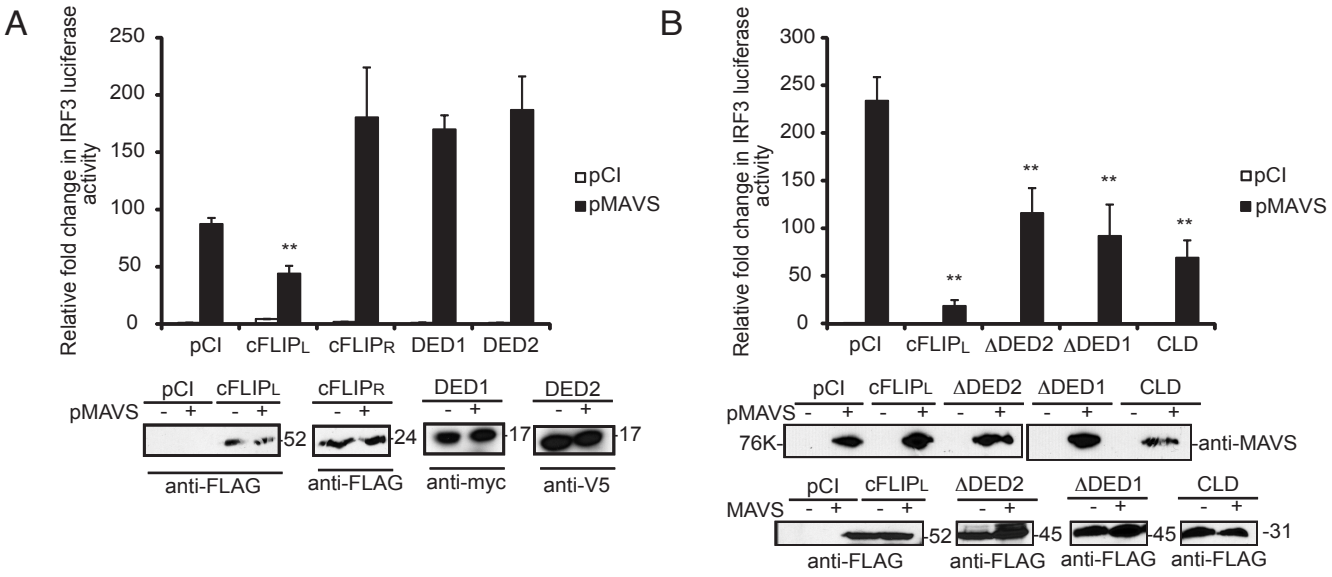
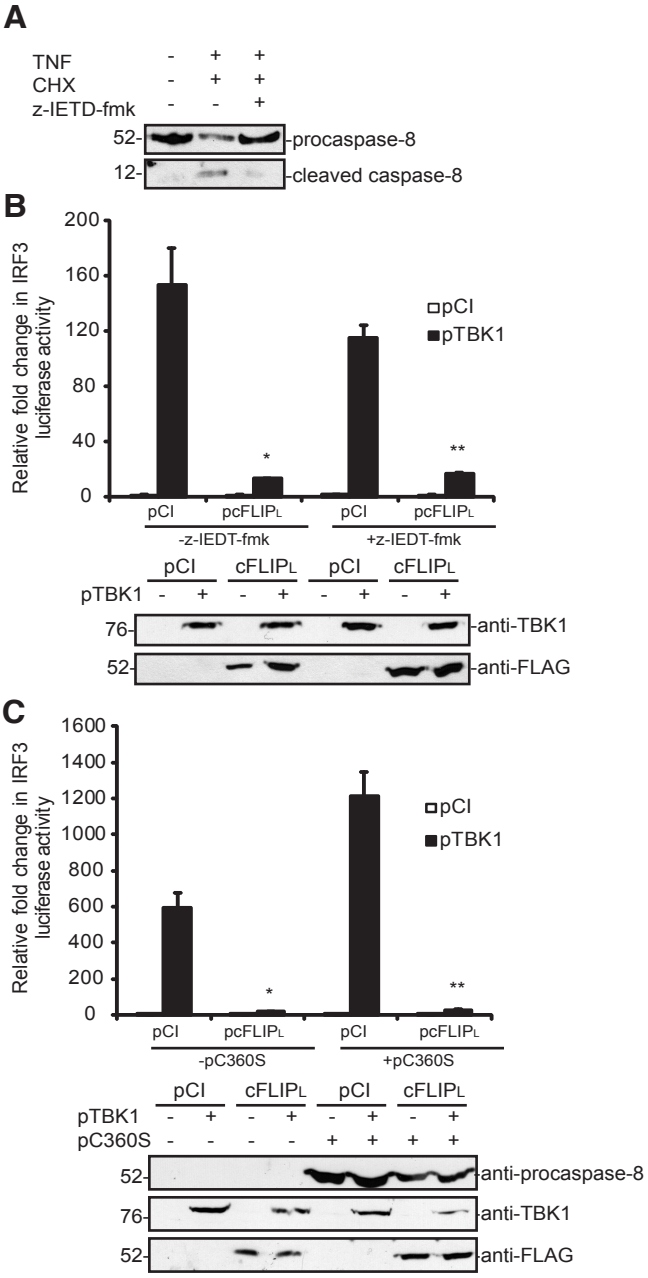
Supplemental Figure 1



Supplemental Figure 1. The CLD of cFLIP$_L$ inhibits MAVS-induced IRF3 activation.

Luciferase reporter assays in 293T cells transiently co-transfected with combinations of plasmids containing wild-type or mutant cFLIP-based constructs (A) (Δ DED1, ΔDED2, or CLD) or (B) (cFLIP$_L$, cFLIP$_R$, DED1, or DED2) and empty vector (pCI) or pMAVS for 24 h. * $P < 0.05$, compared with cells transfected with empty vector (Student's *t*-test). Immunoblot analysis of cellular lysates for cFLIP protein expression also was performed.

Supplemental Figure 2



Supplemental Figure 2. cFLIP$_L$ inhibits IRF3 activation independent of apoptosis.

(A) An immunoblot analysis shows that the concentration of z-IETD-fmk (50 microM) used for these experiments inhibits caspase-8 activation. (B) Inhibition of apoptosis, using z-IETD-fmk, still allows IRF3 activation. Results are presented as fold-induction of luciferase activity, presented relative to those of cells transfected with empty vector (pCI) and incubated in regular medium. * $P < 0.05$ and ** $P < 0.005$. Immunoblotting of cellular lysates shows cFLIP$_L$ and TBK1 protein expression. (C) Inhibition of apoptosis, using co-expression of a dominant negative procspase-8 protein (pC360S), still allows IRF3 activation. Results are presented as fold-induction of luciferase activity, presented relative to those of cells transfected with empty vector (pCI). *$P < 0.05$ and **$P < 0.005$, compared with cells transfected with empty vector. Immunoblotting of lysates show procaspase-8, cFLIP$_L$ and TBK1 protein expression.