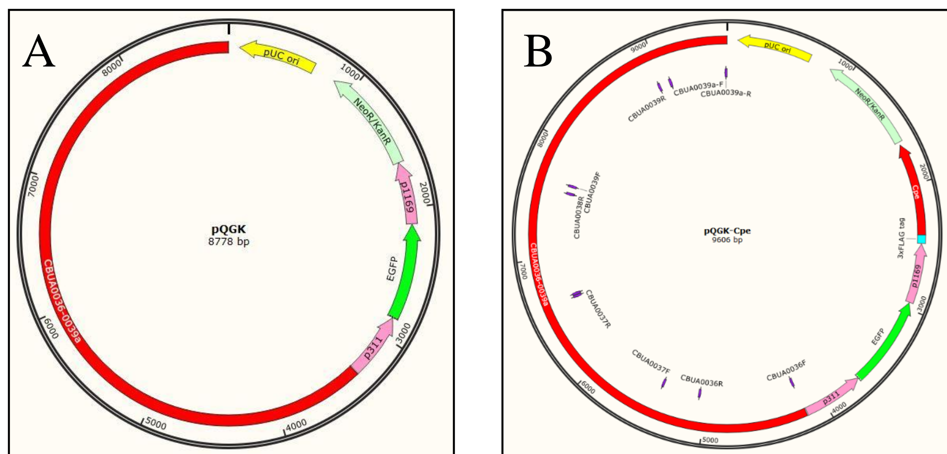
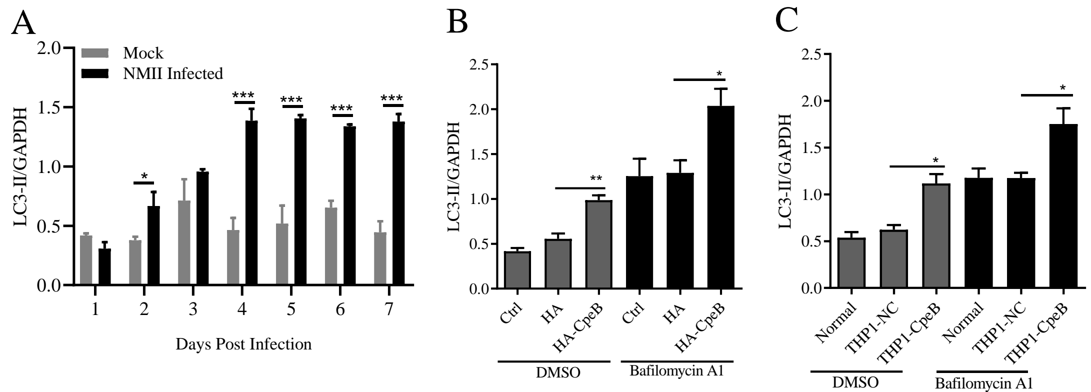
Fig S1



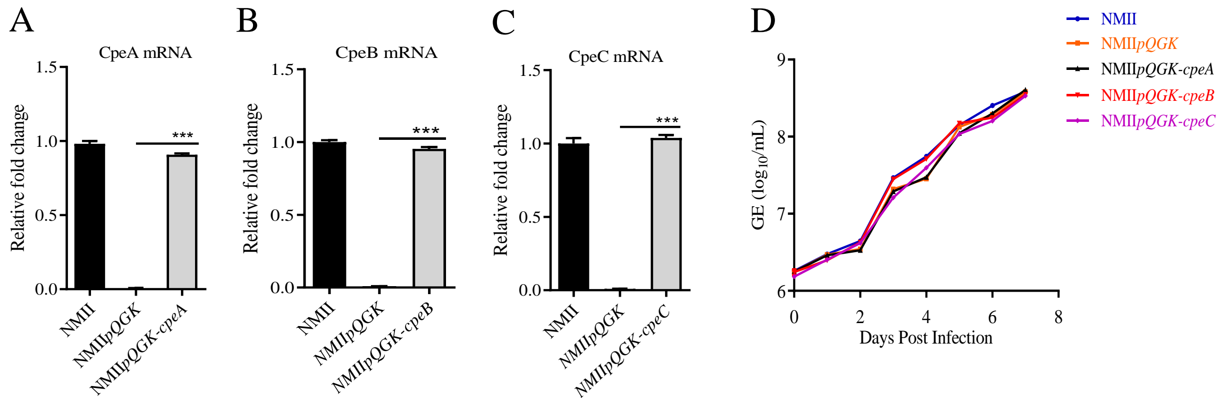
Supplementary Fig 1. Patterns of QpH1-based shuttle plasmids used in the construction of *C. burnetii* mutants. (**A**) pQGK plasmid. (**B**) pQGK-cpe plasmid.

Fig S2



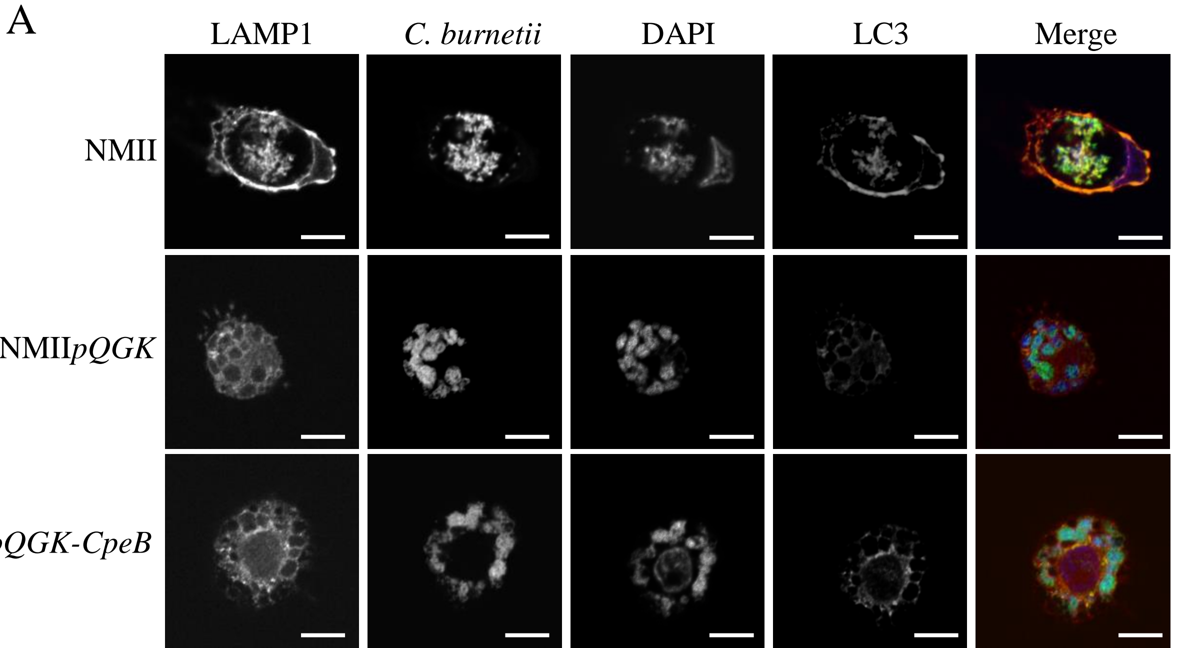
Supplementary Fig 2. The band density of LC3-II in Fig 1 was quantitated by densitometry. A, B and C showed the quantification analysis of Fig 1A, F and G, respectively. GAPDH expression was used as an internal control. The relative level of LC3-II was calculated as follows: the band density of LC3-II in each sample/ that of GAPDH in the same sample. Bars represent the mean ± SD of three independent experiments. ***, $p < 0.001$, **, $p < 0.01$ and *, $p<0.05$.

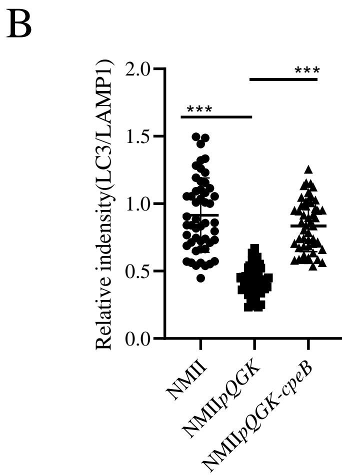
Fig S3

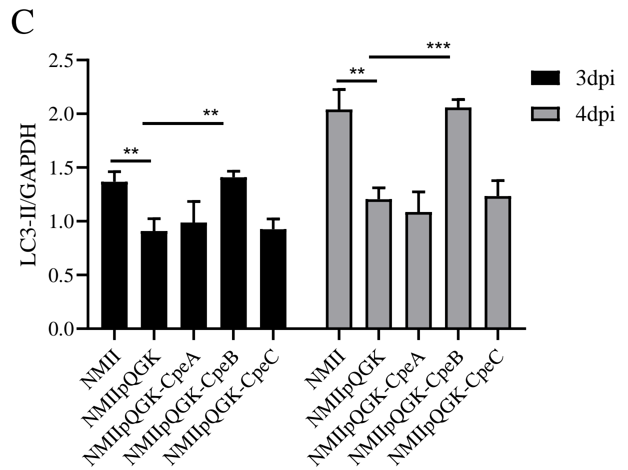


Supplementary Fig 3. (**A-C**) Detection of the efficiency of gene knockout and complementation. mRNA of THP1 cells infected with different *C. burnetii* strains was extracted after 5 days of infection and was quantitated by qRT-PCR. The relative levels of CpeA (A), CpeB (B), CpeC (C) were calculated as follows: the copy number of gene in *C. burnetii* mutants/that in WT *C. burnetii*. (**D**) There was no difference in growth characteristics between WT *C. burnetii* and *C. burnetii* mutants in vitro. WT *C. burnetii* or *C. burnetii* mutants were collected at different time points (1, 2, 3, 4, 5, 6 and 7 d) after culture in ACCM-2. Genomic DNA was extracted and quantitated by qPCR. Experiments were repeated three times independently and the trend was consistent. Data are representative of three independent experiments. Bars represent the mean ± SD of three independent experiments. ***, $p < 0.001$.

Fig S4

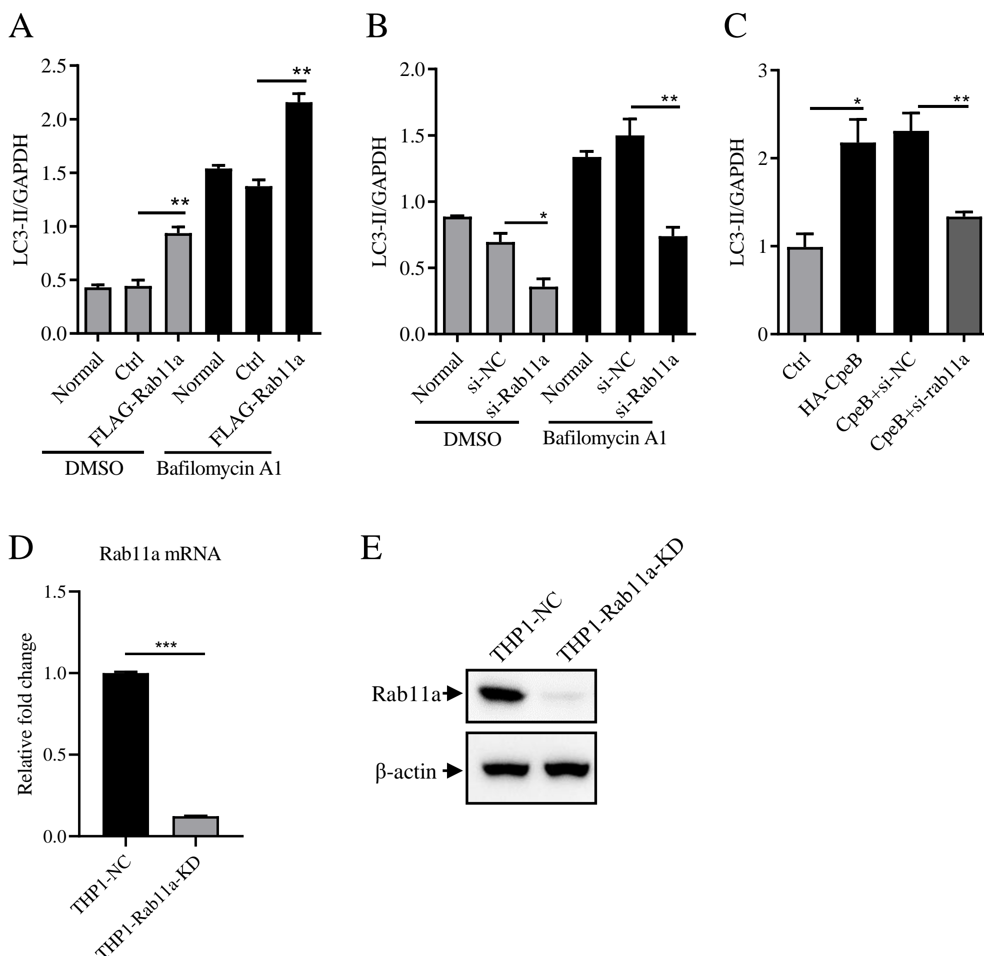






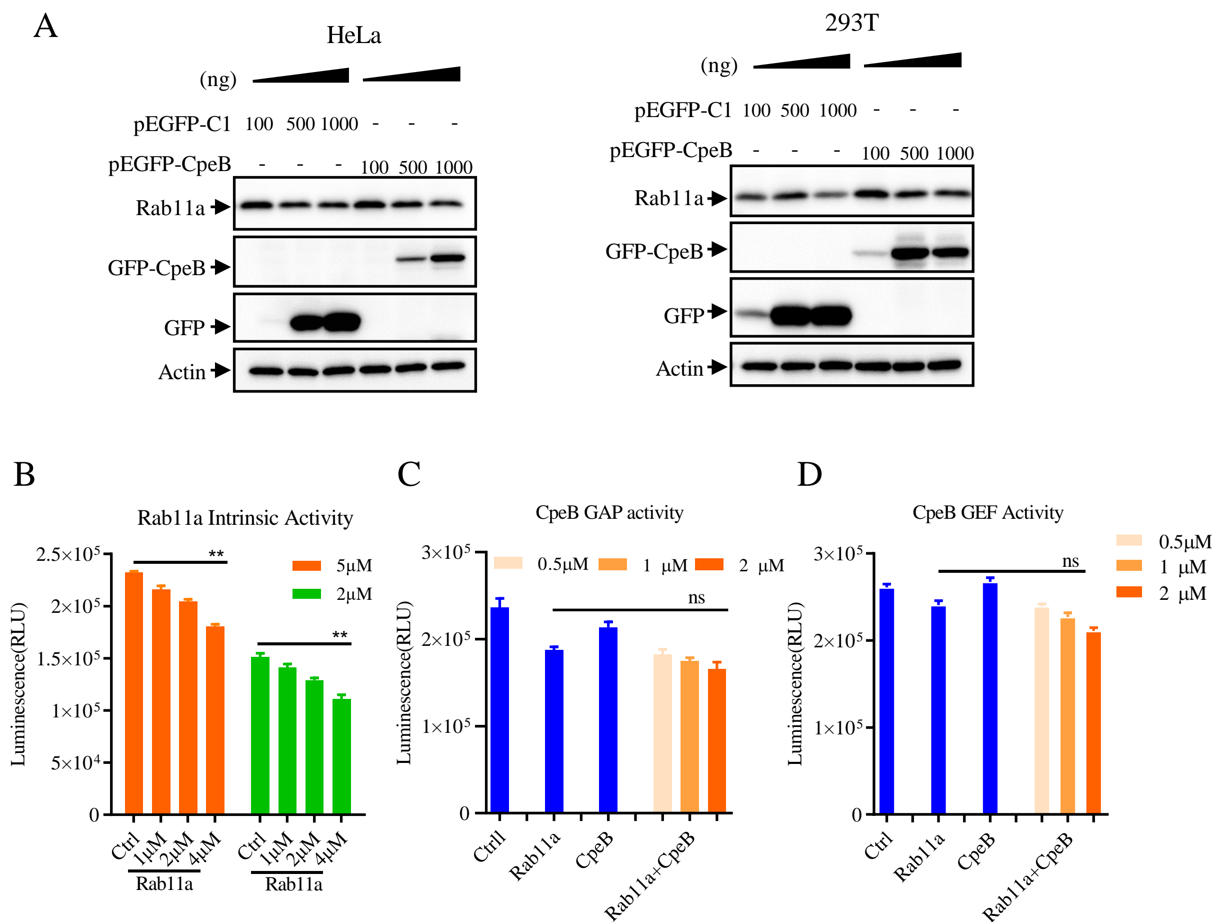
Supplementary Fig 4. (A) Differentiated THP-1 cells were infected with NMII, NMII*pQGK* or NMII*pQGK-CpeB* strains at an MOI of 100 respectively. Three days post infection, cells were fixed and subjected to confocal scanning. LC3 was labeled with anti-LC3 antibody followed by goat anti-rabbit Cy5 antibody. LAMP1 was probed with anti-LAMP1 antibody, followed by goat anti-rabbit Alexa Fluor 594 antibody (red) and NMII was marked by anti-*C. burnetii* serum followed by goat anti-mouse Alexa Fluor 488 antibody (green). Nucleic acid was stained with DAPI (blue). N=7. The samples were observed with a laser confocal scanning microscope (magnification, ×600, bar = 10 μm). (B) The intensity of LC3 presented on vacuole membranes was measured for at least 50 cells using Imaris 9.0.1 software. The relative intensity was calculated as follows: intensity of LC3 in infected cells/intensity of LAMP1 in infected cells. The intensity of LAMP1 was used as a control for the amount of CCVs and autolysosomes generated by *C. burnetii* infection. Bars represent the mean ± SD. ***, $p < 0.001$. (C) The quantification analysis of Fig 2E. The relative level of LC3-II was calculated as follows: the band density of LC3-II in each sample/ that of GAPDH in the same sample. Bars represent the mean ± SD of four independent experiments. ***, $p < 0.001$, **, $p < 0.01$.

Fig S5



Supplementary Fig 5. (A-C) The band density of LC3-II in and Fig 6 was quantitated by densitometry. A, B and C were the quantification analysis of Fig 6B, C and G respectively. GAPDH expression was used as an internal control. The relative level of LC3-II was calculated as follows: the band density of LC3-II in each sample/ that of GAPDH in the same sample. (D) Total mRNA of THP1-NC or THP1-Rab11a-KD cells was extracted and the relative expression of Rab11a mRNA was quantitated by qRT-PCR. GAPDH was used as an internal control. The relative levels of gene expression were calculated as follows: mRNA expression of rab11a gene in THP1-Rab11a-KD cells/ that in THP1-NC cells. (E) THP1-NC or THP1-Rab11a-KD cells were lysed and the samples were examined with Western blotting using an anti-Rab11a antibody. Endogenous β-actin expression was used as an internal control. Bars represent the mean ± SD of three independent experiments. **, $p< 0.01$ and *, $p< 0.05$.

Fig S6



Supplementary Fig 6. CpeB had no effect on the expression or the activation of Rab11a. (**A**) CpeB had no effect on the expression of Rab11a. HeLa (left) or 293T (right) cells were transfected with pEGFP-CpeB or pEGFP-C1 control plasmid at the indicated concentration. Twenty-four hours later, cell lysates were subjected to Western blotting, and the expression of endogenous Rab11a was detected. (**B**) The GTPase activity of Rab11a at different concentrations was detected in the presence of 5 μM or 2 μM GTP. The GTPase/GAP buffer without Rab11a was used as a control. (C-D) CpeB at different concentrations was added to GTPase/GAP buffer (C) or GEF buffer (D) containing 4 μM Rab11a, 1 mM DTT and 5 μM GTP. The corresponding control group was set up at the same time. After incubation and the addition of reaction solution, the full-wavelength luminescence was measured. Each experiment was performed in triplicate and data are representative of three independent experiments. Bars represent the mean ± SD. **, $p < 0.01$, ns for no significance.