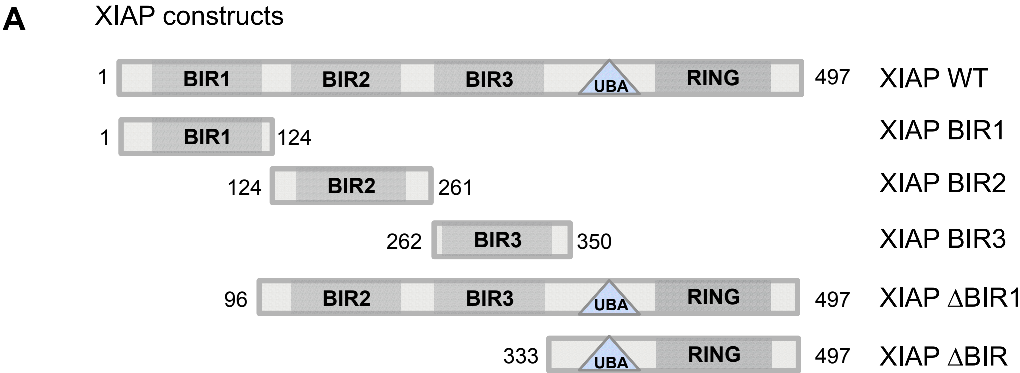
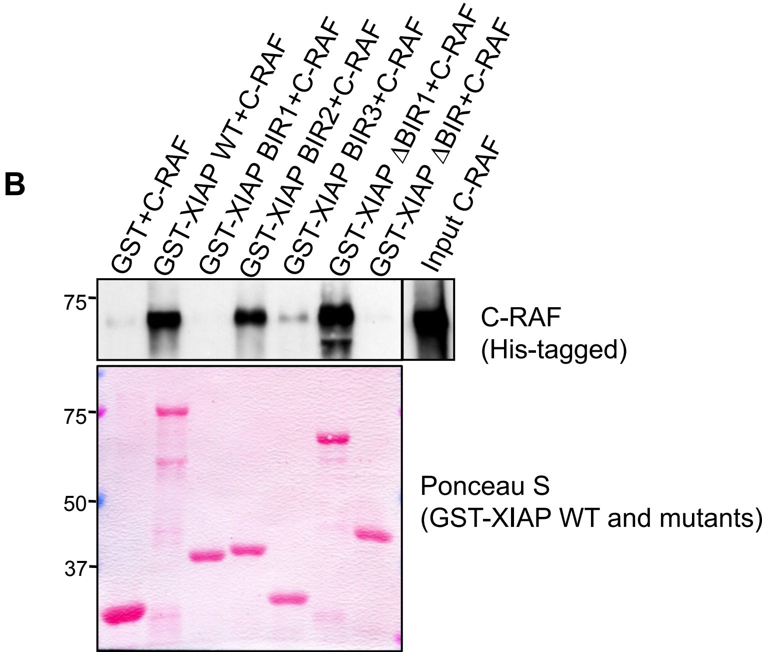
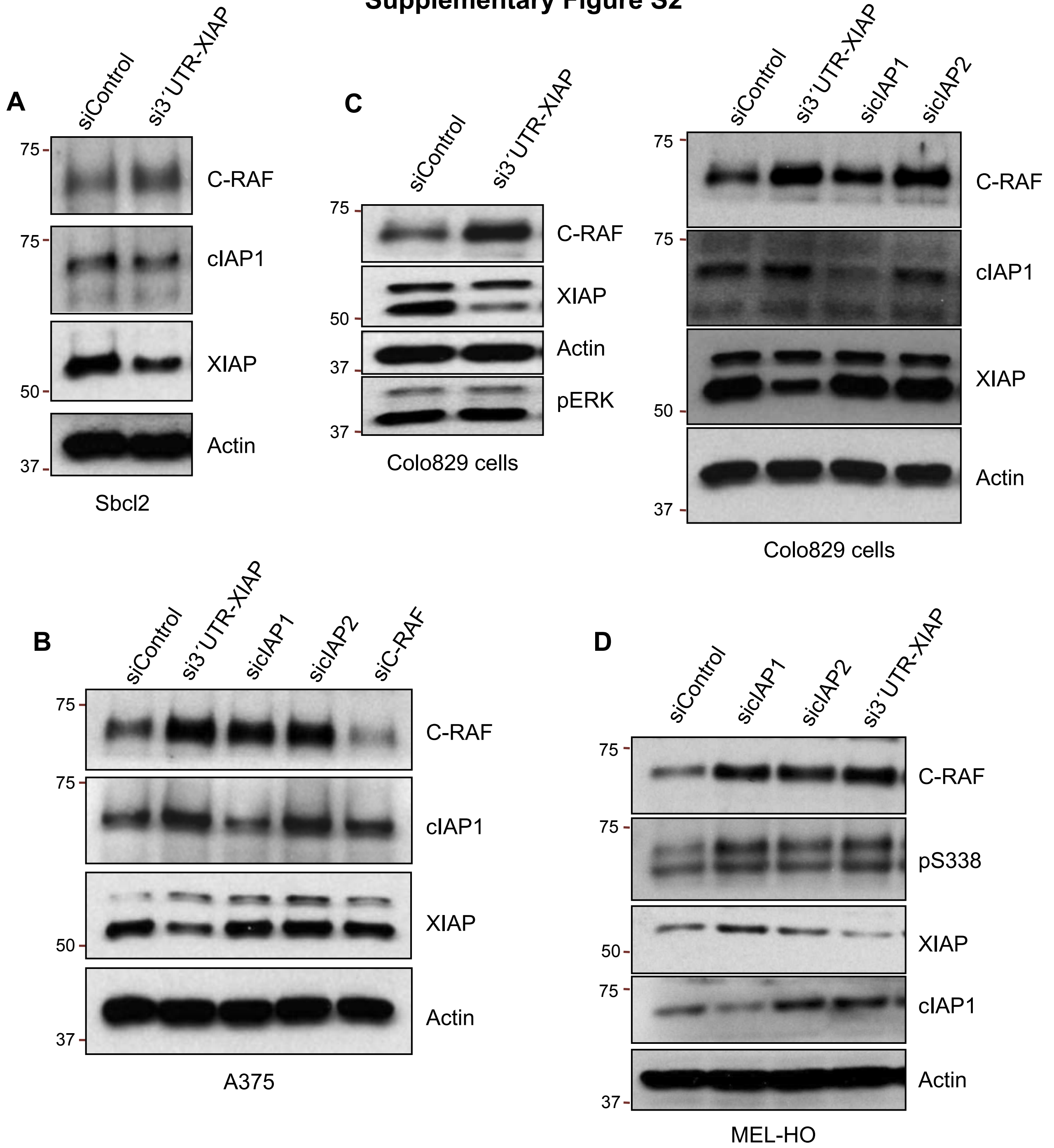
Supplementary Figure S1

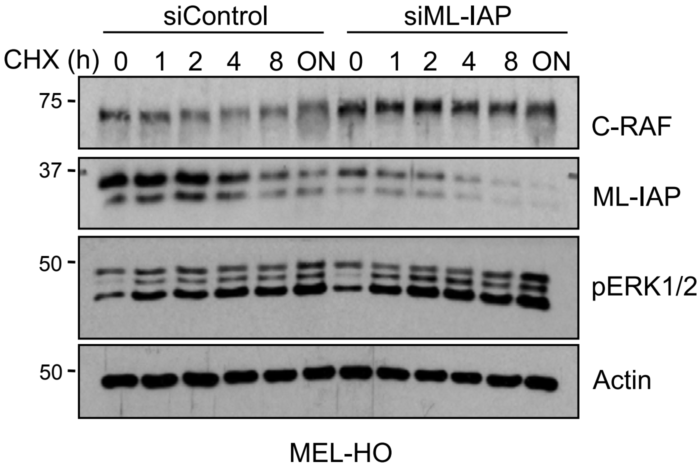


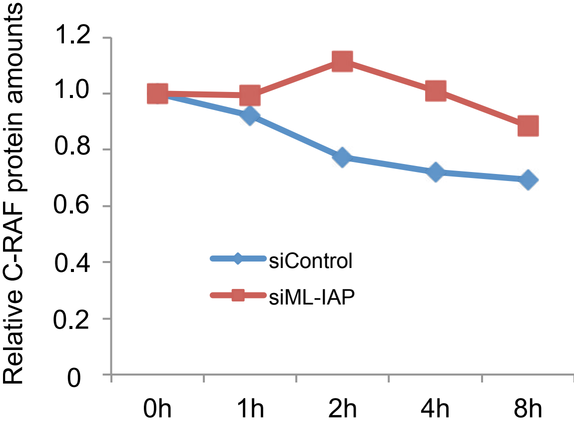


Supplementary Figure S2

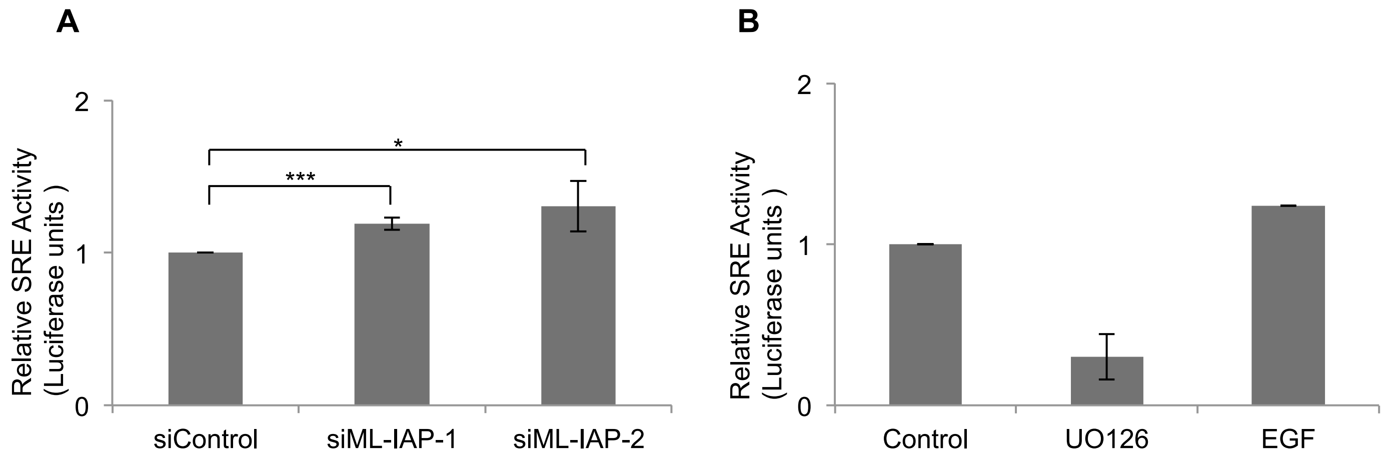


Supplementary Figure S3





Supplementary Figure S4



Supplementary Figure legends

Suppl. Figure S1: C-RAF binds specifically to Type II BIR domains of XIAP. (**A**) Pictorial representation of GST-tagged XIAP WT and BIR domain mutants used for the GST-pulldown experiments with C-RAF. (**B**) GST-tagged XIAP WT and BIR domain mutants were used to pulldown His-C-RAF and then the results were checked by Western blotting.

Suppl. Figure S2: Loss of IAPs leads to increase in C-RAF protein levels. siRNA mediated knockdown of IAPs in Sbcl2 (Ras mutant) (**A**), A375 (BRAF V600E) (**B**), Colo829 cells (BRAF V600E) (**C**), MEL-HO (myc) cells (**D**) lead to an increase in C-RAF levels, which was monitored by Western blot analysis. All siRNAs are validated in melanoma or other cell lines.

Suppl. Figure S3: Loss of ML-IAP leads to C-RAF protein stability. (**A**) Cycloheximide chase in MEL-HO cells with knockdown of ML-IAP shows an increase in the C-RAF protein levels at all timepoints as compared to control cells using western blotting. (**B**) Relative quantification of C-RAF protein levels in (A) by Image J software.

Suppl. Figure S4: Loss of ML-IAP leads to MAPK activation. (**A**) MEL-HO cells were stably transfected with SRE reporter construct having a downstream luciferase reporter. Knockdown of ML-IAP was performed using two different siRNAs and luciferse activity was monitored. Shown is the quantification from three different experiments. (**B**) Control for SRE luciferase assay as shown in (A). UO126 treatment used as a negative control decreases the luciferase activity and EGF stimulation as a positive control increases the SRE luciferase activity. Student t-test was performed to check for the significance (* = $P<0.05$, **= $P<0.01$, ***= $P<0.005$).