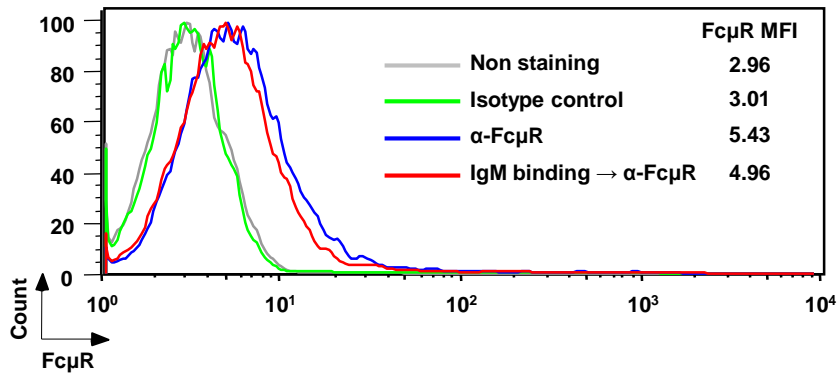
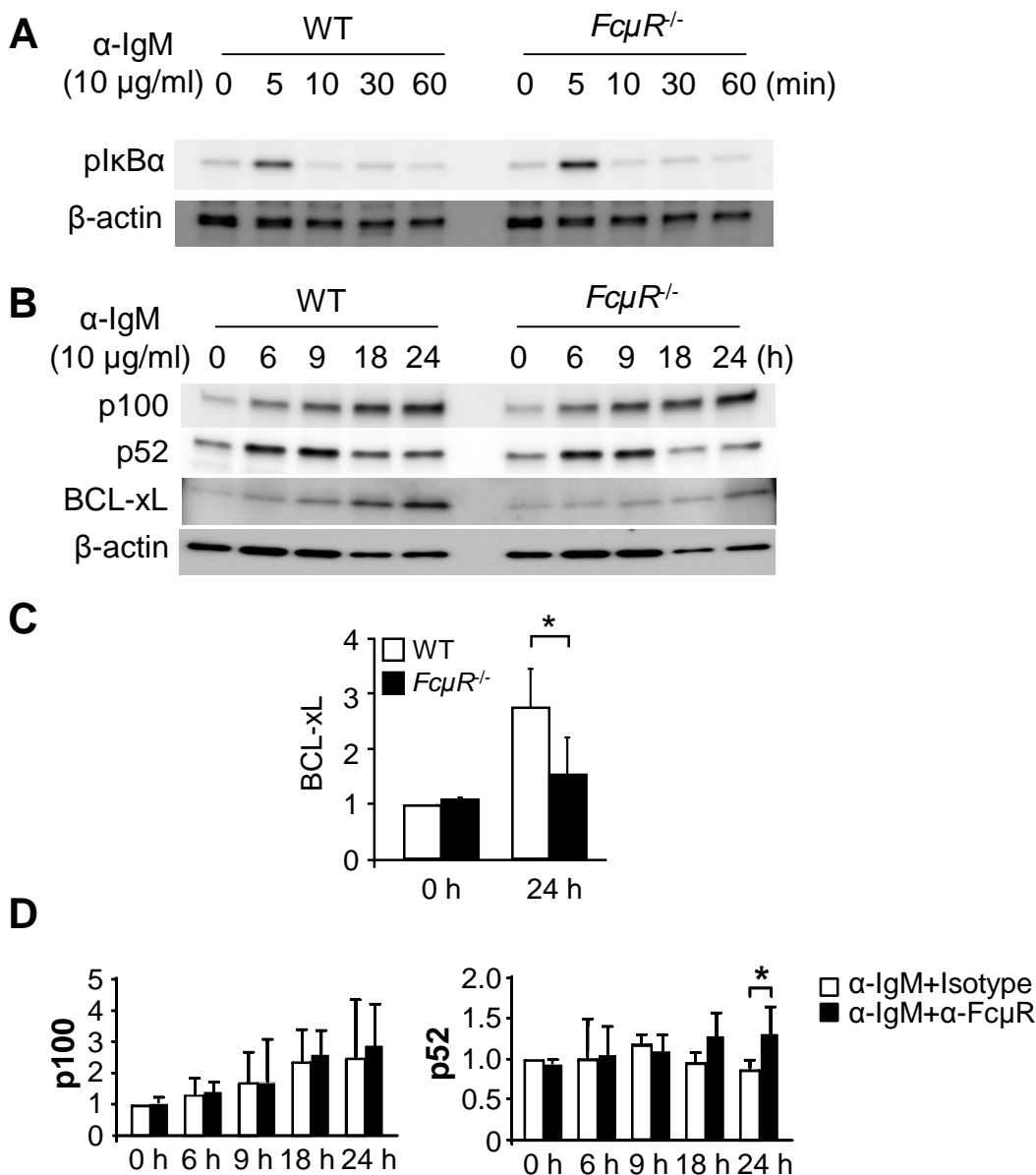


Supplemental Fig. 1. Crosslinking Fc μ R enhanced anti-Ig κ - but not anti-IgD-induced B cell activation. (A) Purified B cells were cultured for 3 days in medium alone or in the presence of 10 μ g/ml of the anti-IgD antibody AMS-9.1 or an isotype control of AMS-9.1, and analyzed for cell sizes (FSC, upper panels) and CFSE dilution (lower panels). (B) Purified B cells were cultured for 3 days in medium alone or in the presence of 10 μ g/ml of AMS-9.1 + 30 μ g/ml of either the 4B5 anti-Fc μ R antibody or an isotype control of 4B5, and then analyzed for cell sizes. (C) Purified B cells were cultured in medium alone or in the presence of 10 μ g/ml of F(ab')₂-anti-mouse κ antibodies for 3 days, and analyzed for cell sizes (upper panels) and CFSE dilution (lower panels). (D) Purified B cells were cultured for 3 days in medium alone or in the presence of 10 μ g/ml of F(ab')₂-anti-mouse κ antibodies + 30 μ g/ml of either the 4B5 anti-Fc μ R antibody or an isotype control of 4B5, and then analyzed for cell sizes.



Supplemental Fig. 2. The 4B5 anti-FcμR antibody (rat IgG_{2a}) binds to FcμR even after IgM binding. WEHI279 B cells were first stained with anti-mouse CD16/CD32 antibody (clone 2.4G2, rat IgG_{2b}) to block FcγR and then left unstained (grey), or stained with an isotype control (rat IgG_{2a}) antibody (green), the 4B5 anti-FcμR antibody or mouse IgM first followed by anti-FcμR antibody. The cells were then stained with FITC-anti-rat IgG_{2a}.



Supplemental Fig. 3. *FcμR* is involved in the activation of the noncanonical NF-κB pathway. WT and *FcμR*^{-/-} B cells were stimulated with α-IgM (10 μg/ml) for the indicated durations and analyzed for IκBα phosphorylation (A) and p100, p52 and BCL-xL protein expression (B). (C) Quantification of BCL-xL protein expression relative to β-actin. The expression at time 0 in WT B cells was set as 1. Mean ± SD of three independent experiments are shown. **p* < 0.05. (D) Crosslinking *FcμR* enhances p52 induction at a later time point after BCR stimulation. WT B cells were stimulated for the indicated times with F(ab)₂-α-IgM (10 μg/ml) in the presence of α-*FcμR* (30 μg/ml) or an isotype control Ab, and analyzed for p100 and p52 protein expression relative to β-actin. Mean ± SD of three independent experiments are shown. **p* < 0.05.