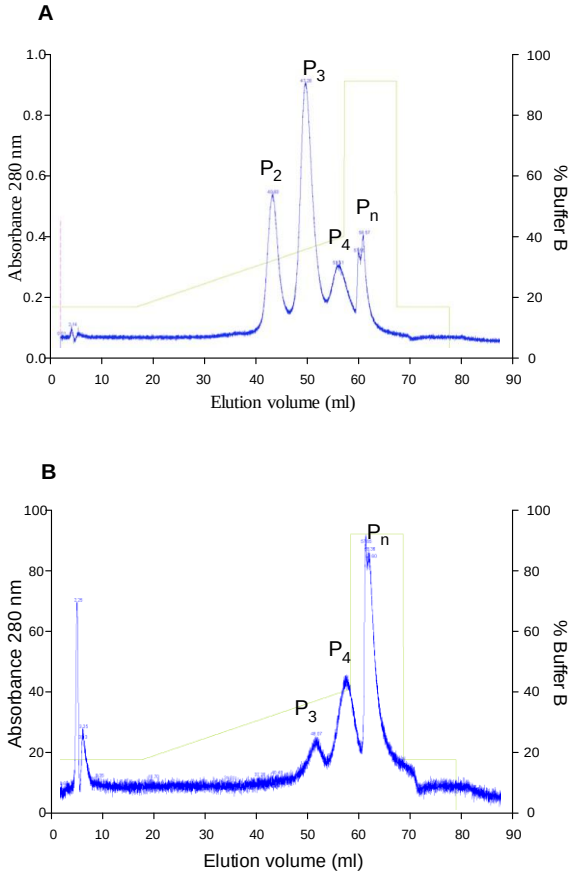


Figure S1. Analysis of purified human properdin and recombinant human properdin by cation exchange chromatography: Properdin samples were analyzed as described by Pangburn (1989). Briefly, the sample (~25-50µg) was diluted with Mono S buffer A (50 mM sodium phosphate, pH 6.0) (ratio of sample to buffer by volume = 1:4) and loaded onto a 1 ml Mono S column (Mono S 5/50 GL – GE Healthcare). The column was washed with 20% Mono S buffer B (50 mM sodium phosphate, 0.5 M NaCl, pH 6.0), and eluted using a 20 ml salt gradient (20-100%) of Mono S buffer B at a flow rate of 0.5 ml/min. The purified human properdin resolved into P_2, P_3, P_4 and P_n as expected (A). The recombinant human properdin showed two peaks within the range of P_3 and P_4 (of the purified human properdin) and an additional peak for the P_n forms. Native human properdin was purified as previously described[1].

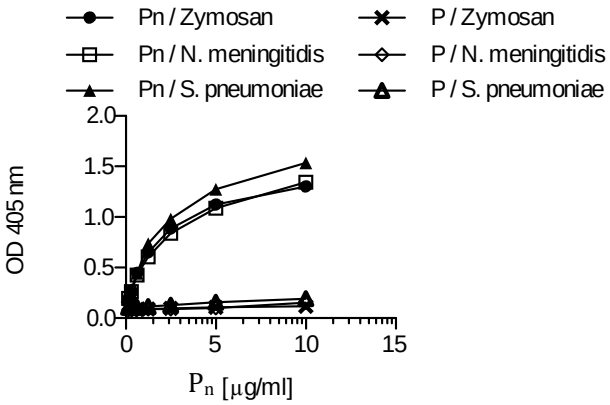
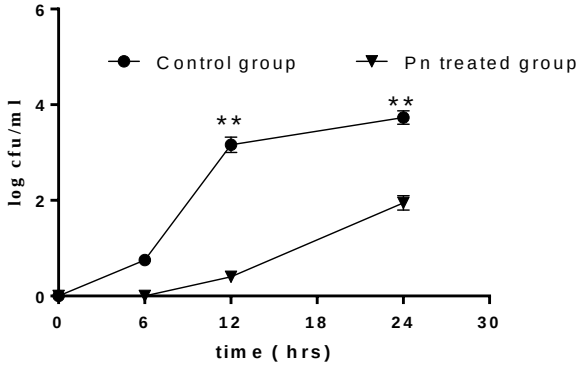


Figure S2. **Binding of recombinant activated (P_n) and native properdin purified from serum (P) to *S. pneumoniae* and *N. meningitidis*.** Microtiter plates were coated with *S. pneumoniae, N. meningitidis* or zymosan as a positive control. Different concentrations of human P_n and native properdin (see figure S1) were incubated with the bacteria. Binding of properdin was detected using polyclonal rabbit anti-properdin antibodies. Activated properdin showed strong binding to both *S. pneumoniae* and *N. meningitidis* while non-activated properdin did not show any binding affinity. Results are means of duplicates and are representative of two independent experiments.



S3. Pn treatment reduces the blood bacteremia when given 6 hrs after intranasal infection with *S. pneumoniae*. Mice were infected intra-nasally with $2.5x10^6$ cfu *S. pneumoniae* D39. Animals in the treated group were injected *i.p.* with 100µg of recombinant murine properdin (P_n) at 6 hrs before infection. P_n treated mice show significantly reduced bacterial load in blood 12 and 24 hrs post infection (means ±SEM; n=7). **p<0.01.

References

1. Ferreira VP, Cortes C & Pangburn MK (2010) Native polymeric forms of properdin selectively bind to targets and promote activation of the alternative pathway of complement. *Immunobiology* 215(11): 932-940.

2. Pangburn MK (1989) Analysis of the natural polymeric forms of human properdin and their functions in complement activation. *J Immunol* 142(1): 202-207.