Dieni et al., http://www.jcb.org/cgi/content/full/jcb.201201038/DC1

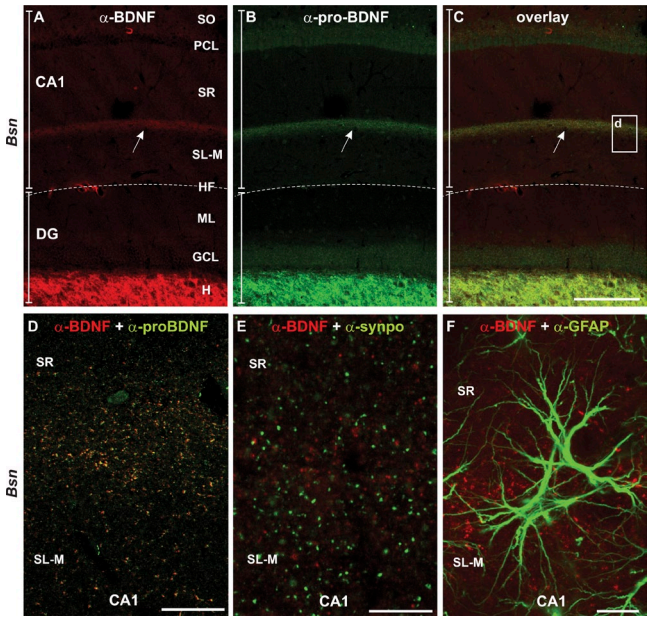

Figure S1. **BDNF labeling is increased in the CA1 region of Bsn mutants.** (A–C) BDNF-IR and pro–BDNF-IR in *Bsn* mutants demonstrates a laminar-specific increase in staining at the border of SR and SL-M (arrows). The dashed lines mark the hippocampal fissure (HF). GCL, granule cell layer; H, hilus; ML, molecular layer; PCL, pyramidal cell layer; SO, stratum oriens. (D) High-resolution confocal image (represented by box d in C) reveals colocalization of BDNF-IR and pro–BDNF-IR at the border of SR and SL-M from a *Bsn* mutant. (E) Colabeling with antibodies against BDNF and synpo reveals segregation of anti-BDNF (presynaptic) and anti-synpo (postsynaptic) signals at the border of SL and SL-M. (F) Colabeling with antibodies against BDNF and GFAP shows a lack of BDNF-IR in astrocytes. Bars: (A–C) 200 µm; (D) 30 µm; (E and F) 10 µm.

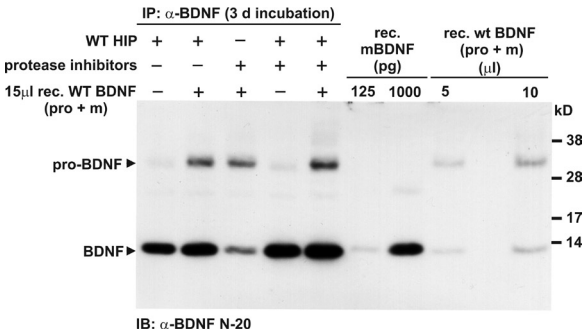

Figure S2. **Recovery of recombinant WT pro-BDNF and mBDNF from hippocampal lysates.** Hippocampal lysates were immunoprecipitated with anti-BDNF for 3 d in the presence or absence of protease inhibitors. IP was performed with or without known amounts of recombinant WT BDNF containing pro-BDNF (pro) and mature BDNF (m). After IP, WB was performed with BDNF-N20 antibodies. Recombinant (rec.) mature BDNF was used as a reference. HIP, hippocampus; IB, immunoblot.