Supplemental Figure S1. Further analysis of Ets1-mediated inhibition of ASC differentiation. (A) Comparison of Ets1 levels in freshly-isolated, resting splenic B cells versus those infected with viruses. For viral infections, splenic B cells were stimulated and infected as described in Fig. 1E. **(B)** qRT-PCR analysis of the levels of membrane-bound (muM) and secreted IgM (muS) expressed by sorted GFP+ B cells stimulated with CpG ODN and infected with an empty retroviral vector (MIGR1) or an Ets1-containing retroviral vector (MIGR1-Ets1). Shown is average and SEM of 3 independent experiments. * $p < 0.05$

Supplemental Figure S2. Ets1 inhibits LPS-driven ASC formation. (A) Ets1 and Ets2 retroviral constructs used for transduction experiments. *LTR*, long terminal repeat; *IRES*, internal ribosomal entry site. **(B)** ELISA analysis of IgM levels in the supernatants of splenic B cells stimulated with LPS and infected with empty retrovirus (MIGR1) or retroviruses encoding Ets1 or Ets2. Shown are the results of 2 experiments. * $p < 0.05$, ** $p < 0.01$.

Supplemental Figure S3. Aligned amino acid sequence of Ets1 and Ets2 showing strong conservation around the MAP kinase phosphorylation site, Pointed domains, autoinhibitory domains and Ets DNA binding domain (which is shown in the yellow highlighting).

Supplemental Figure S4. Post-translational modification of Ets1 at the N-terminus is not required for its activity in blocking ASC formation. (A) Diagram of retroviral constructs used

to infect B cells. The T38A mutation disrupts the Erk phosphorylation site and the K15A mutation disrupts the sumoylation site. **(B)** Splenic B cells were stimulated with CpG ODN and infected with a retrovirus encoding full-length Ets1 or one of the point mutations of Ets1 or an empty retrovirus. Cells were cultured and IgM secretion measured as described in Figure 1E. Shown is the average and SEM of 2 independent experiments. * $p < 0.05$, ** $p < 0.01$.