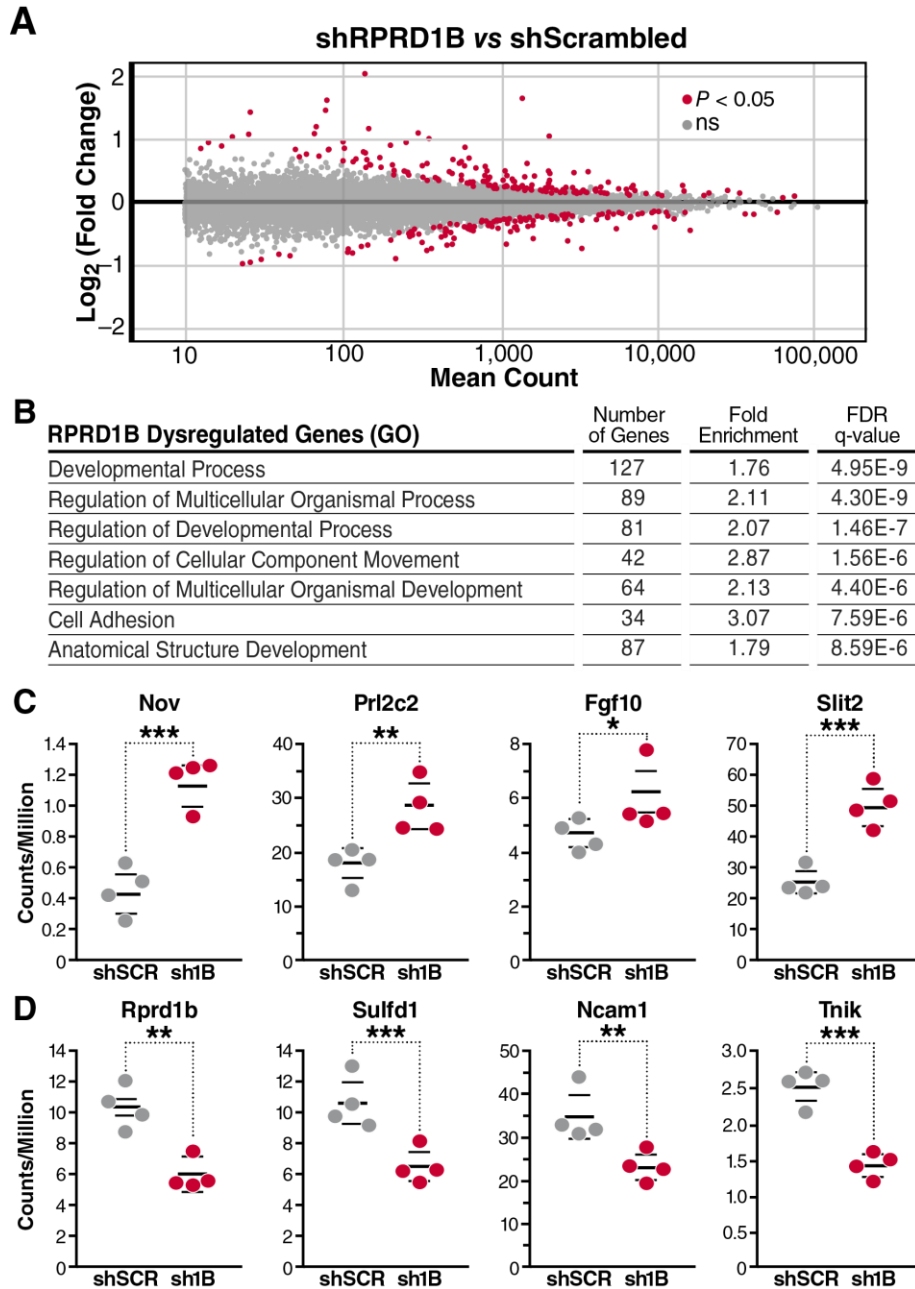


Figure S2. Related to Figure 5. RPRD1B Knockdown Dysregulates Genes Relating to Development and Multicellularity. NIH3T3 cells were treated with shRNAs against RPRD1B (sh1B) or a scrambled sequence (shScr) and selected with puromycin for 1 week.

(A) RNA-seq highlighting 271 significantly dysregulated genes in red.

(B) Gene ontology analysis of genes significantly dysregulated in response to RPRD1B knockdown.

(C) DESeq counts per million for selected upregulated genes associated with regulation of multicellular organismal process.

(D) DESeq counts per million for selected downregulated genes in response to RPRD1B knockdown.

Data is shown as mean \pm SEM. * $p < 0.05$; ** $p < 0.01$; *** $p < 0.005$ using a one-tailed T test.