

Supplementary Figure 1. MAP2-induced protrusions contain stable microtubules.

NIE115 cells transfected with MAP2-GFP and stable microtubules were visualized by immunofluorescence microscopy using antibodies to acetylated (Ace MT) Tubulin.

Images of three representative experiments are shown. Scale bar, 50 μm .

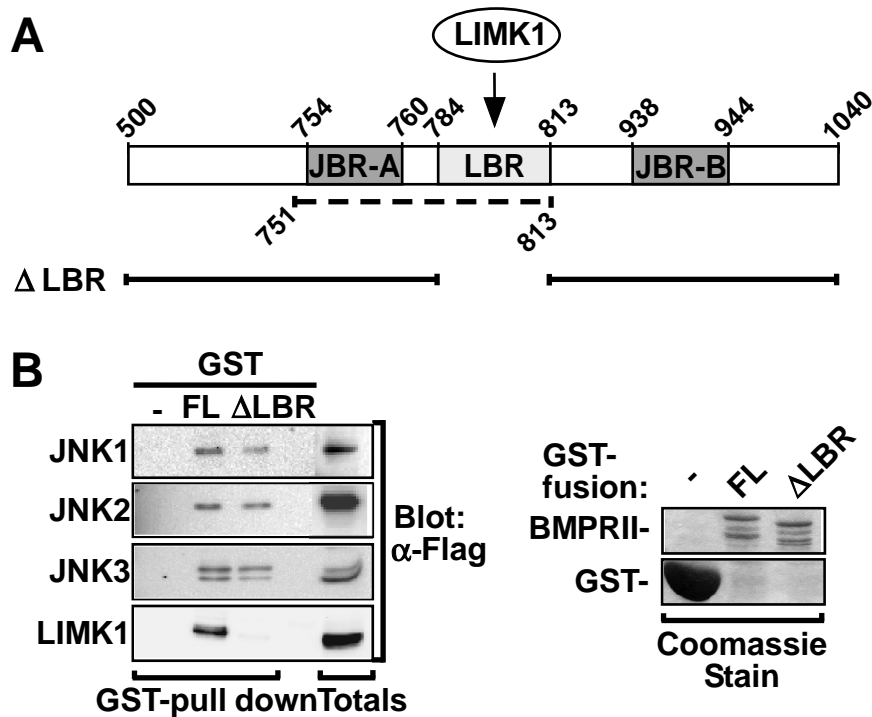
JBR-A

human	QIYPLPKQQNLPKRPTSLPLNTKNSTKE	768
mouse	QIYPLPKQQNLPKRPTSLPLNTKNSTKE	
rat	QLYPLPKQQNLPKRPTSLPLNTKNSTKE	
chicken	QVYPLPKQQNLPKRPTSLPLNTKNSTKE	
frog	QLYPLPKQQNLPKRPTSLPLNTKNSGKE	
zebrafish	AMFPLPKQQNLPKRPTSLPLNTKNPGRE	
pufferfish	TIFPLPKQQNLPKRPSLQLRTKPTKKE	
consensus	---PLPKQQNLPKR RP-SL-L -TK---E	
fly	LGE EKSNLVTALRRPNNLDLNPRLDKPP	
consensus	----- RP--L-L -----	

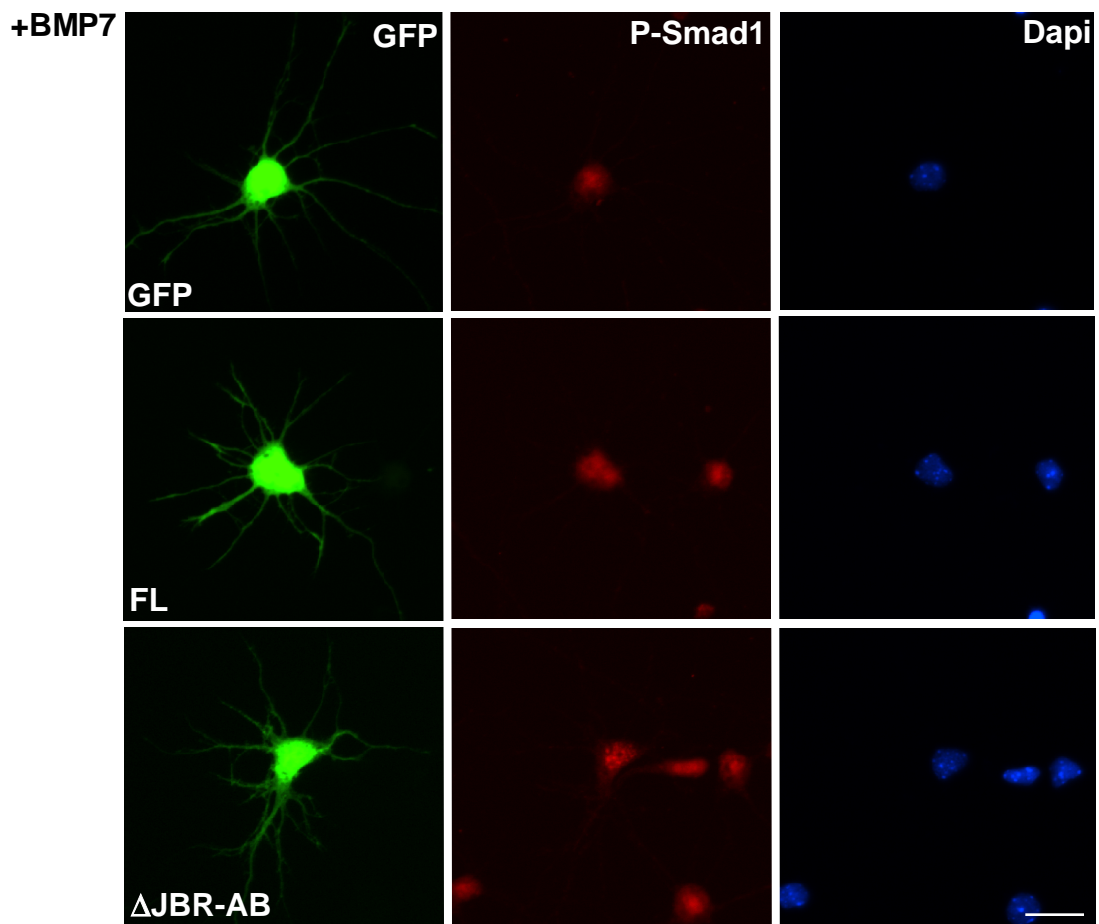
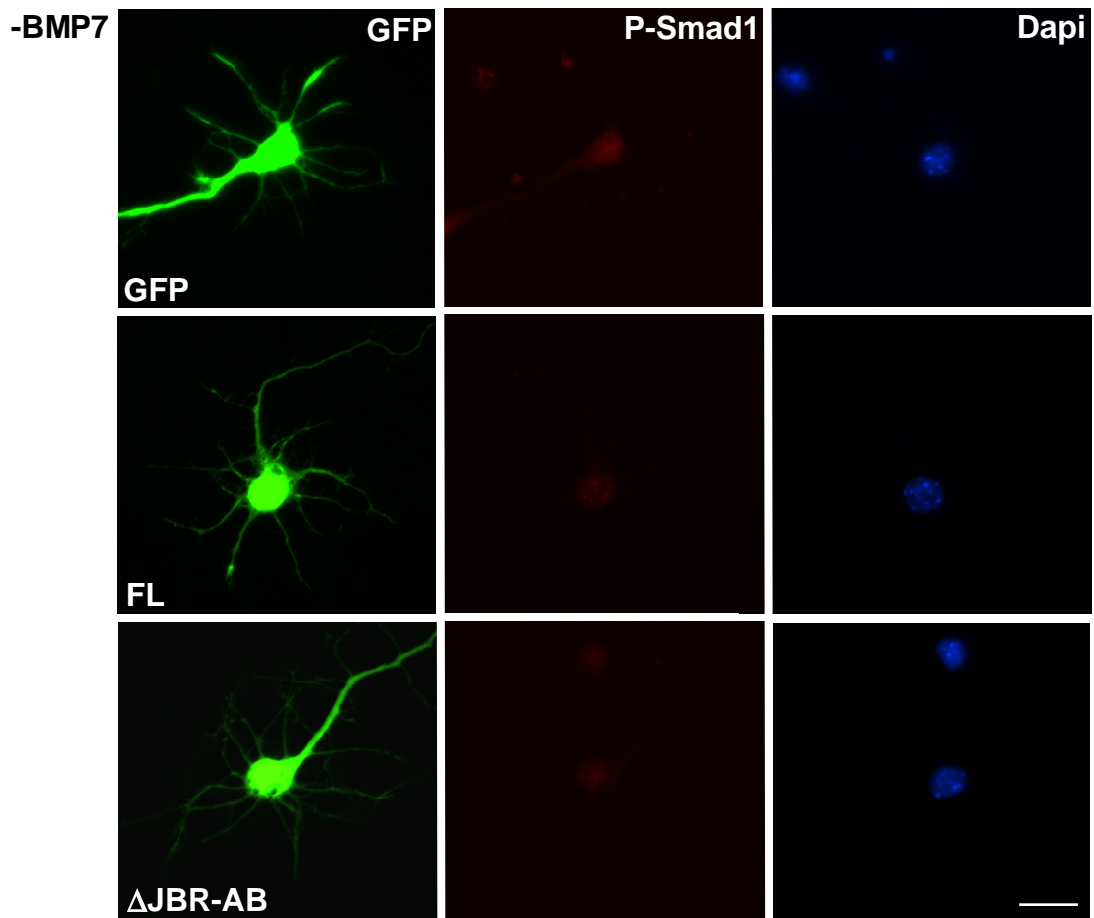
JBR-B

human	AADPGPSKPRRAQRPNSLDLSATNVLDG	951
mouse	AADPGPSKPRRAQRPNSLDLSATNVIDG	
rat	AADPGPAKPRRAQRPNSLDLSATNVIDG	
chicken	ARNPGQTQTRRAQRPNSLDLSATNSLDS	
frog	TR---QRQTRRAQRPNSLDLSSSSIMDN	
zebrafish	LR---PPKPRRPERPNSLDLSASS--QD	
pufferfish	EALLRQPRARRPERPNSLDLSFTT--QD	
consensus	----- RR--RPNSLDLS -----	
fly	TTGQGPTEQQMR RQHSLEV FREVFSGR	
consensus	----- R--SL -----	

Supplementary Figure 2. Multiple alignment of human BMPRII with various homologs. Alignment of human BMPRII with mouse, rat, chicken, frog, zebrafish, pufferfish and fly homologs. The positions of amino acids in the human protein are indicated. Red boxes represent JBR-A and JBR-B. The consensus sequences including and excluding fly are shown in blue. Dashes indicate gaps introduced to maintain optimal alignment as previously described in Wong, W.K., Morse, J.H., Knowles, J.A. (2006). Evolutionary conservation and mutational spectrum of BMPRII gene. *Gene* 368, 84-93.



Supplementary Figure 3. JNK binding is retained upon deletion of LBR on BMPRII. (A) A schematic representation of GST fusion construct of the BMPRII tail lacking the newly refined LBR is shown. Dashed line below BMPRII tail schematic indicates the previously defined LBR. (B) COS-1 cells were transiently-transfected with Flag-JNK1,2,3 or LIMK1-Flag, and cell lysates were incubated with bacterially-expressed GST fusion proteins. The interaction was visualized by anti-Flag immunoblotting. Levels of GST fusion proteins were confirmed by Coomassie blue staining (right). Note that deletion of the LBR abrogated LIMK1 binding, while JNK1, 2 and 3 retained association with the BMPRII tail.



Supplementary Figure 4. Deletion of JBR-AB on BMPRII does not impair canonical Smad signalling. Primary cortical neurons were infected with adenoviruses encoding GFP empty vector, BMPRII full length (FL), or BMPRII Δ JBR-AB and stimulated with BMP7 for 60 min. The localization of phosphorylated Smad1 in GFP expressing cells was visualized by immunofluorescence microscopy using phospho-Smad1,5,8 primary antibody and Alexa Fluor 546-conjugated secondary antibody. Nuclei were stained using DAPI. Scale bar, 20 μ m.