

cells (D).

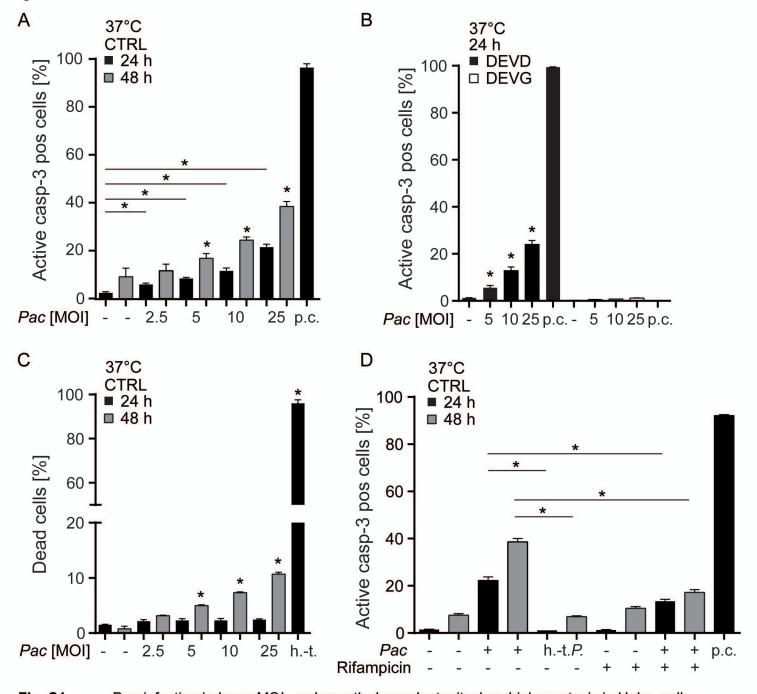


Fig. S1 Pac infection induces MOI- and growth-dependent mitochondrial apoptosis in HeLa cells.

HeLa cells were infected with *Pac* (MOI from 2.5 to 25) and analyzed for cell death and caspase-3 activation. **A**, HeLa CTRL cells were analyzed for activation of caspase-3 by flow cytometry 24 and 48 h after infection. **B**, DEVD (caspase-3 reporter cell line) and DEVG (control cell line) were infected for 24 h to analyze effector caspase activation. **C**, HeLa CTRL were infected with *Pac* for 24 and 48 h and stained with a Live/Dead stain to determine percentage of dead cells in the population. **D**, we heat-treated *Pac* (h.-t.*P*, 1 min 95°C) prior infection or infected CTRL cells in the presence or rifampicin (10 ng/μl) as indicated for 24 or 48 h and analyzed the cultures for aspase-3 activation.

Positive control (p.c.),, treatment with McI-1-inhibitor S63845 (500 nM) and ABT-737 (1 μM) for 1 h. h.-t., heat-treated (1 min at 95°C). Data are shown as means/SEM of at least three independent experiments. \*, p<0.05 (paired two tailed t-test) between uninfected (24 h or 48 h) and infected cells (24 h or 48 h) (A-C). \*, p<0.05 (paired two tailed t-test) between *Pac* (untreated) and *Pac* (heat-treated or rifampicin treated) infected