**Suppl. Fig. 1.** Anti-LTβR mAb induced p52-RelB activation is specifically inhibited in p52- and RelB-deficient fibroblasts. Wild-type (A), relB-/- (B), nfkβ1-/- (C), and nfkβ2-/- MEFs (D) were either left untreated (un, lane 1), treated with TNF (lanes 2, 4, and 6), or stimulated with agonistic anti-LTβR mAb (lanes 3, 5, and 7). Nuclear extracts were prepared and analyzed in EMSAs. Complexes were identified using following Abs: lanes 1-3, preimmune serum (p.i.); lanes 4 and 5, anti-RelB; lanes 6-7, anti-RelA (panels A, B, and D) and anti-p52 (panel C). Arrowheads indicate supershifted complexes retained in the slot. I/II, complex I/II (for more details see text).

**Suppl. Fig. 2.** RelB and p52 physically interact in LTβR-stimulated fibroblasts. Fibroblasts were either uninduced (un) or treated with anti-LTβR mAb. Lysates were immunoprecipitated with RelB-specific Abs and the precipitated material was analyzed by Western blotting for p52/p100 levels. Lanes 1 and 2, wild-type; lane 3, nfkβ2-/-; lane 4, relB-/- fibroblasts.

**Suppl. Fig. 3.** Ongoing protein synthesis is required for processing of p100 to p52 and the induction of nuclear RelB. Wild-type fibroblasts were either left untreated (un) or stimulated for 8 h with anti-LTβR mAb in the absence (-) or presence (+) of emetin (10 µg/ml) to block protein translation. Levels of p100 and p52 in whole cell extracts and of RelB in nuclear extracts were detected by Western blotting.