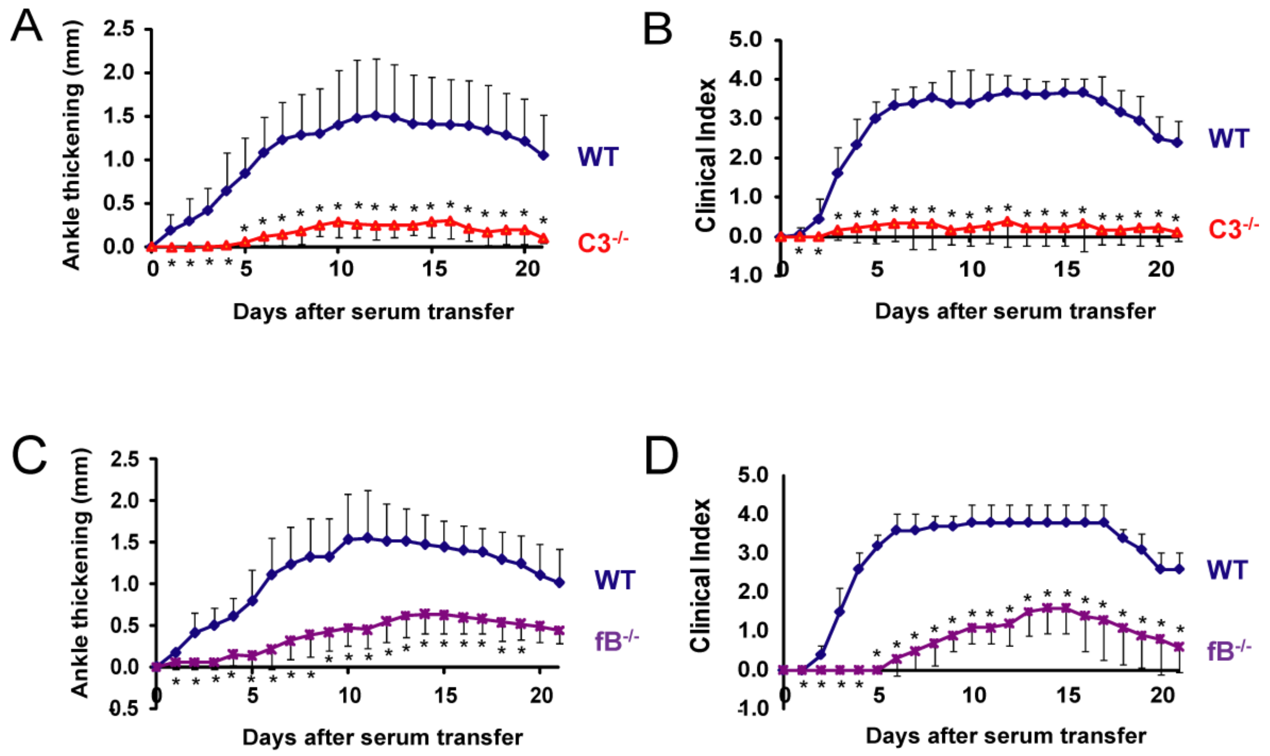
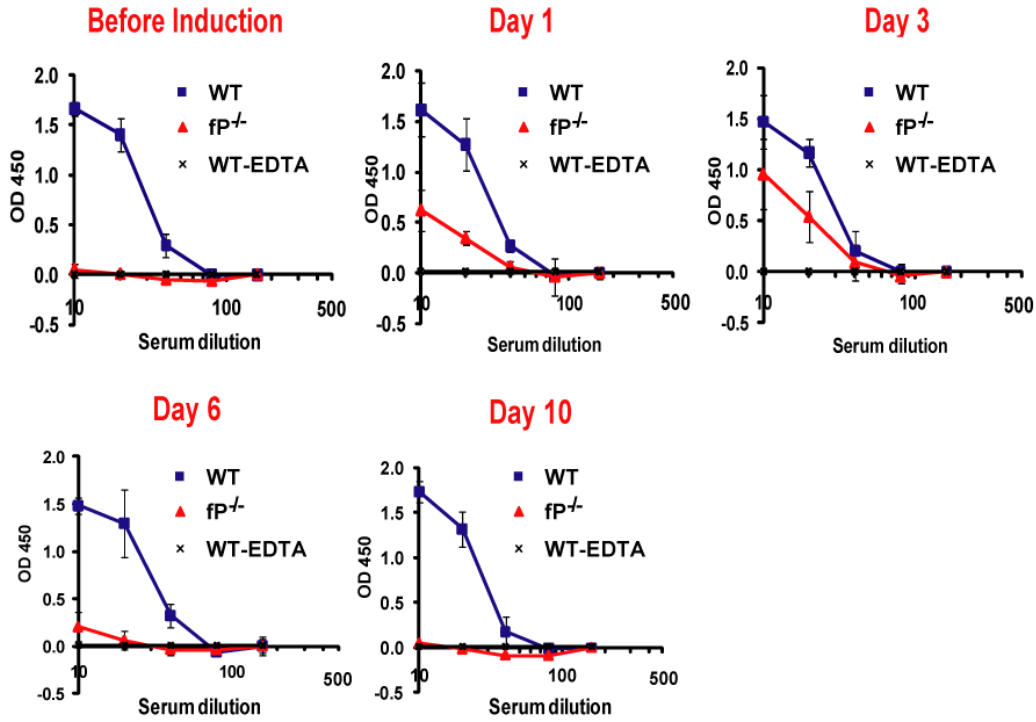


Supplemental Fig 1

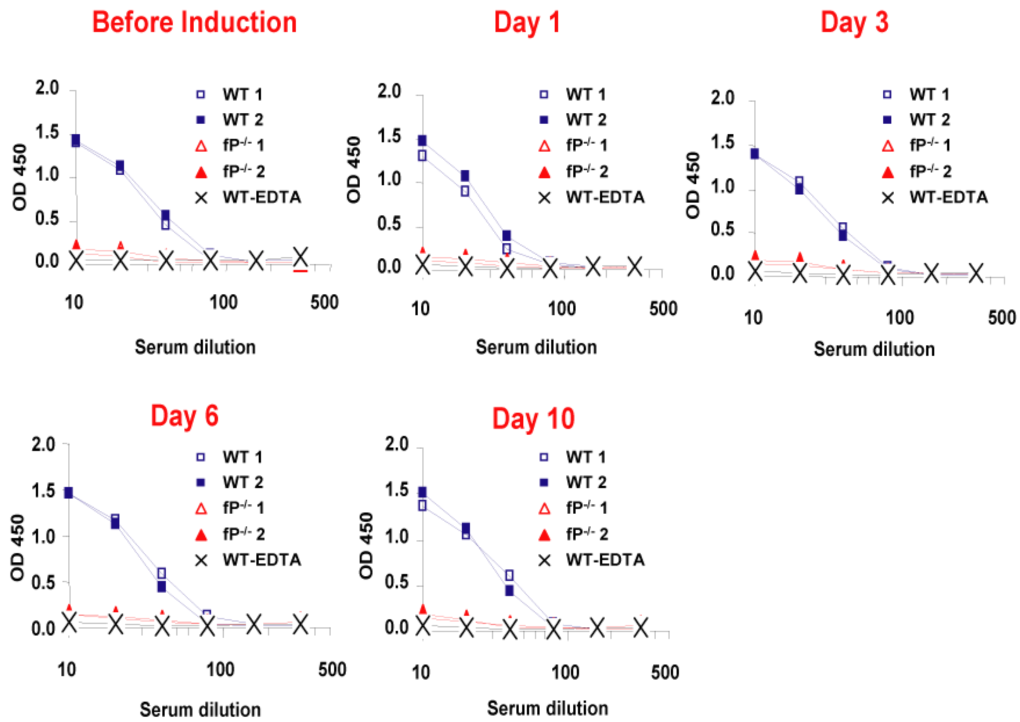
Supplemental Fig 1

Arthritis induction by adoptive transfer of K/BxN mouse serum and confirmation of involvement of the AP complement. (**A**), (**B**) K/BxN mouse serum induced arthritis in WT and $C3^{-/-}$ mice as assessed by ankle thickening (**A**) or clinical index (**B**) (n=9 mice per group). (**C**), (**D**) K/BxN mouse serum induced arthritis in WT and $fB^{-/-}$ mice as assessed by ankle thickening (**C**) or clinical index (**D**) (n=5 mice per group). * $P<0.05$, non-parametric Wilcoxon/Kruskal-Wallis test.

A



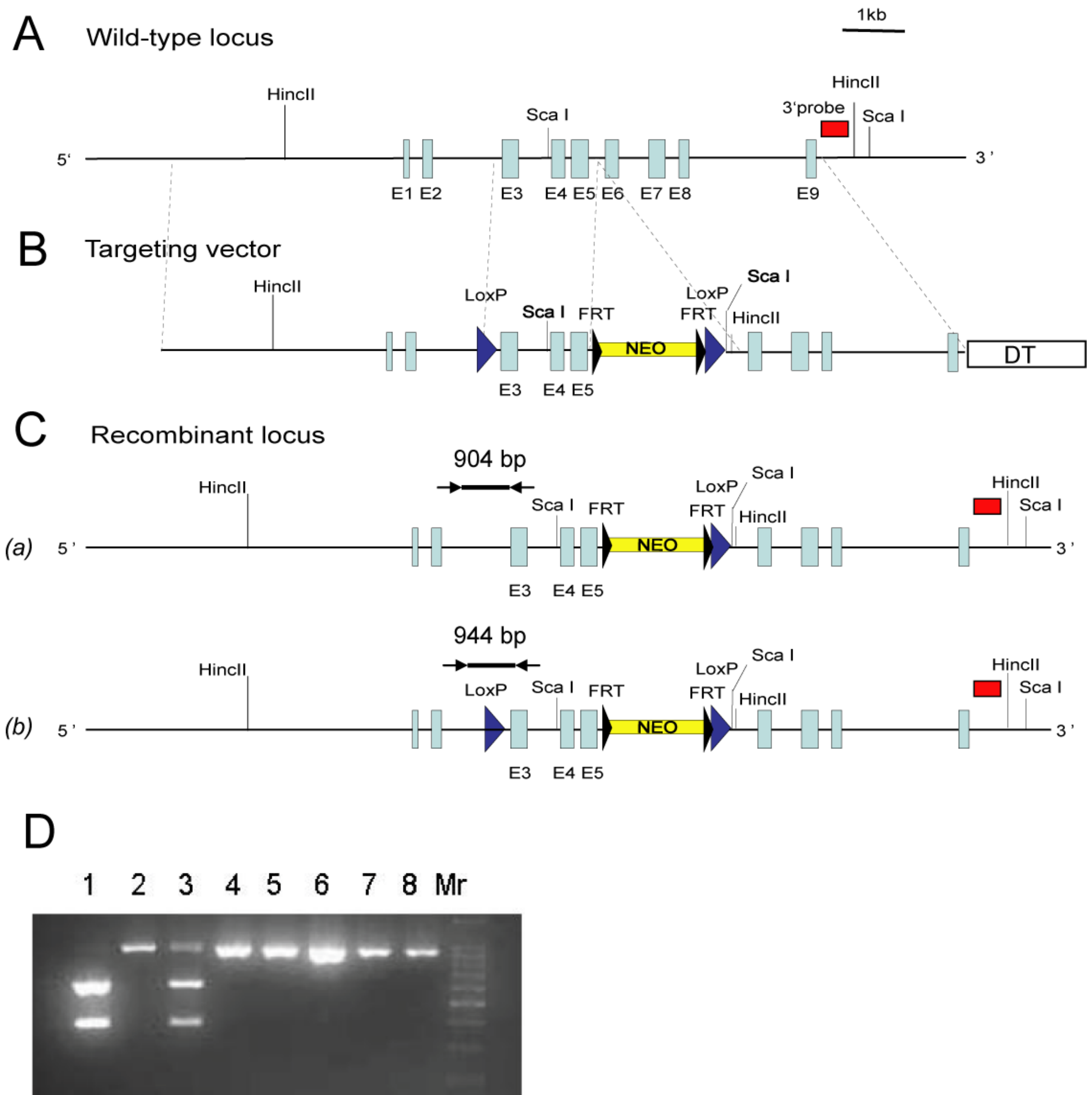
B



Supplemental Fig 2

Supplemental Fig 2

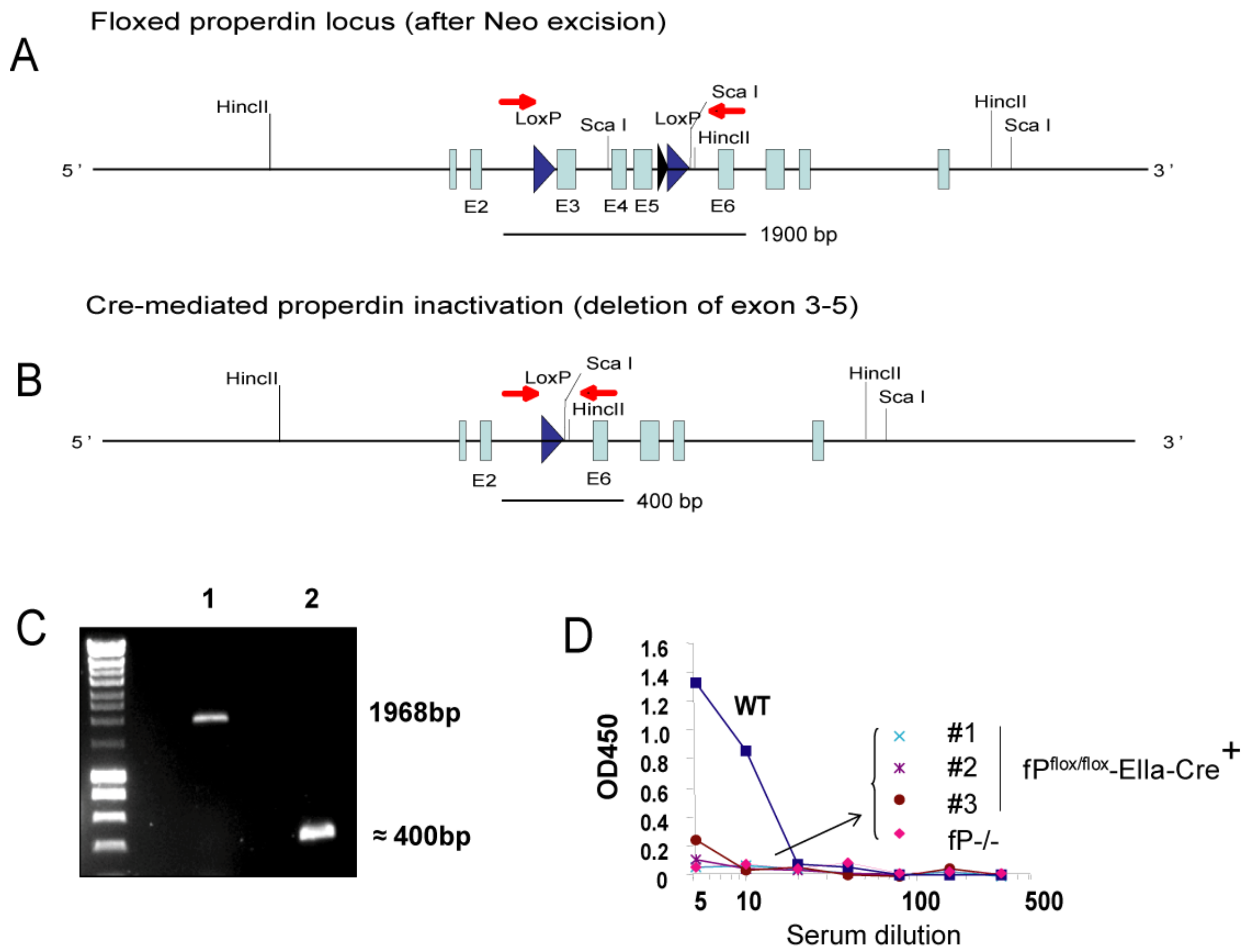
Adoptive transfer of K/BxN mouser serum but not IgGs transiently and partially restored AP complement activity to $fP^{-/-}$ mice. (**A**) LPS-induced AP complement activity in the sera of WT and $fP^{-/-}$ mice receiving K/BxN mouse serum (n=3 mice per group). (**B**) LPS-induced AP complement activity in the sera of WT and $fP^{-/-}$ mice receiving K/BxN mouse IgG (n=3 for WT, n=6 for $fP^{-/-}$, data from two representative mice in each group are shown). EDTA-treated WT mouse serum was used as a control. $fP^{-/-}$ mice received K/BxN mouse serum or IgG on day 0 and 2.



Supplemental Fig 3

Supplemental Fig 3

Gene targeting strategy to create a floxed mouse properdin gene. (**A**) Genomic structure of the WT mouse properdin gene which is composed of 9 exons (E1 to E9). (**B**) Structure of the targeting vector. DT: diphtheria toxin; NEO: neomycin. (**C**) Two potential homologous recombination outcomes that were found in positive ES cell clones identified by Southern blot using a 3' probe (red rectangle). In type (*a*), the 5' LoxP site was not incorporated into the recombinant locus and such ES cell clones were not desirable. In type (*b*), the 5' LoxP was present and exons 3-5 of the properdin gene were therefore flanked by two LoxP sites as intended. (**D**). Screening of type (*b*) recombination by restriction enzyme (EcoR V) digestion of PCR products using primers spanning the expected 5' LoxP site. Since an EcoR V restriction enzyme site was added to the 5' LoxP sequence, the presence of the 5' LoxP site in a given ES cell clone would be indicated by EcoR V digestion of the PCR product (lane 3). EcoR V-resistant PCR products (lanes 2, 4, 5, 6, 7, 8), on the other hand, indicate ES cell clones lacking the 5' LoxP site. Mr: molecular weight markers.



Supplemental Fig 4

Supplemental Figure 4

Generation and validation of a properdin-floxed mouse by gene targeting. (**A**) Schematic diagram showing the floxed properdin gene structure after NEO excision via breeding with a FLPe transgenic mouse. Exons (E) 3-5 were flanked by two LoxP sites (big arrow heads). A residual FRT site (small arrow head) 3' to exon 5 remained after NEO deletion. Red arrows indicate the direction and approximate location of primers used for genotyping. (**B**) Schematic diagram showing the mutated properdin gene after Cre-mediated deletion of exons 3-5. (**C**) PCR analysis of tail DNA from $fP^{flox/flox}$ -Ella-Cre^{-} (lane 1) and $fP^{flox/flox}$-Ella-Cre^{+} mice (lane 2) showing the presence (1968 bp product) and absence (≈400 bp product) of exons 3-5, respectively. The location and direction of primers used are indicated by red arrows in panel **A** and **B**. (**D**) ELISA plate assay showing the lack of LPS-induced AP complement activity in the sera of 3 $fP^{flox/flox}$-Ella-Cre^{+} mice. WT and $fP^{-/-}$ mouse serum was used as a positive and negative control, respectively.