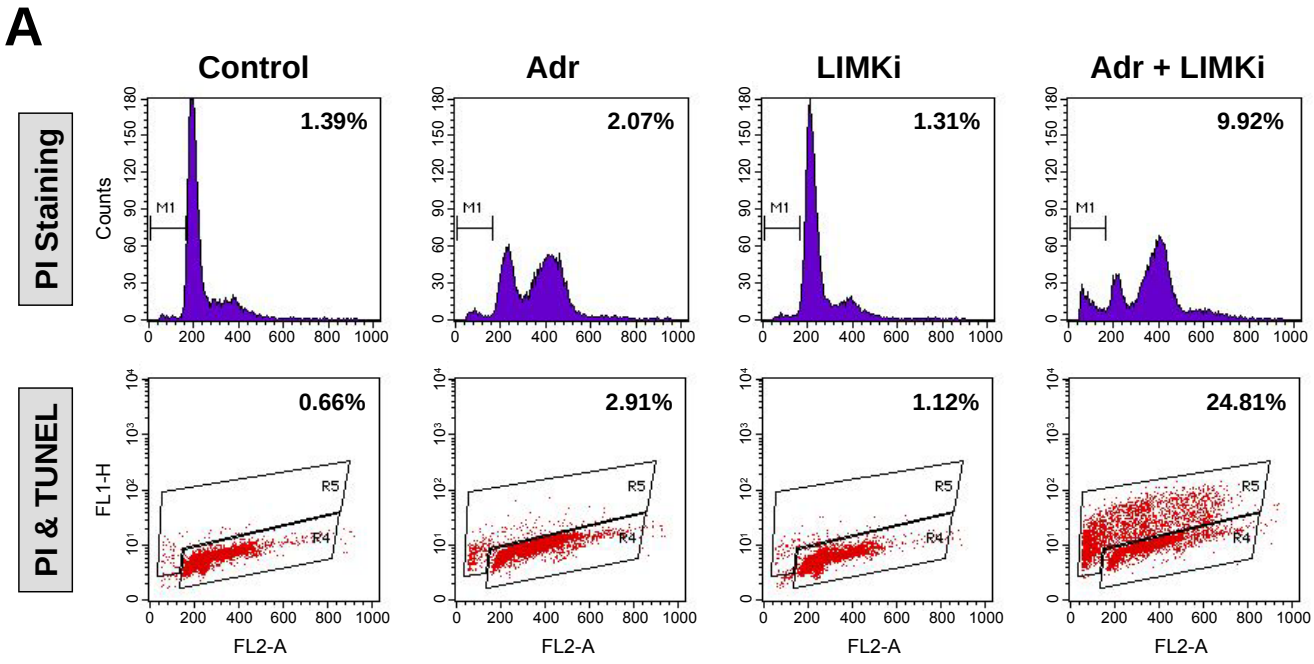
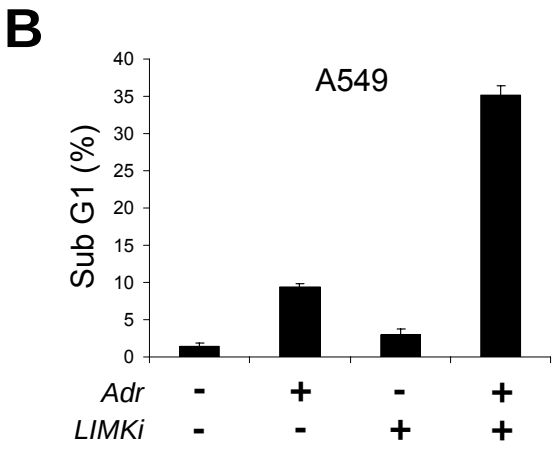
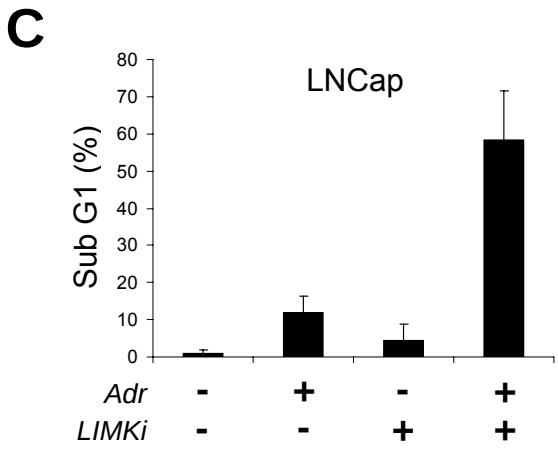
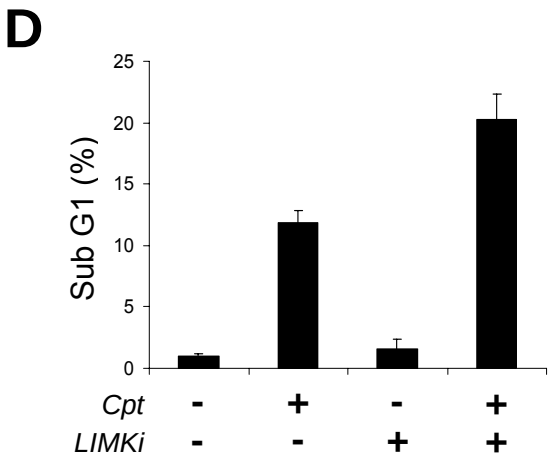
Fig. S7


A
Control
Adr
LIMKi
Adr + LIMKi
PI Staining
PI & TUNEL
Counts
FL1-H
FL2-A
M1
R4
R5
1.39%
2.07%
1.31%
9.92%
0.66%
2.91%
1.12%
24.81%


B
A549
Sub G1 (%)
Adr
LIMKi


C
LNCap
Sub G1 (%)
Adr
LIMKi


D
Sub G1 (%)
Cpt
LIMKi

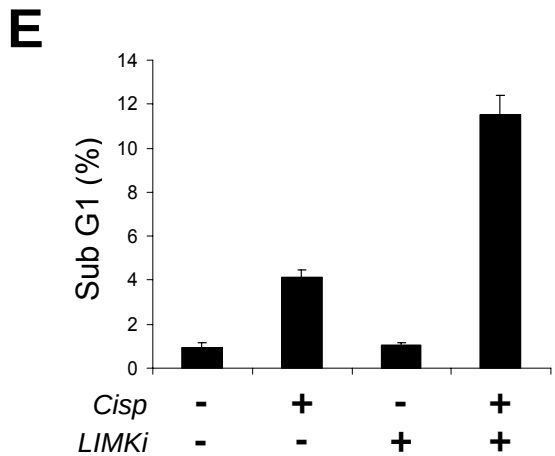

E
Sub G1 (%)
Cisp
LIMKi

Figure S7 LIMK inhibiton synergizes with DNA-damaging agents to promote apoptosis. MCF-7 cells were treated with (**A**) Adr (0.2 μg/ml) in the presence or absence of LIMKi (10 μM) for 72 hours. Adherent and non-adherent cells were collected and processed for PI/TUNEL staining. Representative FACS profiles of PI staining of DNA content and TUNEL staining are shown. Percentages of apoptotic cells as measured by sub-G1 DNA content (M1 population) or TUNEL-positive cells (R5 population) are shown in each panel. (**B**) A549 cells and (**C**) LNCaP cells were treated with Adr (0.4 μg/ml and 0.3 μg/ml, respectively) in the presence or absence of LIMKi (10 μM) for 72 hours. MCF-7 cells treated with (**D**) 0.6 μM camptothecin (Cpt) or (**E**) 100 μM cisplatin (Cisp) in the presence or absence of LIMKi (10 μM) for 72 hours. Adherent and non-adherent cells were collected and processed for PI staining. Apoptotic cell death is shown as the mean percentage sub-G1 cells ± SEM (n=3-6).