

SUPPLEMENTAL MATERIAL

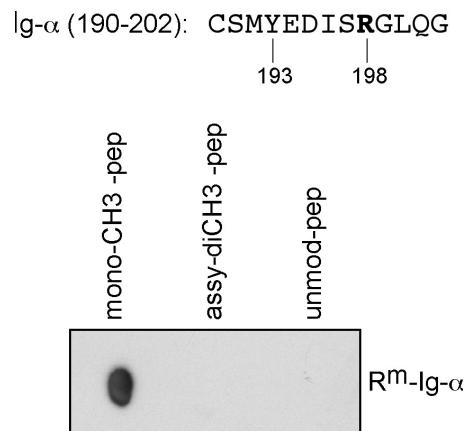
Infantino et al., <http://www.jem.org/cgi/content/full/jem.20091303/DC1>

Figure S1. Anti-methyl-Ig α antibody specifically recognizes monomethylated R198 Ig α . Amino acid sequence of the Ig α methyl peptide used to generate the anti-methyl-Ig α -specific antibody. A dot blot of mono-methyl, asymmetric-dimethyl, or unmodified Ig α peptides (190–202 aa) is shown using an anti-R^m-Ig α antibody raised against monomethylated R198 Ig α . One representative dot blot out of three is shown.

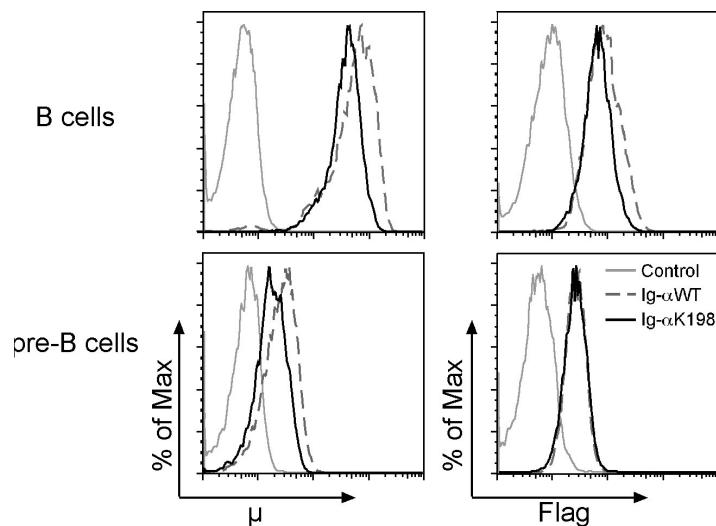


Figure S2. Analysis of BCR and pre-BCR expression. Ig α KO cells (control) reconstituted with the WT or mutant forms of Ig α and $\lambda 1$ were analyzed for BCR surface expression. Flow cytometry with an anti- μ antibody and an anti-flag antibody indicates surface IgM and Ig α , respectively (top). The pre-BCR containing WT or mutant Ig α was detected using an anti- μ antibody, and surface expression of Ig α is shown with an anti-flag antibody (bottom). Similar results were obtained in three independent experiments.

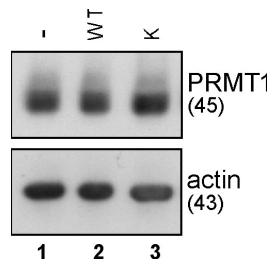


Figure S3. PRMT1 expression in B cells. Western blot with whole-cell lysate from the Ig α KO cells (–) reconstituted with λ 1 and the WT or mutant form of Ig α . Endogenous PRMT1 was detected using an anti-PRMT1 antibody. The loading control is shown with an antiactin antibody. Apparent molecular weights are indicated. Comparable results were obtained in three independent Western blots.

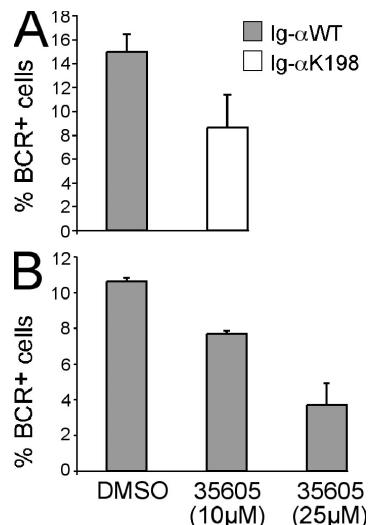


Figure S4. In vitro differentiation assay. The percentage of BCR $^{+}$ cells 5 (A) and 4 d (B) after IL-7 withdrawal is plotted. Error bars indicate means \pm SEM from three independent experiments.