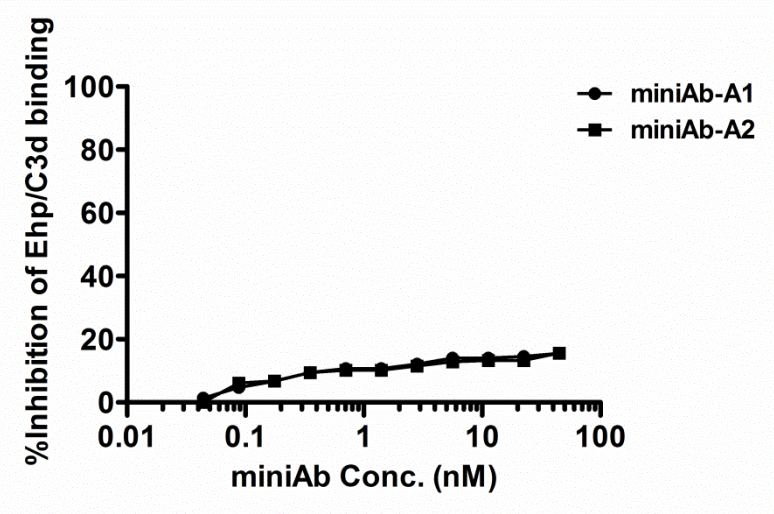
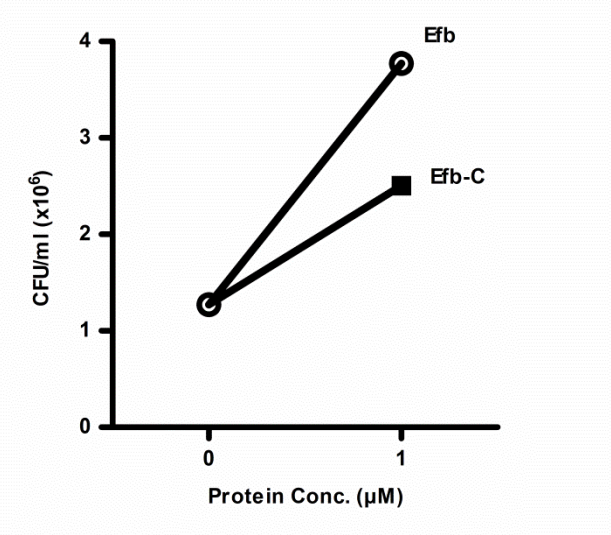
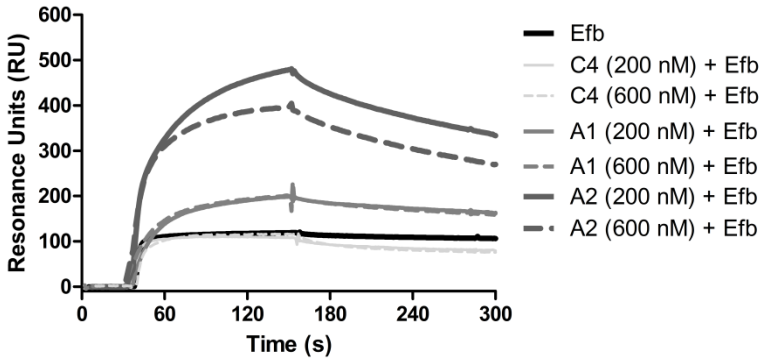


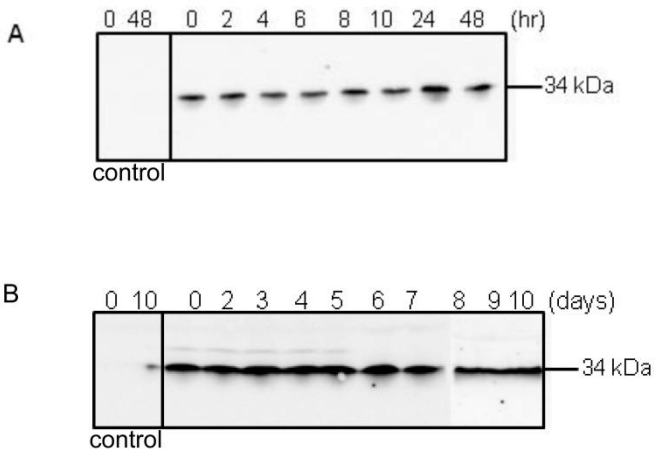
Supplemental Figure 1. MiniAb-A2 does not block the interaction of Ehp with C3d.The effect of miniAb-A2 on blocking the formation of the Ehp-C3d complex was determined by a competitive ELISA. Ehp (5µg/ml) was pre-incubated with miniAbs at various concentrations followed by the addition of 1µg/ml of recombinant C3d. O.D. (405nm) corresponding to bound C3d was measured in an ELISA plate reader. The percentage of inhibition of binding is plotted against the miniAb concentration, and compared to the sample that does not contain any miniAb. The graph is representative of three independent experiments



Supplemental Figure 2. Comparison of full-length Efb and Efb-C in the whole blood model of *S. aureus*-induced bacteremia. Both recombinant Efb and Efb-C promote the growth of *S. aureus* (CFU/ml) at 1 µM. No significant difference is observed in the *S. aureus* growth-promoting effect of the two proteins. The data are representative of three independent experiments.



Supplemental Figure 3. MiniAb-A1 potently competes Efb binding to Fg-coated surfaces. A mixture of miniAb-A2 with Efb (100nM) produced a 4 to 5-fold increase in signal relative to Efb alone when injected over a surface of immobilized fibrinogen, consistent with a larger analyte (i.e. Efb-A2 complex) interacting with Fg. However, miniAb A1 produced a modest increase in signal when co-injected with Efb, indicating that the Efb-A1 complex is a less competent binder of fibrinogen. Mixtures of Efb with control miniAb-C4 produced responses with a similar magnitude and kinetic profile as that observed for Efb alone.



Supplemental Figure 4. Pronounced stability of human miniAbs in mouse serum. MiniAb-A1 (100 µg/ml) was incubated in mouse serum for (A) 0-48 h and (B) for 0-10 days at 37^{o}C. Samples were analyzed by 10% SDS-PAGE and the miniAb was detected by western blot. No miniAb cleavage fragments were detected at any time point.