Supplementary Tables.Supplementary Table 1. Primers used

Primer Name	Sequence
Well-ID adaptor	CACGACCGGTGCTCGATTTAG(well-ID)GGG
FW long primer1	GAGAGACTGACAGCGTATCGCCTCCCTCGCGCCATCAG (plate-ID)CACGACCGGTGCTCGATTTAG

FW short primer1	GAGAGACTGACAGCGTATCGCCTC
kappa GSP1	CGATTGGAGGGCGTTATCCAC
lambda GSP1	TYTGTGGGACTTCCACTGCTC
gamma GSP1	TCTTGTCCACCTTGGTGTTGCTG
FW primer2	CGTATCGCCTCCCTCGCG
kappa GSP long primer2	CTATGCGCCTTGCCAGCCCGCTCAG(plate-ID)TCAGATGGC GGGAAGATGAAGAC
lambda GSP long primer2	CTATGCGCCTTGCCAGCCCGCTCAG(plate-ID)GAGGAGGGY GGGAACAGAGTGAC
gamma GSP long primer2	CTATGCGCCTTGCCAGCCCGCTCAG(plate-ID)GGAAGTAGT CCTTGACCAGGCAG
RV primer2	CTATGCGCCTTGCCAGCCC

FW, forward; RV, reverse; GSP, gene-specific primer.

SUPPLEMENTARY FIGURE LEGENDS

Supplemental Figure S1. Plasmablast paired-chain dendrograms of patient 1 (A) and patient 2 (B) were used for selecting antibodies with shared gene rearrangements for recombinant expression. Dendrograms were generated by barcode-enabled matching and bioinformatic analysis of the cognate heavy- and light-chain pairs of antibody sequences. S1, S2, S3, S4, S5, S6, S7, S8, S9, and S10 denote the ten recombinant antibodies that were selected for recombinant expression. Circles denote antibodies that bind *S. aureus*, and squares denote antibodies that do not. Recombinant antibodies from shared gene families are denoted in blue, and antibodies not sharing gene rearrangements are denoted in green.

Supplemental Figure S2. Sequence analysis of the heavy- and light-chain variable regions from patient 1 reveals a putative clonal family. (A) V_H-region sequence analysis of IGHV5-a family from CDR1 to CDR3 reveals five sequences (identified by unique plate- and well-ID combination) with shared somatic mutations relative to germline V-D-J sequence “HV5-a|HD6-13*01|HJ6*02.” (B) VL-region sequence analysis of the recovered light-chain sequences of reveal shared mutations relative to the germline V-J sequence “IGLV7-46*02|IGLJ3*02.”

Supplemental Figure S3. Dot plot representation of antibody binding to the cell surface of fixed *S. aureus* Wood46 at three different growth phases in LB broth, to *S. aureus* Wood46 grown to stationary phase in the presence of NIH/3T3 fibroblasts, and to DH10B *E. coli*, as assessed by flow cytometry. Data are aggregated from the experiments performed in Fig. 3.

Supplemental Figure S4. (A) Time course showing the percent of bacterial internalization by undifferentiated THP1 cells after incubation with opsonizing reagent (*S. aureus*-specific polyclonal antibody) and F26 (influenza HA-specific antibody). (B) Histogram representations of THP1 internalization of CFSE-labeled, fixed *S. aureus*. *S. aureus* Wood46 co-cultured with NIH/3T3 fibroblasts were isolated, fixed, labeled with CFSE, and incubated with THP1 monocytes for 40 minutes before flow cytometric analysis. S.a., *S. aureus*; op, anti-*S. aureus* polyclonal rabbit IgG antibodies.