Supporting Information

Conner et al. 10.1073/pnas.1006894107

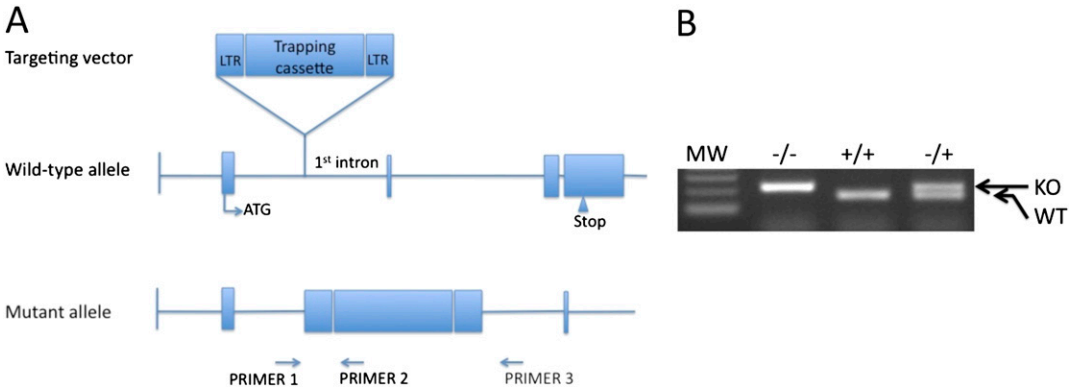

Fig. S1. The targeting vector VICTR48, wild-type *Irak1bp1* allele, and targeted allele are shown. Positions of amplimers from the endogenous locus (primers 1 and 3) and targeting cassette (primer 2) are indicated.

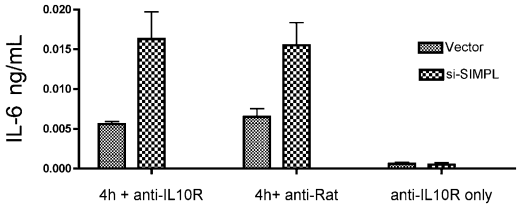

Fig. S2. Blocking the IL-10 receptor pathway does not contribute to the effect of IRAK1BP1 on up-regulation of IL-6. RAW264.7 macrophages stably expressing IRAK1BP1-specific or control (nonhomologous) shRNA constructs were incubated for 30 min with either IL-10 receptor-specific or control (anti-Rat) antibodies followed by stimulation with 100 ng/mL LPS. IL-6 mRNA was measured by ELISA.

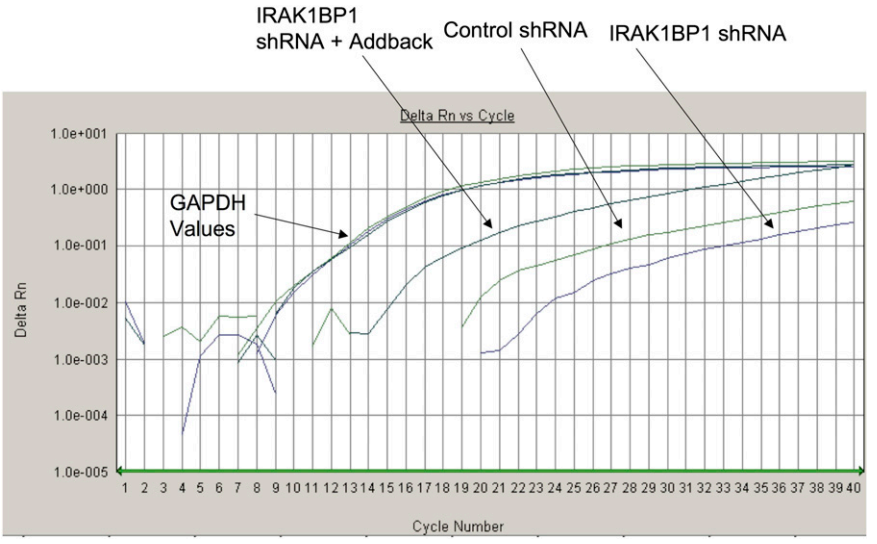

Fig. S3. Assessment of IRAK1BP1 mRNA in RAW264.7 shRNA and add-back expressing cells. RAW264.7 cells were stably transduced with an shRNA construct targeting the ORF of IRAK1BP1 or a control hairpin with no homologous sequence in the mouse genome (1). Add-back cell lines were generated by reinfecting IRAK1BP1 shRNA-expressing cells with a construct containing three silent mutations within the target sequence. Comparison of IRAK1BP1 mRNA levels in unstimulated RAW264.7 cells is shown.

1. Conner JR, Smirnova II, Poltorak A (2008) Forward genetic analysis of Toll-like receptor responses in wild-derived mice reveals a novel antiinflammatory role for IRAK1BP1. *J Exp Med* 205:305–314.

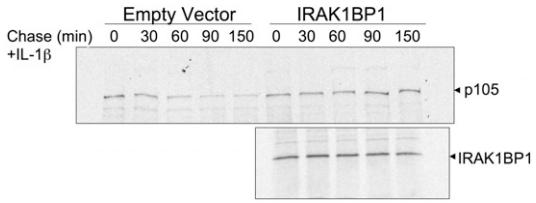

Fig. S4. L929 fibroblasts overexpressing IRAK1BP1 or empty vector controls were transfected with YFP-labeled p105. Cells were pulsed with 35S-methionine for 40 min and stimulated with 100 ng/mL IL-1β during the chase period for the times indicated. Labeled p105 and IRAK1BP1 were detected using immunoprecipitation with anti-YFP and anti-FLAG antibodies, respectively, followed by radiographic exposure.