

Expanded View Figures

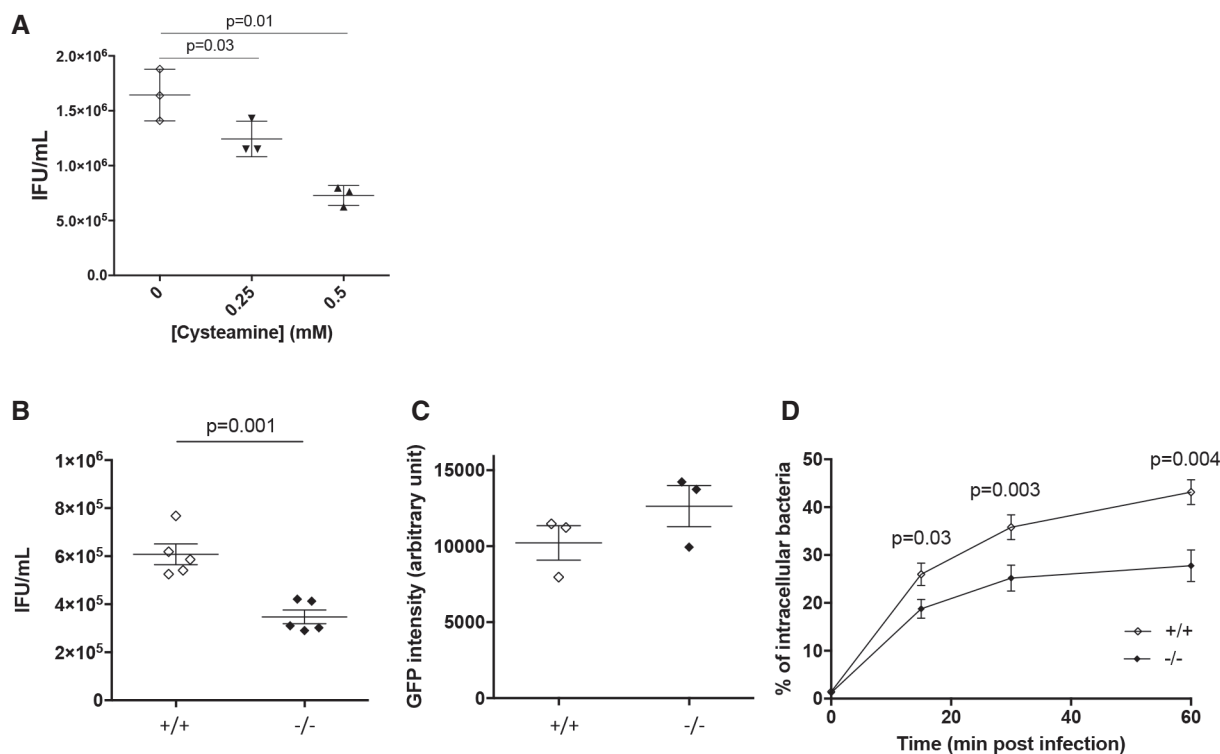


Figure EV1. TG2 is beneficial for bacterial development (related to Fig 2).

- A HeLa cells were pre-treated with the indicated concentrations of cysteamine for 2 h before being infected with L2^{incD}GFP at MOI = 0.15. Thirty hours later, the cells were disrupted and bacterial titers (IFU) were determined by re-infecting fresh HeLa cells as described in the methods. The mean ± SD of three independent experiments is shown. *P*-values of Student's paired *t*-test are indicated.
- B TG2^{+/+} and TG2^{-/-} MEFs were infected with L2^{incD}GFP at MOI = 0.15. Thirty hours later, the cells were disrupted and bacterial titers were determined by re-infecting fresh TG2^{+/+} cells as described in the methods. The mean ± SD of five independent experiments and *P*-value from Student's paired *t*-test are shown.
- C To measure bacterial adhesion, TG2^{+/+} and TG2^{-/-} MEFs were incubated at 4°C for 4 h with L2^{incD}GFP at MOI = 10 before being washed and fixed as described in the methods. The mean ± SD of three independent experiments is shown.
- D TG2^{+/+} and TG2^{-/-} MEFs were infected with L2^{incD}GFP at MOI = 10 and fixed at the indicated time. Extracellular bacteria were differentially labeled as described in the methods. The mean ± SD of three independent experiments, and *P*-values from Student's paired *t*-test, is shown.

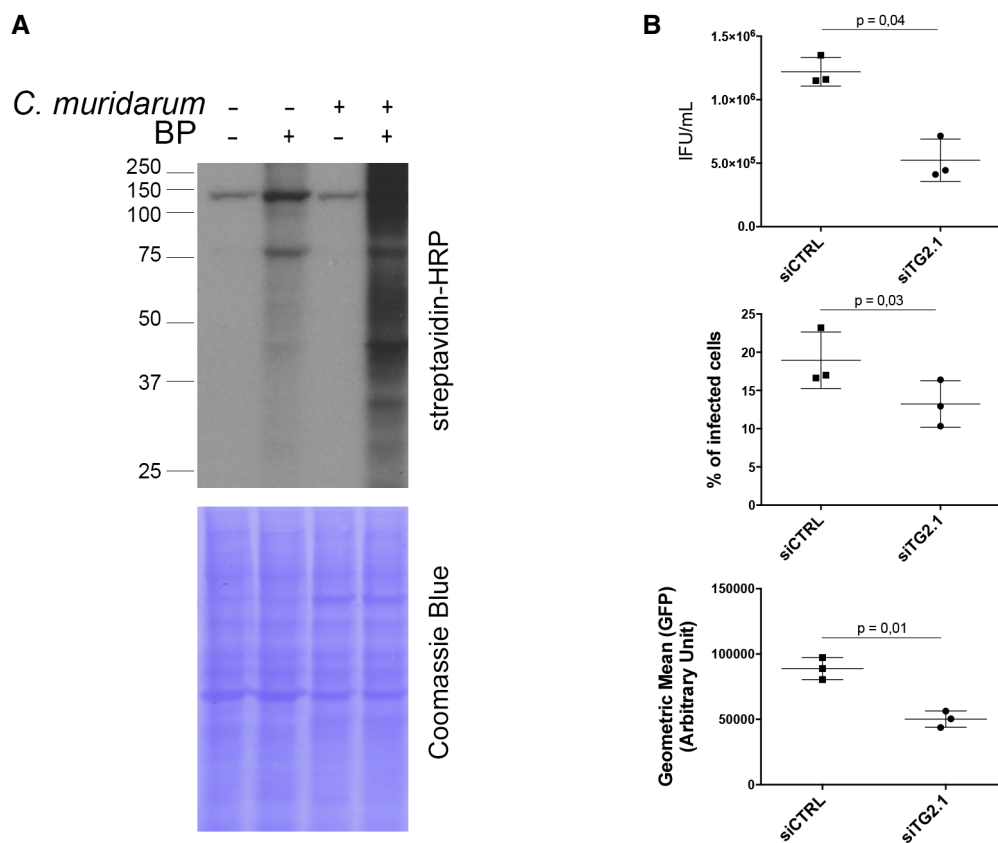


Figure EV2. Infection by *C. muridarum* activates TG2, which favors bacterial growth (related to Fig 2).

- A Whole cell lysates were prepared with HeLa cells infected or not for 48 h with *C. muridarum* (MOI = 1) in the presence or not of BP. Cell lysates were run on SDS-PAGE, proteins were transferred to a membrane, and BP incorporation was revealed with HRP-conjugated streptavidin.
- B HeLa cells were transfected with siRNA against TG2 for 48 h before being infected in duplicates with *C. muridarum* at MOI = 0.15. Thirty hours later, one set of cells were disrupted and bacterial titers (IFU = inclusion-forming unit) were determined by re-infecting fresh HeLa cells as described in the methods. The mean \pm SD of three independent experiments and *P*-values of Student's paired *t*-test are shown (top). Duplicate wells were incubated further for a total of 48 h before the cells were fixed, permeabilized with 0.3% Triton X-100, and stained with rabbit antibodies against *C. muridarum* GroEL followed with A488-coupled anti-rabbit secondary antibodies. Samples were analyzed by flow cytometry, and the percentage of infected cells (middle) and the mean fluorescence of the infected population (bottom) \pm SD are shown for three independent experiments; *P*-values of Student's paired *t*-test are indicated.

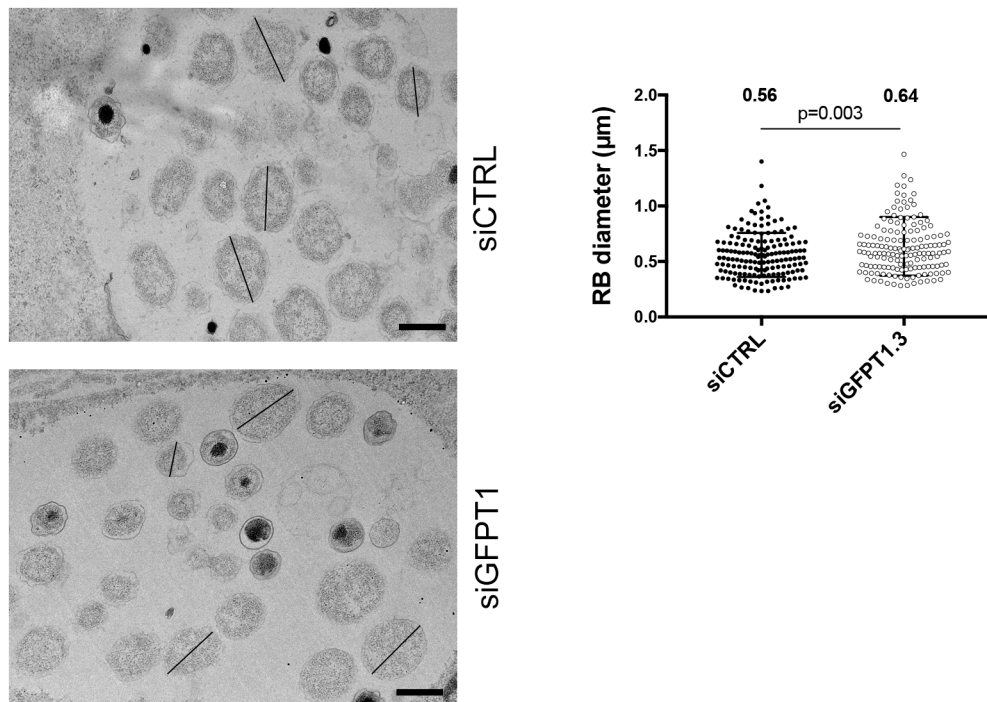


Figure EV3. GFPT silencing affects bacterial division (related to Fig 6).

HeLa cells treated for 48 h with siRNA targeting GFPT1 or not (siCTRL) were infected with *C. trachomatis* (MOI = 1), fixed 30 hpi, and processed for transmission electron microscopy. Lines show example of measured RB diameters. Scale bar = 600 nm. RB diameters were measured using ImageJ on > 300 bacteria in one experiment. Each dot represents one RB; the mean value \pm SD and *P*-value of Student's paired *t*-test are indicated.