The plasminogen binding protein PbsP is required for brain invasion by hypervirulent CC17 Group B streptococci

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Figure-S1 (Teti)

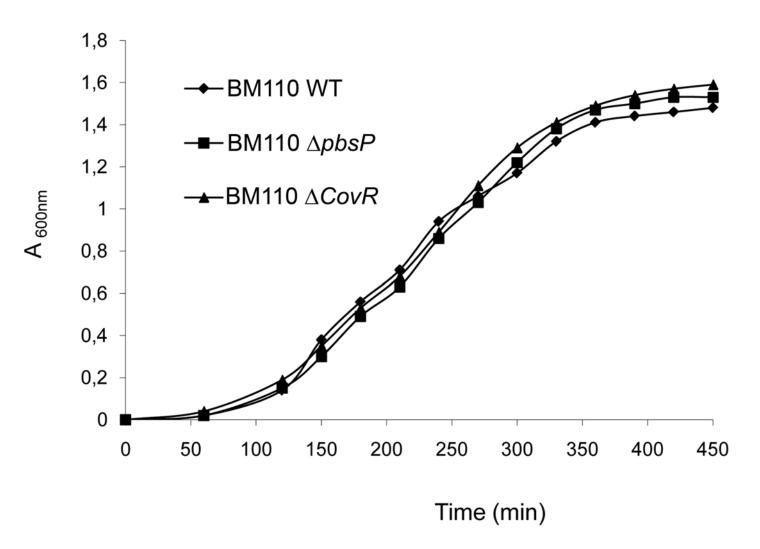
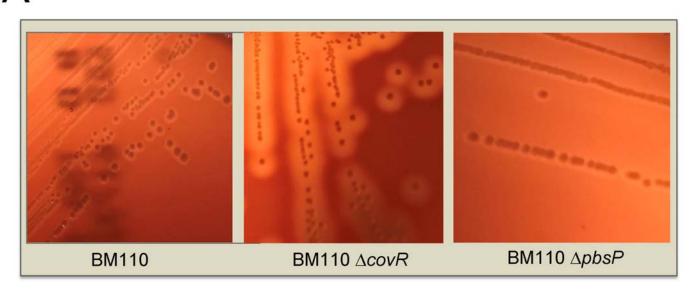


Fig. S2 (Teti)

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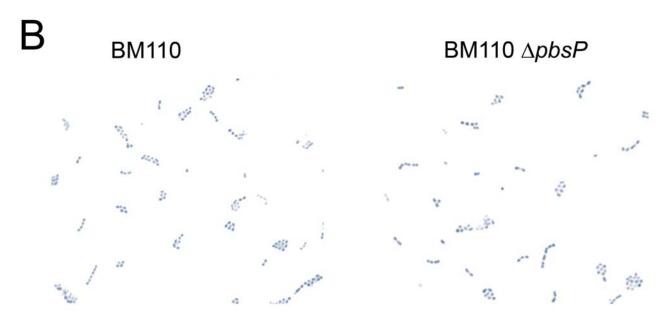


Fig. S3 (Teti)

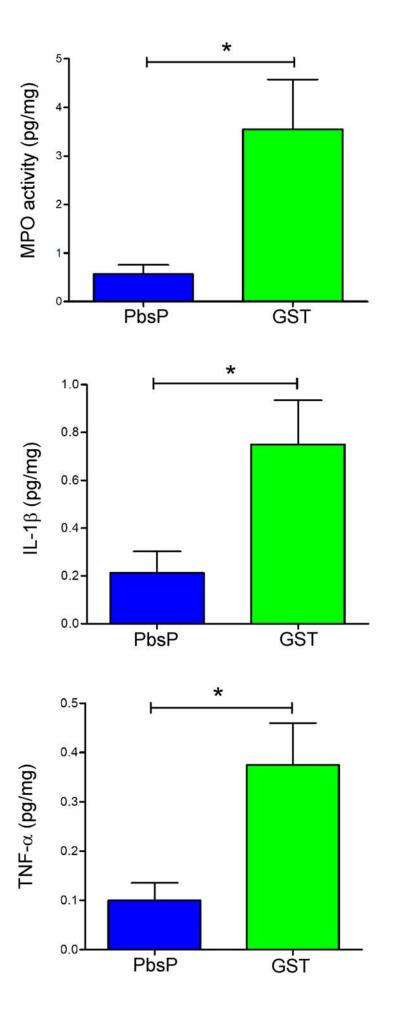


Figure-S4 (Teti)



Figure S1. Growth in Todd-Hewitt broth of GBS BM110 mutants.

Growth curves of BM110 (WT, diamonds), pbsP deletion mutant (BM110 $\Delta pbsP$, squares) and CovR deletion mutant (BM110 $\Delta CovR$, triangles) GBS strains in Todd-Hewitt broth.

Fig. S2. Colony morphology and microscopic appearance of GBS BM110 mutants.

(A) Colony morphology of wild-type BM110 strain (BM110) and its CovR or pbsP deletion mutants (BM110 $\Delta CovR$ or BM110 $\Delta pbsP$, respectively) on blood agar. (B) Cell morphology after Gram stain of wild-type BM110 strain (BM110) and its pbsP deletion mutant (BM110 $\Delta pbsP$).

Fig. S3. PbsP-based immunization prevents GBS-induced encephalitis. MPO, IL-1 β and TNF- α protein levels were measured in organ homogenates from mice immunized with recombinant PbsP fused to glutathione-S-transferase (GST-PbsP) or with GST as a control. Data are expressed as means \pm standard deviations of three duplicate observations, each conducted with a different animal, in the course of a single experiment. *, p<0.05 by one way ANOVA.

Figure S4. Image from the same blot shown in Fig 1, obtained with a higher exposure

Table S1

RT-PCR primer and probe sequences

pbsp_up TGATGGTGGCTACCTCTGTTAGAA

pbsp_low AGTCGTTTTGGTCATCGCTACA

gyr_up AACTGGGAAGGGATCAATTGTCT

gyr_low CTTTCCCATTTGAAGTTGTTTCAA

FAM-pbsp_probe ATGGCAACGCCGACAC

VIC-gyr_probe ACGTTCACGGACTGAA