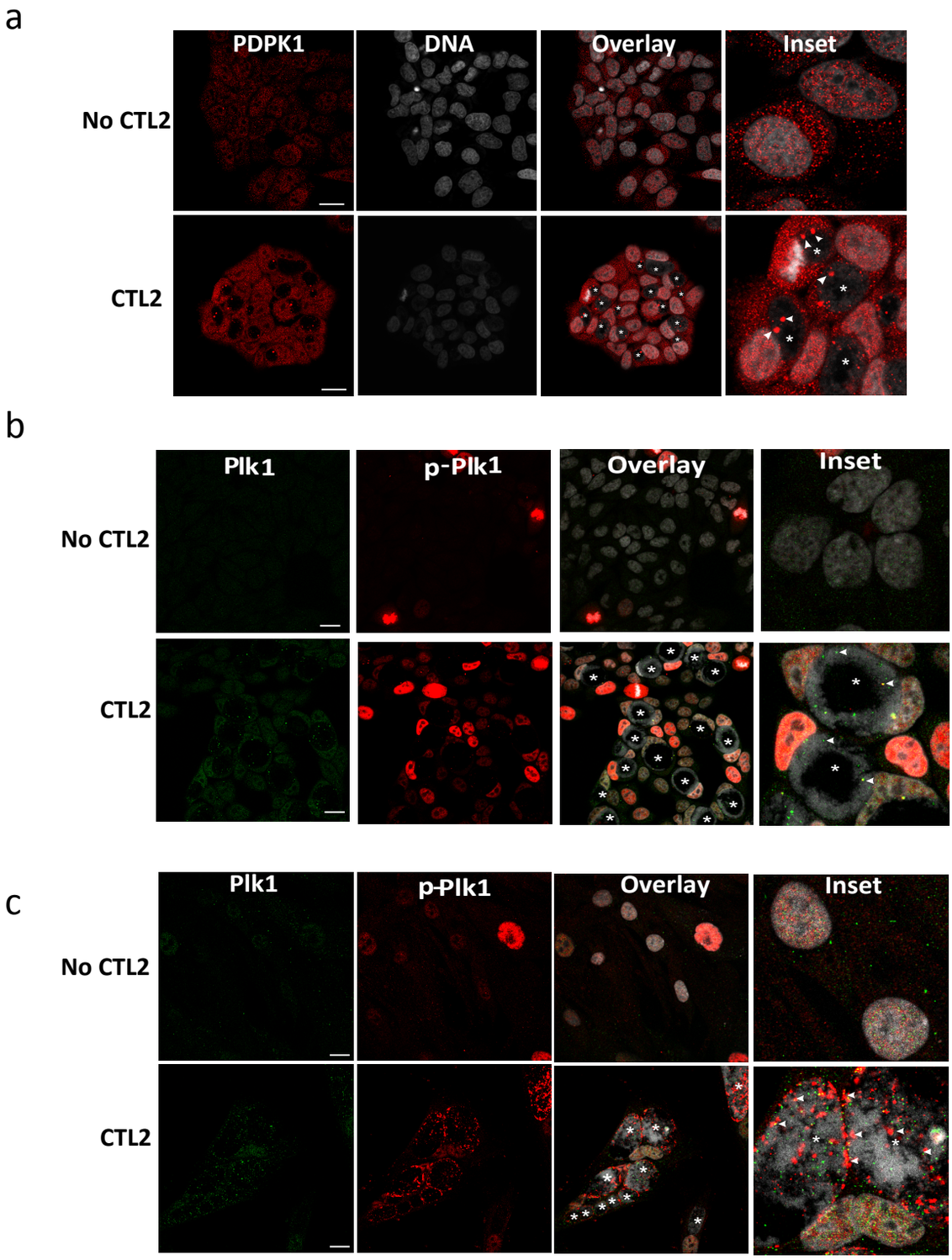
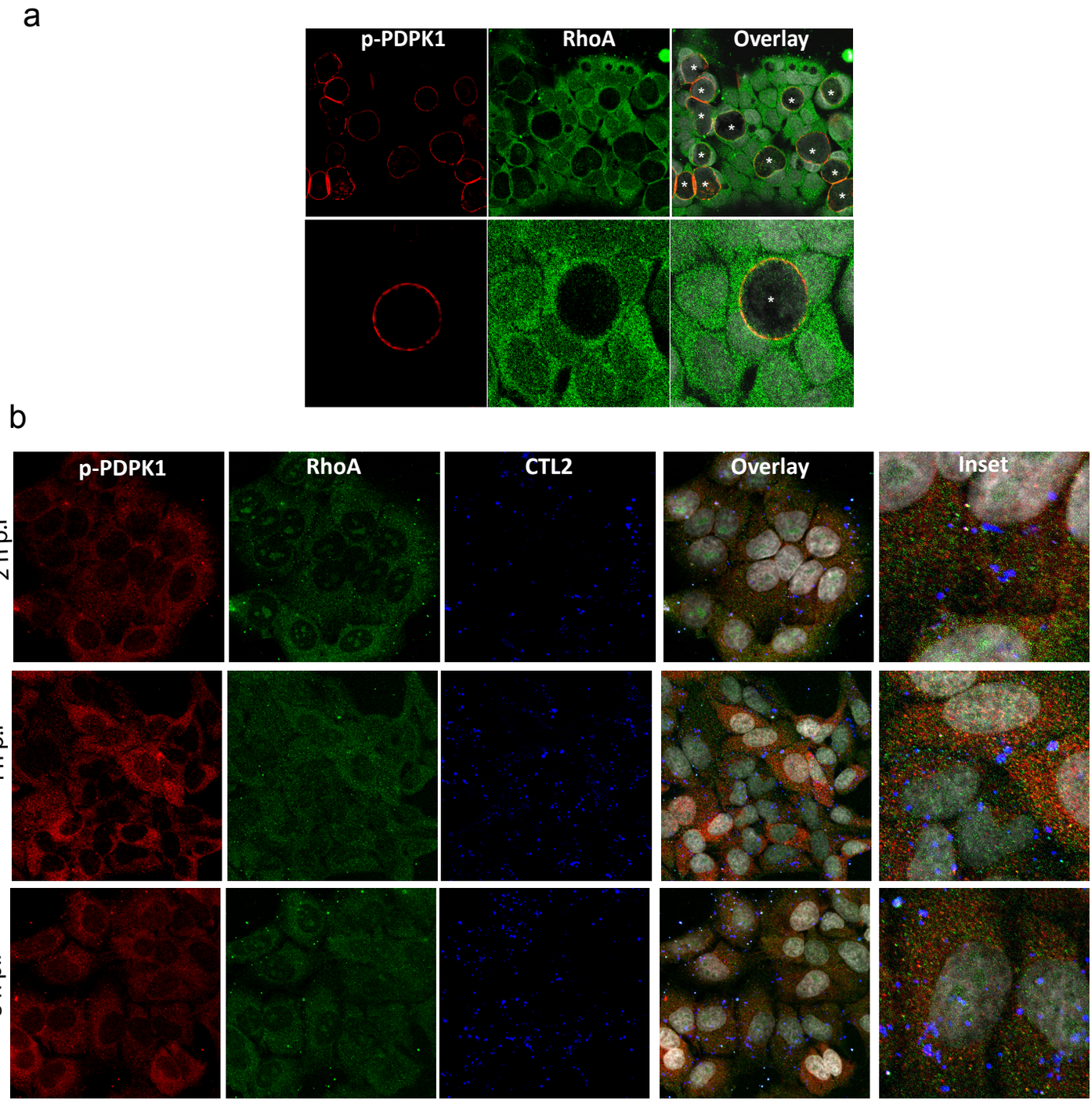


Supplementary Figure 1: PDPK1 and PLK1 exhibit vesicular staining throughout the cytoplasm with minimal localization at the bacterial inclusion

a) Monolayers of HeLa cells were infected with *Ctr* (MOI 0.5) for 48 h p.i. and labelled with anti-PDPK1 antibody and DAPI. Infection induced partial accumulation of PDPK1 (arrowheads) at the inclusions (asterisks).

b-c) Monolayers of HeLa cells (b) and fallopian tube mesenchymal stem cells (c) infected with *Ctr* (MOI 0.5) for 48, and labelled with antibodies against PLK1 and p-PLK1 and DAPI. PLK1 and p-PLK1 staining exhibited vesicular distribution in infected cells with minor accumulation (arrowheads) at the inclusions (asterisks). Scale bar: 30 µm.

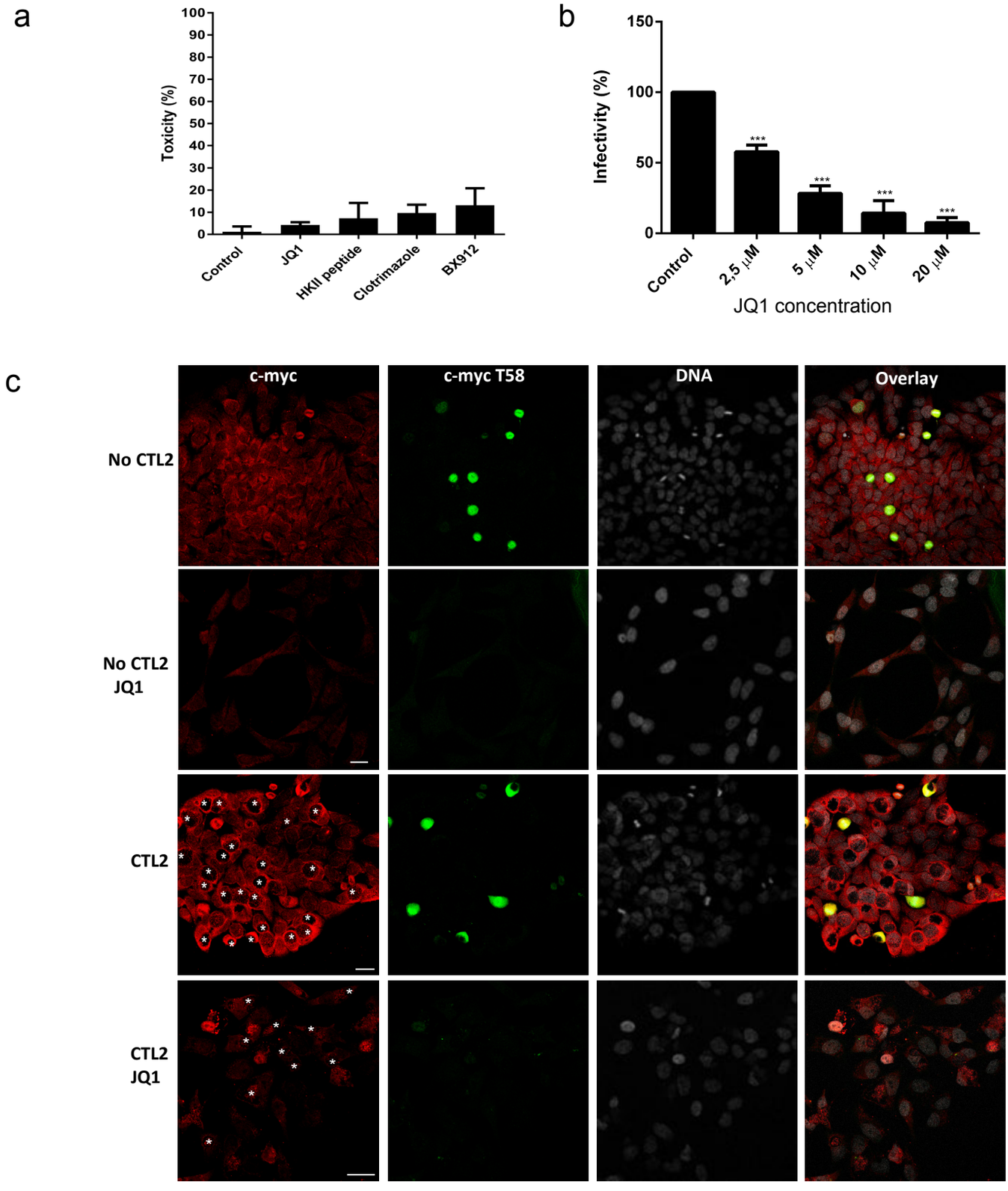


Supplementary Figure 2: p-PDPK1 but not RhoA is recruited specifically to the bacterial inclusions at mid and late stages of infection only

a) Monolayers of HeLa cells infected with CTL2 (MOI 0.5) for 48 h p.i. were labelled with anti p-PDPK1 and anti-RhoA antibodies and DAPI. Infection induced accumulation and recruitment of p-PDPK1 (asterisks) but not RhoA at the inclusions.

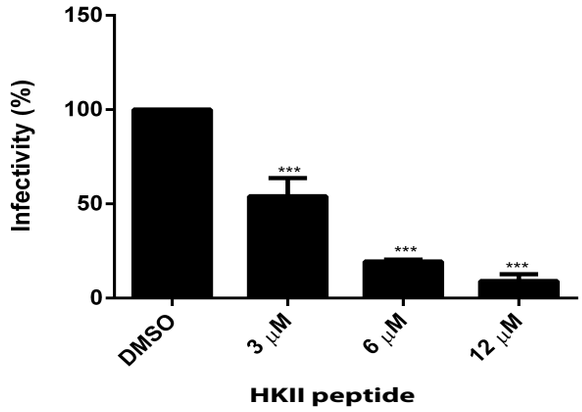
b-c) Monolayers of HeLa cells infected with CTL2 (MOI 50) for 2-8 h p.i. were labelled with antibodies against p-PDPK1, RhoA and CTL2 and DAPI. Neither p-PDPK1 nor RhoA is not recruited to the inclusions at these time points. c) Western blotting analysis revealed that levels of MYC phosphorylated at Ser-62 remained unchanged upon infection at early time points. β-actin served as loading control.

d) Monolayers of HeLa cells infected with CTL2 (MOI 0.5) for 2 h were treated with 1 μg/ml cycloheximide for an additional 46 h. Cycloheximide did not inhibit the increase MYC phosphorylated at Ser-62 upon infection. β-actin served as loading control.

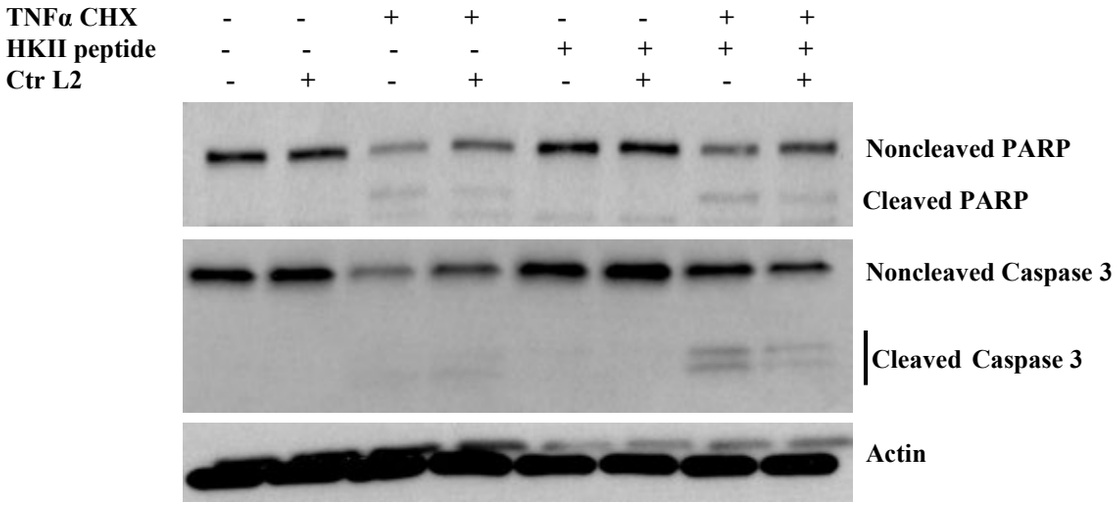


Supplementary Figure 3: JQ1 abrogates Chlamydia replication and inhibits Myc/HKII induction
a) Monolayers of HeLa cells were treated with the indicated drugs (20 µM JQ1 for 44 h, 20 µM HKII peptide for 24 h, 50 µM clotrimazole for 24 h or 10 µM BX912 for 36 h) before measuring lactate dehydrogenase released into the medium as a measure of cell toxicity.
b) Fallopian tube mesenchymal stem cells were infected with CTL2 (MOI 0.5), treated with increasing doses of JQ1 at 10 h p.i and the generation of infectious EBs determined via infectivity assay. Results depicted as mean ± SD normalized to control of three independent experiments; ***$p < 0.0001$.
c) JQ1 abrogates the induction of MYC in HeLa cells infected with CTL2 (MOI 0.5). Cells were labeled with antibodies against MYC, p-MYC and DAPI. Scale bar: 30 µm.
d-e) Western blotting analysis from human primary fallopian mesenchymal stem cells (d) and epithelial cell organoids (e) showing increased levels of HKII protein at 48 h p.i. in CTL2-infected whole cell lysates. The increase was abrogated in MSCs treated with the inhibitor JQ1. β-actin served as loading control.

a



b



Supplementary Figure 4: HKII competitive peptide abrogates bacterial replication and sensitizes primary cells to apoptosis

a) Fallopian tube mesenchymal stem cells were infected with CTL2 (MOI 0.5) and treated with different doses of HKII competitive peptide for 24 h p.i before determining the generation of infectious EBs via infectivity assay. Results are depicted as mean ± SD normalized to controls of three independent experiments; ***$p < 0.0001$.

b) Apoptosis induction upon TNFα treatment was analyzed by western blotting using antibodies to cleaved PARP and caspase 3 in fallopian tube mesenchymal stem cells infected with CTL2 (MOI 1). Infected cells showed greatly reduced levels of cleaved PARP and caspase 3, however, 10 μM HKII peptide inhibitor treatment from 24 h p.i for 8 h completely re-sensitized cells to undergo apoptosis. β-actin served as loading control.