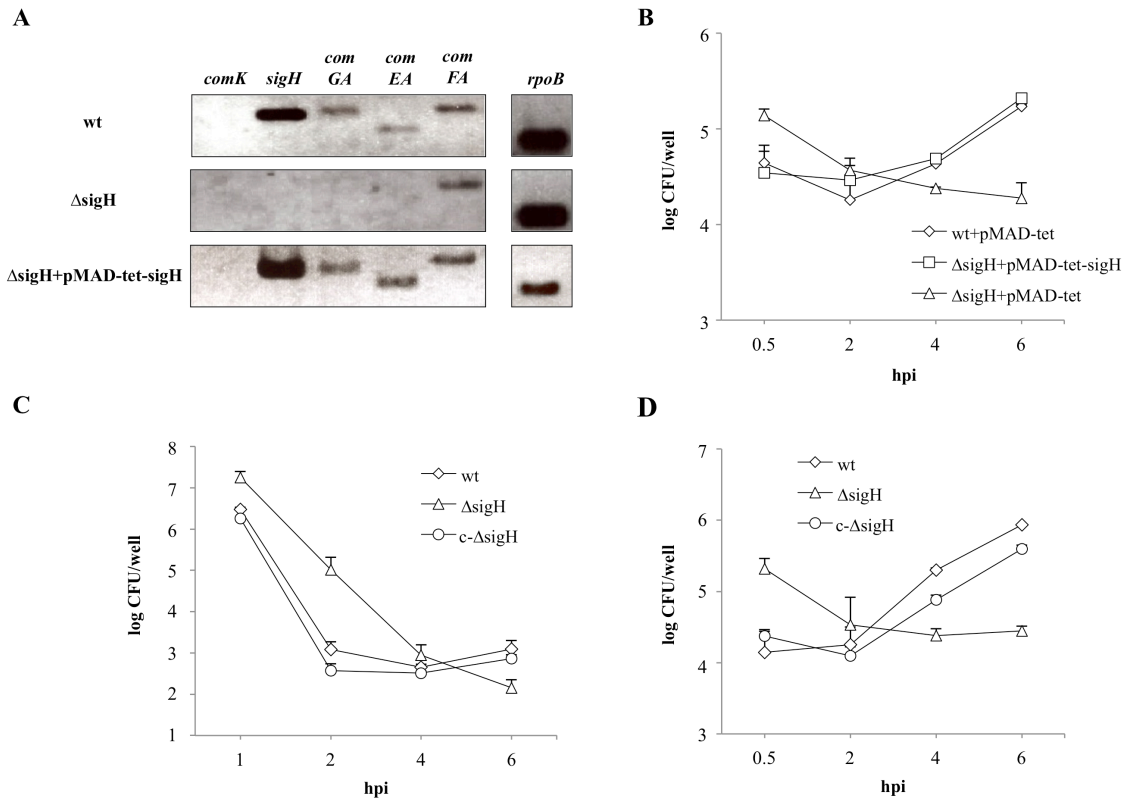

Fig. S1 Complementation of *sigH*. **A.** Semi-quantitative RT-PCR of *comK*, *sigH*, *comGA*, *comEA*, and *comFA* genes. wt: phage-cured EGDe strain; ΔsigH: *sigH* deletion strain; ΔsigH+pMAD-tet-sigH: *sigH* deletion strain complemented with pMAD-tet-sigH. Sampling point is shown in Fig. 2. A representative result is shown from 3 independent experiments. The *sigH* complementation restored the expression of *comGA, comEA*. **B.** Intracellular growth in RAW 264.7 macrophages. wt+pMAD-tet: phage-cured EGDe strain carrying pMAD-tet; ΔsigH+pMAD-tet: *sigH* deletion strain carrying pMAD-tet; ΔsigH+pMAD-tet-sigH: *sigH* deletion strain complemented with pMAD-tet-sigH. Means of 3 independent experiments are shown. The *sigH* complementation restored the intracellular growth in macrophages. **C.** Intracellular growth in HeLa cells. We noted that cells carrying pMAD-tet or pMAD-tet-sigH exhibited heterogeneous colony size after the passage through HeLa cells due to unknown reasons. Therefore, we used a chromosomally complemented strain, c-ΔsigH. wt: phage-cured EGDe strain; ΔsigH: *sigH* deletion strain; c-ΔsigH: *sigH* chromosomal complementation of ΔsigH. c-ΔsigH recovered the growth deficiency in HeLa cells, confirming that the disruption of the *sigH* locus leads to the growth deficiency. Means of at least 2 independent experiments are shown. **D.** Intracellular growth on RAW264.7 cells. Strains are the same as in C. In line with the plasmid-complemented strain (ΔsigH+pMAD-tet-sigH), the chromosomally cured c-ΔsigH also recovered the intracellular growth in macrophages. Means of at least 2 independent experiments are shown.