Contribution of Classical Complement Activation and IgM to the Control of *Rickettsia* Infection

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SUPPLEMENTAL DATA

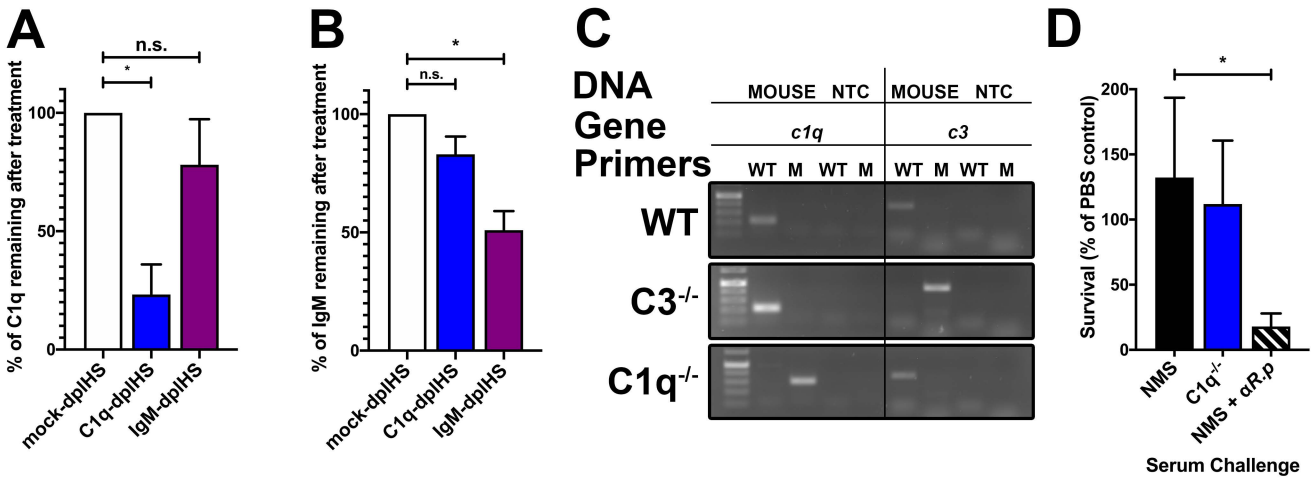

Figure S1 (A) Densitometric analysis of anti-C1q western blots to determine depletion of C1q protein in C1q-dplHS and IgM-dplHS as compared to mock-dplHS. * $p<0.05$ by 1 way ANOVA with multiple comparison to mock-dplHS control. (B) Densitometric analysis of anti-IgM western blots to determine depletion of IgM protein in C1q-dplHS and IgM-dplHS as compared to mock-dplHS. * $p<0.05$ by 1 way ANOVA with multiple comparison to mock-dplHS control. (C) Genotype analysis of WT, $C3^{-/-}$, and $C1q^{-/-}$ mice. Using (from top) mouse DNA or no template control (NTC); primer sets for the *c3* or *c1q* gene; with primer sets for the wild type (WT) or mutant (M) allele. $C3^{-/-}$ are homozygous for the *c3* mutant allele and $C1q^{-/-}$ are homozygous for the *c1q* mutant allele. (D) *In vitro* evaluation of complement-mediated killing of *R. parkeri* by incubation of $3x10^6$ of the bacteria in normal mouse serum (NMS, black), $C1q^{-/-}$ mouse serum (blue), and NMS plus anti-*R. parkeri* antibody (NMS + αRp, striped). *$p<0.05$ by unpaired t test.