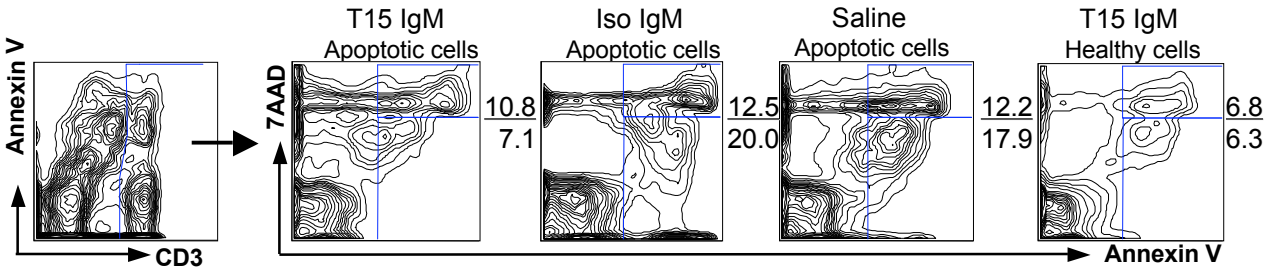
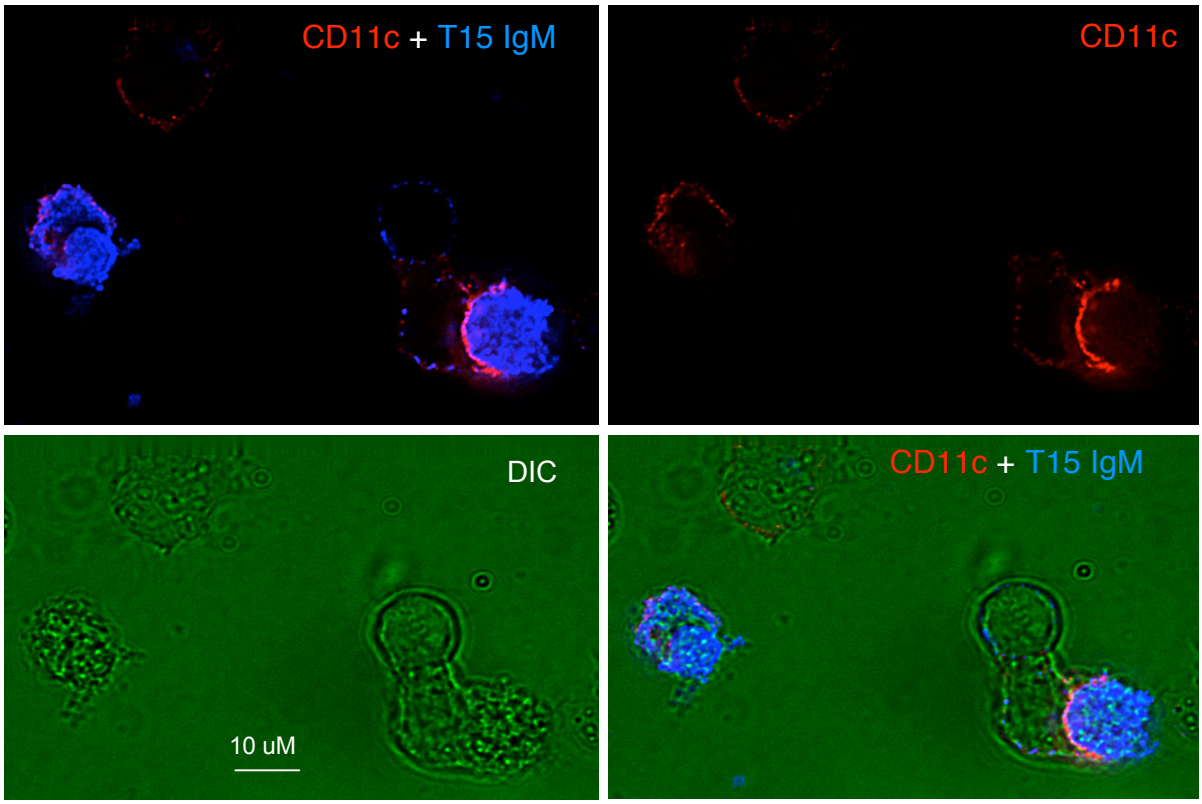
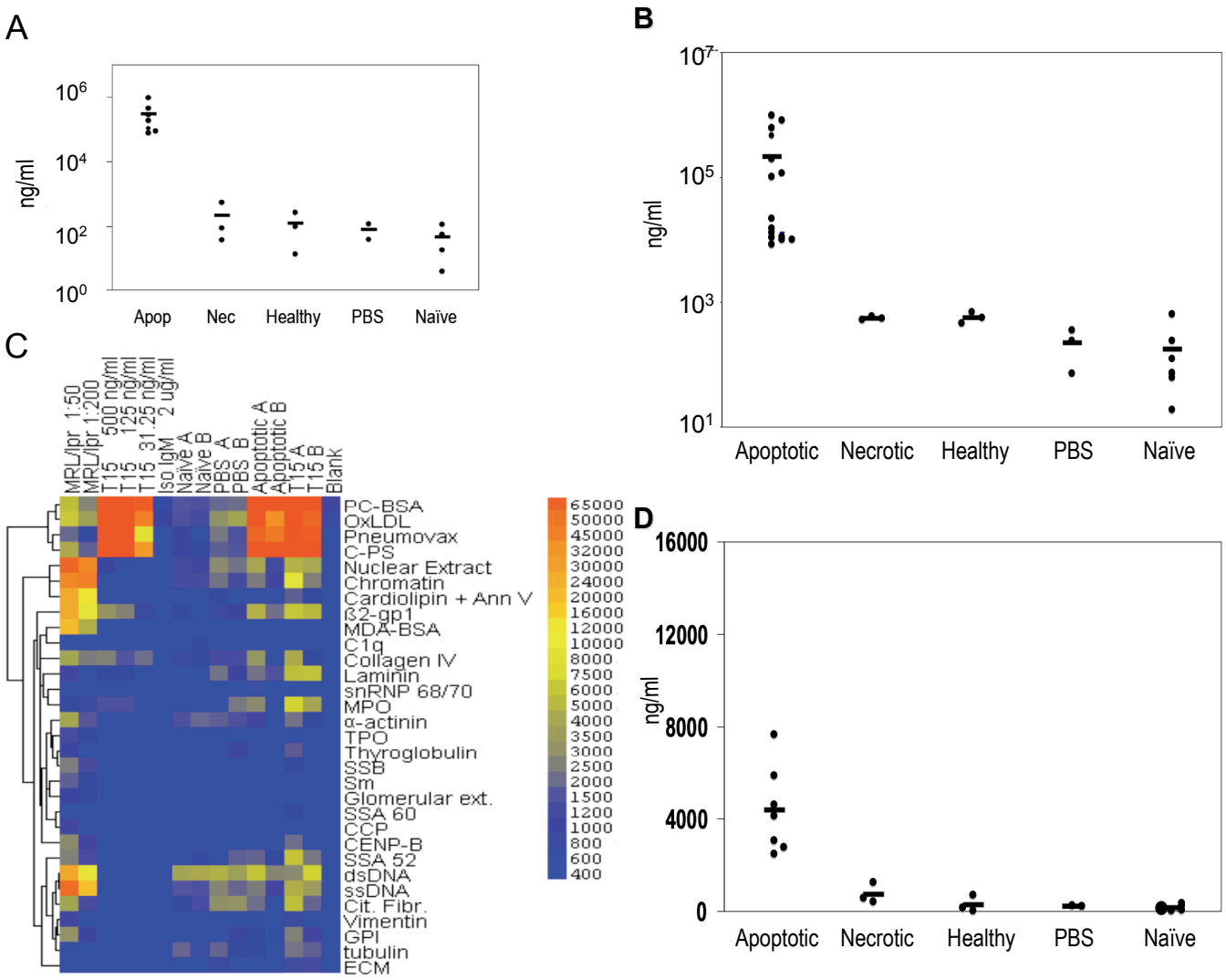


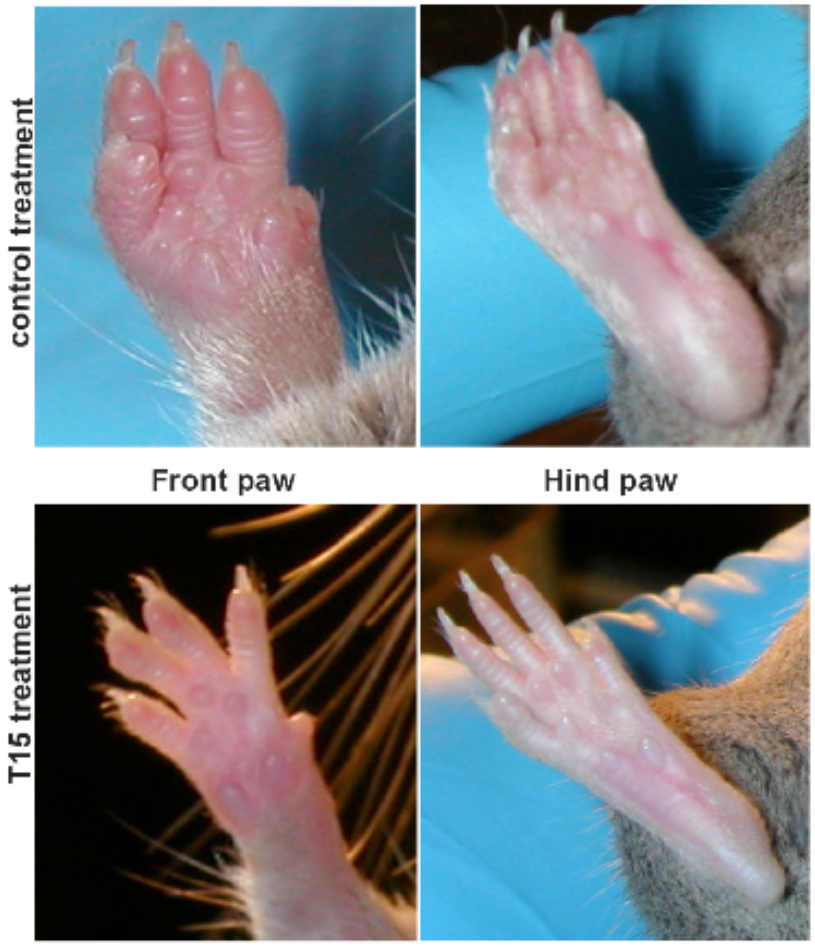
Supplemental 1: T15-NAb enhances the clearance of both early and late apoptotic thymocytes. Treatment of B-cell deficient mice with T15-IgM but not isotype control enhances in vivo clearance of apoptotic thymocytes. Mice received 1 mg IgM (T15-NAb or isotype control) or saline intravenous 16 hr before i.p. instillation of fresh thymocytes or apoptotic thymocytes. At 10 min after thymocyte instillation, peritoneal cells were recovered with ice cold HBSS/EDTA, and flow cytometric analyses using staining for CD3 (for thymocytes/T cells), and to identify dying cells with 7 AAD and Annexin V (top panel). To evaluate clearance of instilled dying cells, after gating on CD3+ cells that primarily identified transferred thymocytes, the clearance of thymocytes at early stages (7AAD- AnnexinV+) and late stages of death (7AAD+AnnexinV+) were evaluated. Values represent the proportion of mononuclear peritoneal CD3+ cells at early (Annexin V+ 7AAD-) and late (Annexin V+ 7AAD+) stages of death from a representative mouse in each group. T15-NAb greatly enhanced clearance of early apoptotic cells compared to saline/negative control treatment (P=0.002), or isotype control (P=0.004). There were no differences in the numbers of overall recovered peritoneal cells. Four or more mice in each group were evaluated.



Supplemental 2: Cultures of purified BM-derived CD11c+ immature DC contain T15-NAb coated apoptotic cells, which are identified by characteristic morphologic changes of nuclear condensation, but not viable cells. The phagocytic capabilities of these cultured DC enables engulfment of cells that spontaneously die in culture, which was confirmed by Annexin V and TUNEL staining. Apoptotic cell-T15-NAb complexes, identified by APC fluorescence, are engulfed by viable DC via pseudopodia lined with CD11c. Under these same conditions, apoptotic cells were not stained by the IgM**-i**sotype control (not shown). Differential interference contrast (DIC) microscopy.



Supplemental 3: Apoptotic cells but not necrotic cells induce high levels of T15 anti-PC antibodies. (**A**) T15-clonotypic anti-PC antibodies are induced by apoptotic cells. For assays, wells were coated with PC-albumin and developed with biotin-tagged anti-T15 clonotypic antibody (AB1-2). Values for individual mice were derived from T15-IgM standard curves, with horizontal bars representing mean values. (**B**) Apoptotic-cell infusions induce high levels of IgM anti-PC antibodies. For assays, wells were coated with PC-albumin and developed with tagged anti-IgM antibody. Serum values for individual mice were derived from T15-IgM standard curves, with horizontal bars representing mean values. (**C**) IgM-binding with an autoantigen microarray with replicate arrays reacted with monoclonal Abs (concentrations shown) or post-treatment sera at 1:200, unless otherwise indicated. Sera were obtained from naïve age-matched mice, or at d16 from mice that received three infusions of T15-IgM (T15 A and T15 B), saline (PBS A & PBS B), apoptotic (Apop) thymocytes, primary necrotic (Nec) thymocytes or freshly isolated (Healthy) thymocytes, on d0, 7 and 14. Naïve 6-month-old lupus-prone MRL/lpr mice, which have spontaneous high levels of IgM anti-PC antibodies, are included for comparisons. Results are representative of two independent experiments, with a limited version previously reported . **(D**) IgG anti-PC antibodies are induced by apoptotic cells. Values from ELISA as described above, using an IgG anti-PC Moab standard. Sera were obtained d16 from mice that received treatment of apoptotic (Apop), necrotic (Nec) or freshly isolated (Healthy) thymocytes of d0, 7 and 14.



Supplemental 4: T15-NAb protects from inflammatory arthritis. DBA/1 mice were immunized with CII and boosted with CII on d20. T15-NAb at 2mg/dose or control treatments were administered weekly. Images of representative paws from CIA mice in T15-NAb-treatment and IgM-isotype control-treatment groups. Other data from these studies are presented in Figure 8.