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Macrophage scavenger receptor 1 (Msr1, SR-A) influences B cell autoimmunity by regulating soluble autoantigen concentration

Supplemental Figure 1: Decreased incidence and severity of arthritis and endocarditis in *Msr1*^{-/-} K/BxN mice. (A) Histological analysis of ankle (left panels) and mitral valve (right panels) pathology in *Msr1*^{+/+}, *+/-*, and *-/-* K/BxN mice demonstrates protection from both joint and cardiac valve pathology in non-arthritic *Msr1*^{-/-} K/BxN mice. For ankles, original magnification = 10x, bar (in fourth row) indicates 200 microns. For mitral valves, original magnification = 40x; bar (in fourth row) indicates 100 microns. (B) Five non-arthritic *Msr1*^{-/-} K/BxN mice (filled circles) were aged up to 15 weeks, during which any mild arthritis resolved or remained equivalent to the 8-week time point, as demonstrated by changes in ankle thickness. The data from the *Msr1*^{+/+} K/BxN mice (reproduced from Figure 1B) are shown for comparison. Plotted values are means \pm SEM. * $p \leq 0.05$, ** $p \leq 0.01$.

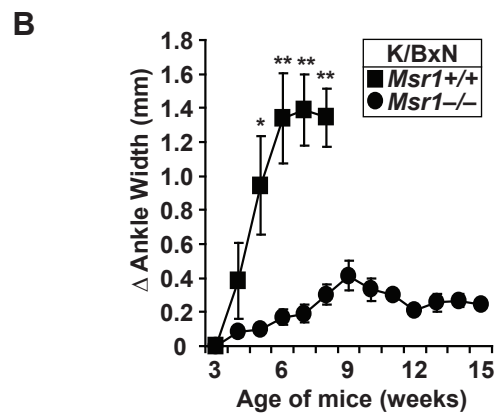
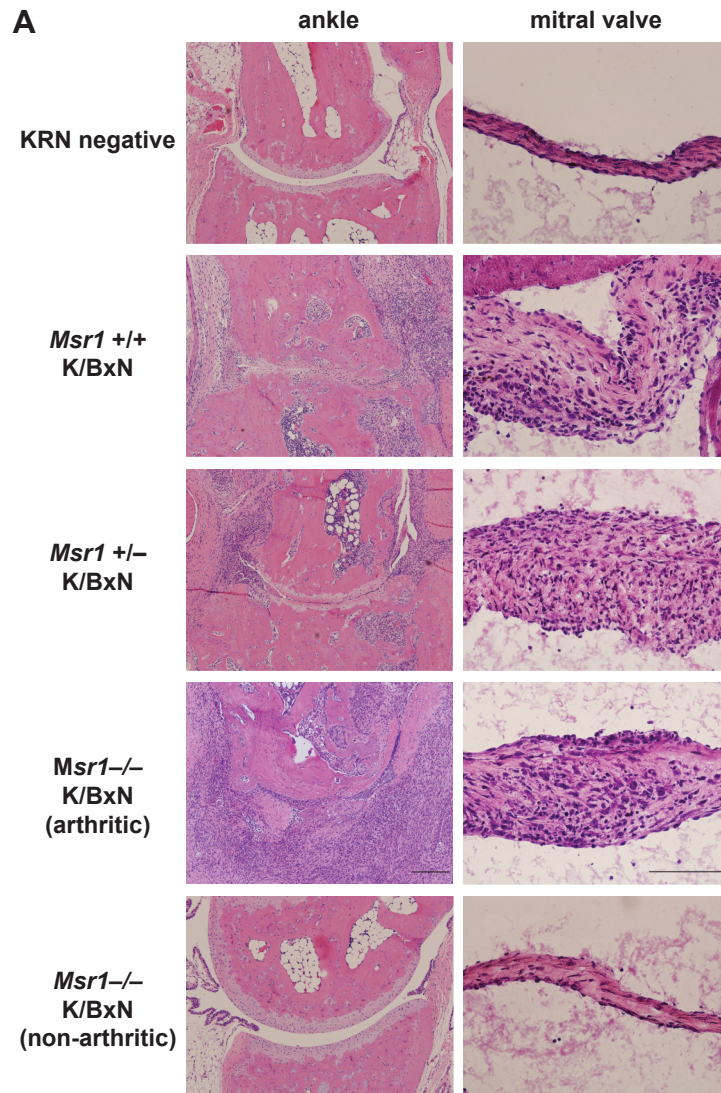
Supplemental Figure 2: *Msr1*-sufficient host environment permits *Msr1*-deficient K/BxN hematopoietic compartment to drive arthritis and B cell activation

Rag1^{-/-} recipient mice were sublethally irradiated (300 Rad) and transplanted with 10×10^6 bone marrow cells from *Msr1*^{+/+} K/BxN mice or *Msr1*^{-/-} K/BxN mice as indicated. (A) The development of arthritis in both groups was determined by weekly arthritis scoring and ankle measurements. Values shown are means \pm SEM. $n = 3$ mice/group. (B) B cells in the reconstituted mice were analyzed for their ability to bind to GPI-tetramer, for having undergone isotype switching express IgG1 intracellularly, and transitioning to a CD38^{low} GL7⁺ activated

phenotype. Numbers indicate the percentage of cells in each quadrant or gate. **(C)**

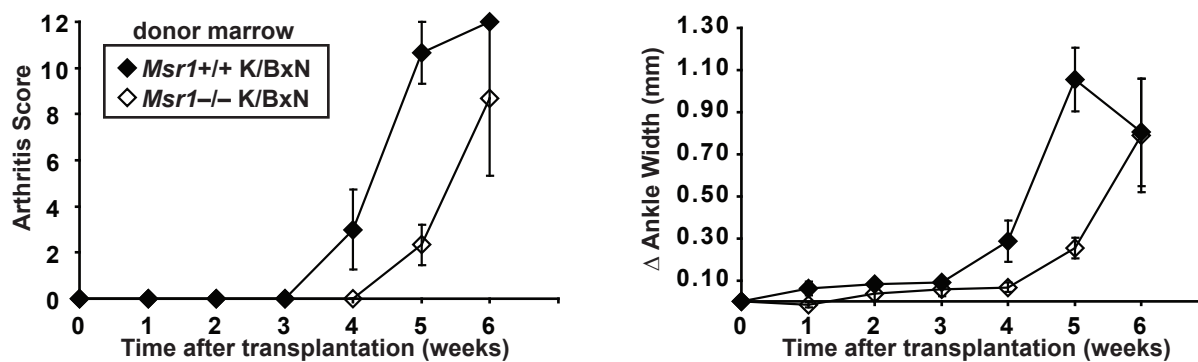
Representative flow cytometric plot of CD3⁻ F4/80⁺ macrophages in the reconstituted mice analyzed for expression of the congenic markers differentiating the donor cells (CD45.1⁺ CD45.2⁺) from the host cells (CD45.1⁻ CD45.2⁺). The numbers shown are percentages. In analysis of multiple mice, the percent of macrophages that were host-derived ranged from 38-63%. **(D)** Similar analysis for CD3⁺ T cells and B220⁺ B cells reveals that they are entirely donor-derived.

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Supplemental Figure 1

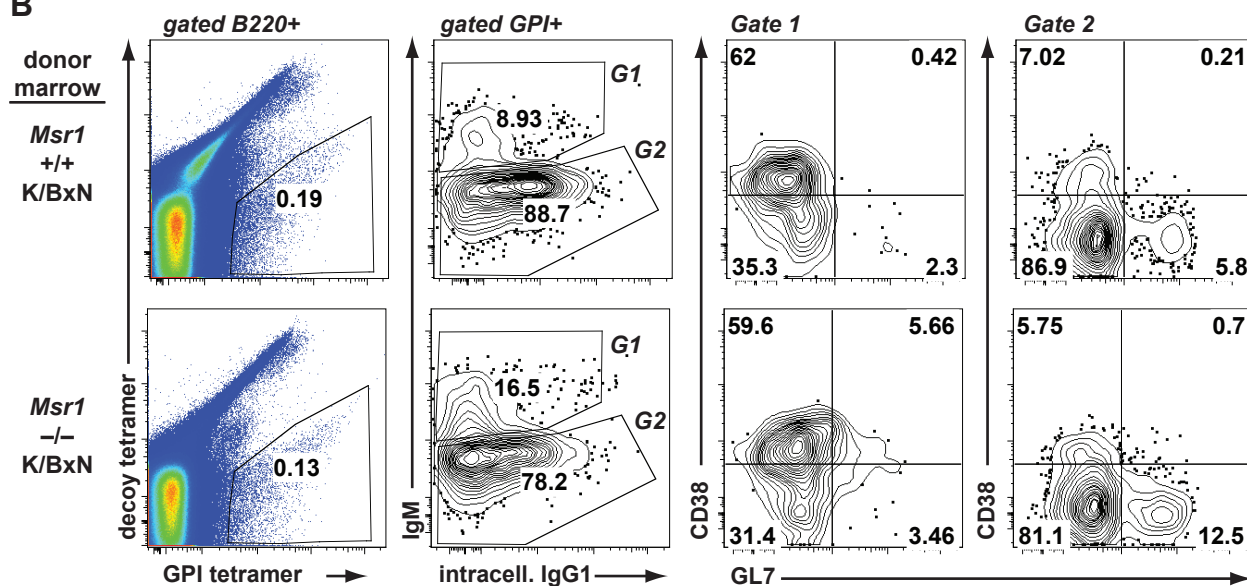


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Supplemental Figure 2

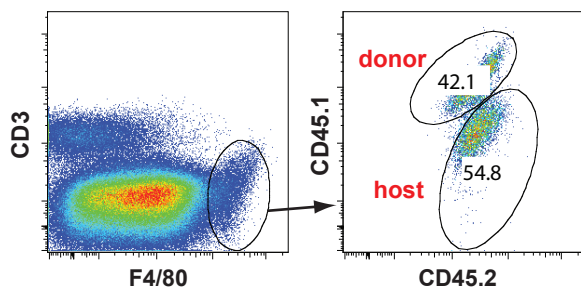
A



B



C



D

