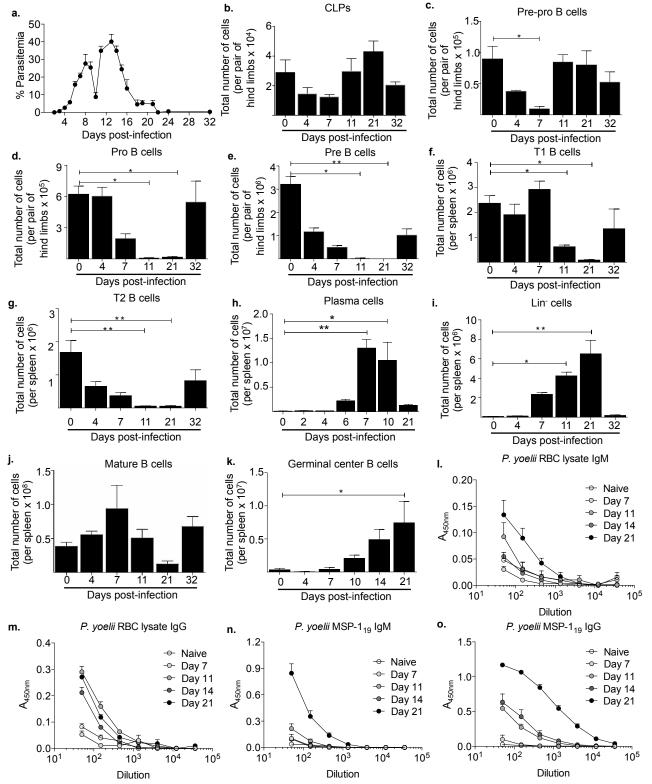
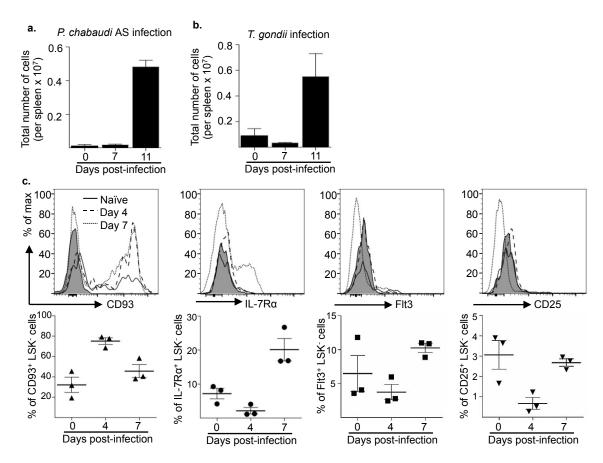


Supplemental Figure 1. Gating strategies to identify progenitor cell and B cell precursor populations. a. Live bone marrow cells stained with Lin markers (CD3e, CD4, CD5, CD8, B220, NK1.1, CD11b, CD11c, Gr-1, and Ter-119), Sca-1, c-kit, CD93, IL-7Rα; lymphocytes were gated based on FSC-A and SSC-A. Lin⁻ population was gated based on the FMO control. LSK⁻cells (Lin⁻Sca-1⁺c-kit), HSCs (Lin⁻Sca-1⁺c-kit⁺CD93⁻), MPPs (Lin⁻Sca-1⁺c-kit⁺CD93⁺), and CLPs (Lin⁻Sca-1¹oc-kit¹olL-7Rα⁺) were selected based on the gating strategy shown. **b.** Live bone marrow cells were stained with Lin markers (CD3e, CD4, CD5, CD8, NK1.1, CD11b, CD11c, Gr-1, and Ter-119), CD19, B220, CD93, IgM, CD43, and CD24. Lymphocytes were gated based on characteristic FSC-A and SSC-A morphology. Gating strategy for selecting pre-pro B cells (Lin⁻B220⁺CD19⁻CD43⁺CD93⁺IgM⁻IgD⁻CD24⁺), pro-B cells (CD19⁺B220⁺CD93⁺CD43¹n¹CD24¹n¹IgM⁻IgD⁻), immature B cells (CD19⁺B220⁺CD93⁺IgM⁻IgD⁻) and mature B cells (CD19⁺B220⁺CD93⁻IgM†IgD⁻) is shown. **c.** Lymphocytes in the spleen were gated based on FSC-A and SSC-A morphology. T1 B cells (CD19⁺B220⁺CD93†IgM⁺CD23⁻), T2 B cells (CD19⁺B220⁺CD93†IgM⁺CD23⁻), and T3 B cells (CD19⁺B220⁺CD93†IgM⁻CD23⁺) were gated as shown based on the indicated phenotypes.

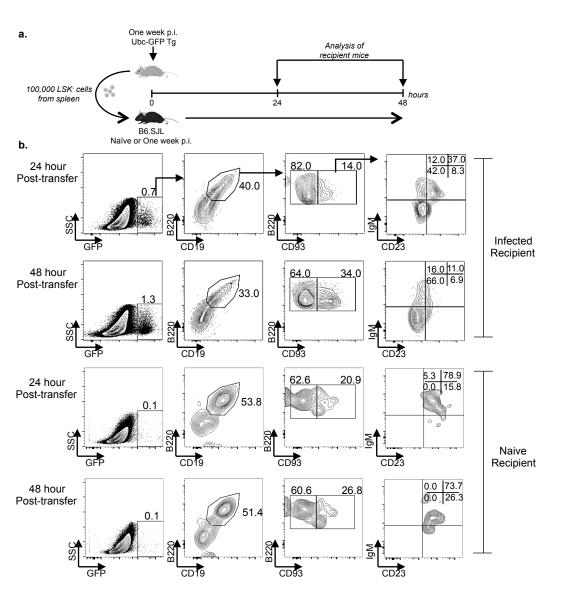




Supplemental Figure 2. Effect of *Plasmodium yoelii* 17X infection on lymphoid progenitors and B-cell precursors in the bone marrow and spleen of C57BL/6 mice a. Representative parasitemia curve following i.p. infection of 10⁵ pRBC into WT C57BL/6 mice. Total number of **b.** CLPs, **c.** Pre-pro B cells, **d.** Pro B cells, and **e.** Pre B cells, from the bone marrow of naïve (day 0) or *P. yoelii* 17X infected mice based on the gating strategy shown in Supplemental Figure 1. Total number of splenic **f.** T1 B cells, **g.** T2 B cells, **h.** Plasma cells. **i.** Lineage negative (Lin⁻) cells, **j.** Mature B cells, and **k.** Germinal center B cells from naïve (day 0) or *P. yoelii* 17X infected mice based on the gating strategy shown in Supplemental Figure 1. Relative (**I, m**) *P. yoelii* 17X infected RBC lysate (**n, o**) or MSP-1₁₉—specific antibody response as measured by ELISA at days 0, 7, 11, 14 and 21 p.i. Data are representative of two independent experiments, with n = 3-5 mice per group/ time point; Error bars (**a-o**) S.E.M.



Supplemental Figure 3. LSK⁻ cell phenotype with different parasite infections. Total number of LSK⁻ cells in the spleen of naïve or C57BL/6 mice after days 7 and 11 of a. *Plasmodium chabaudi* AS infection or b. *Toxoplasma gondii* infection. c. Representative histogram plots and percentage of bone marrow derived LSK⁻ cells showing expression of CD93 (triangle), IL-7R α (circle), Flt3 (square), and CD25 (inverted triangle) in naïve (solid line) and C57BL/6 mice after 4 (dashed line), and 7 (dotted line) days of *P. yoelii* 17X infection. The FMO (fluorescent minus one) controls are shown as grey filled histograms. Data are representative of two independent experiments (c), with n = 3-5 mice per group/ time point; Error bars (a-c) S.E.M.



Supplemental Figure 4. Splenic LSK⁻ cells differentiate into B cells within 24 hours during *Plasmodium* infection. a. LSK⁻ cells were sorted from the spleen of Ubc-GFP Tg mice, one week post *P. yoelii* 17X infection, and 100,000 cells (GFP+CD45.2+) were transferred into naïve congenic (CD45.1) mice or at the same stage of infection (day 7 p.i.). The spleens of recipient mice were harvested 24 and 48 hour post-transfer. b. The splenocytes were enriched for donor (CD45.2+) cells and fluorochrome conjugated antibodies were used to subanalyze the donor (GFP+) cell population based on the gating strategy displayed.