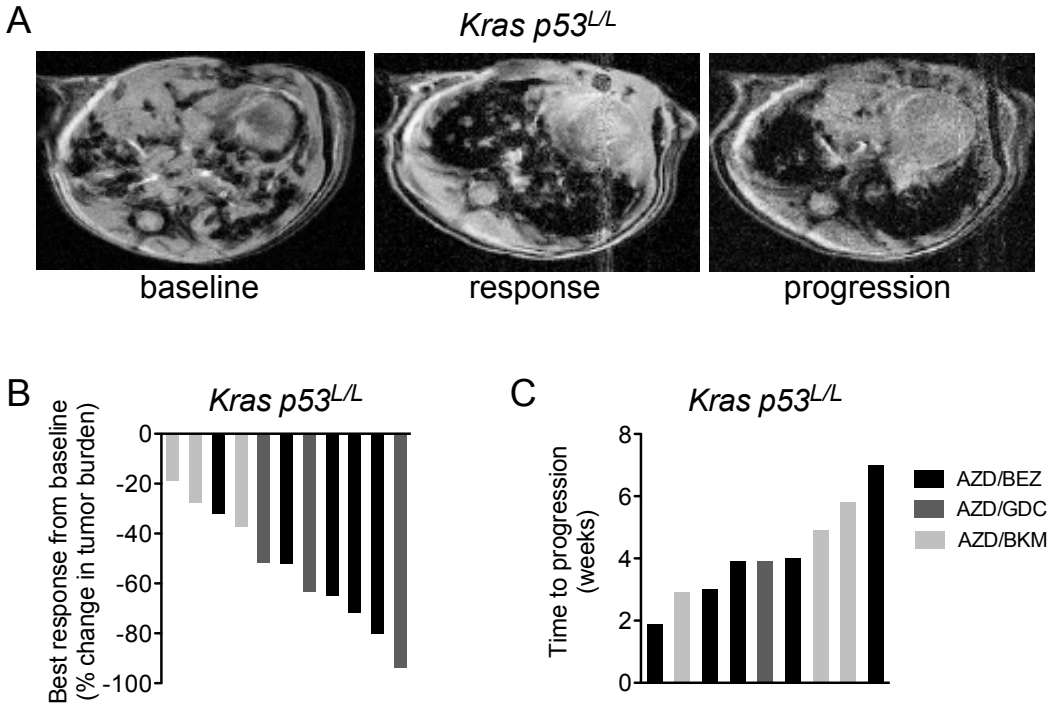
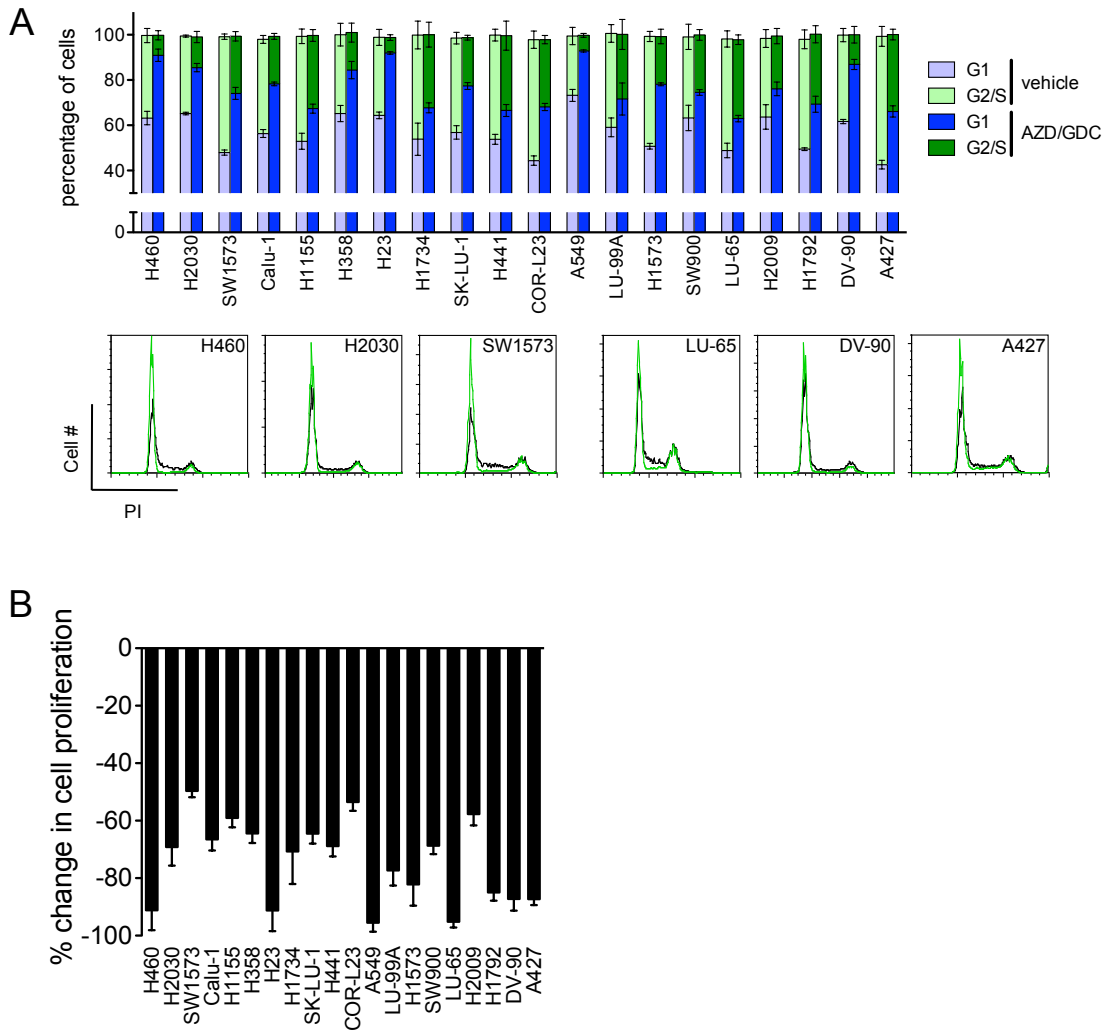


D

	Response (%)		TTP (weeks)	
AZD + BEZ	-60 ± 8	(n=5)	4.0 ± 0.8	(n=5)
AZD + GDC/BKM	-49 ± 11	(n=6)	4.4 ± 0.6	(n=4)
All	-54 ± 7	(n=11)	4.1 ± 0.5	(n=9)

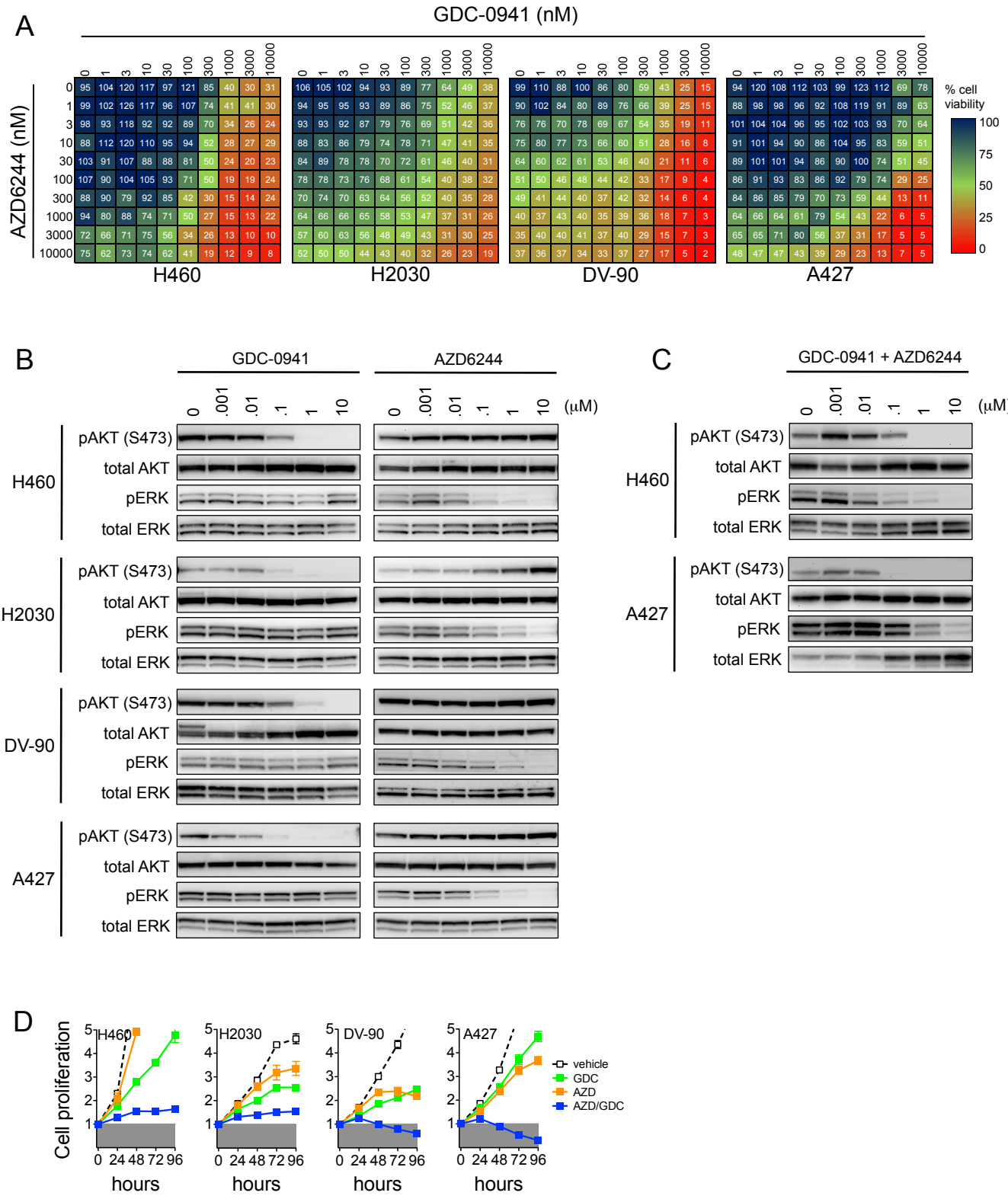
Supplemental Figure 1. Response of *Kras* mouse models to MEKi/PI3Ki
Kras p53$^{L/L}$ mice were treated with MEKi/PI3Ki (AZD6244/BEZ235, AZD6244/GDC-0941 or AZD6244/BKM120) and tumor burden measured by MRI scan.
A. Representative MRI images showing baseline, maximal response, and progression.
B. Waterfall plot depicting best tumor response as percentage decrease from baseline.
C. Waterfall plot showing time to progression of tumors.
D. Average response and time to progression for MEK inhibition combined with dual PI3KI/mTOR inhibition (BEZ235) versus pure PI3K inhibition (GDC-0941 and BKM120).



Supplemental Figure 2. MEKi/PI3Ki induces G1 arrest and inhibits proliferation of *KRAS* mutant NSCLC cell lines.

A. Cells were treated with 1 μM AZD6244 and 1 μM GDC-0941 for 24 hours, stained with propidium iodide, and cell cycle sub-populations were analyzed by flow cytometry. Data shown are mean and standard error of three independent experiments. Lower panel shows representative cell cycle histograms (black - vehicle; green - AZD/GDC).

B. Cells were treated with 1 μM AZD6244 and 1 μM GDC-0941 for 72 hours and cell proliferation was determined by CellTiter-Glo viability assay. Data are plotted as percentage of vehicle treated cells and are the mean and standard error of three independent experiments.



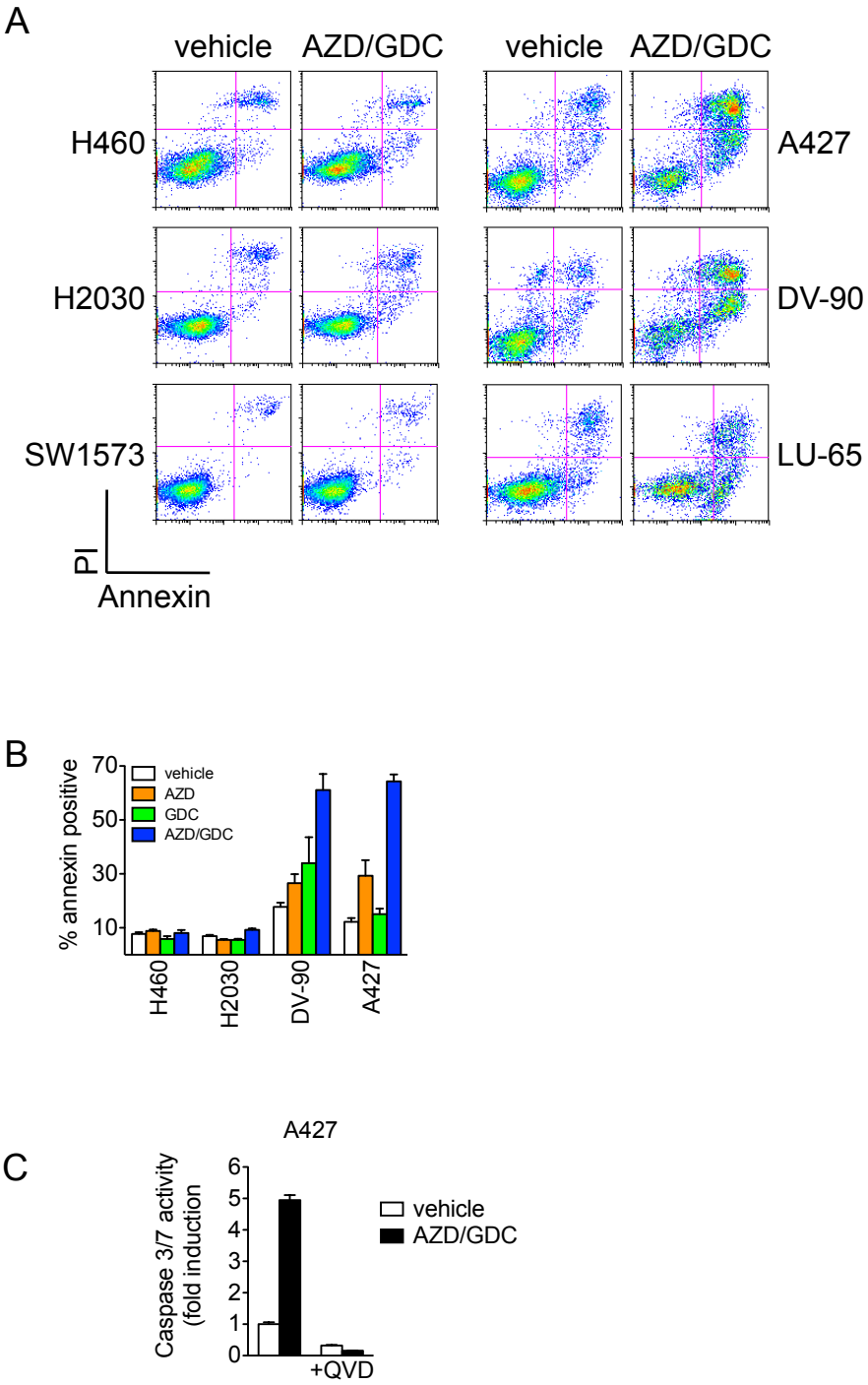
Supplemental Figure 3. Combined MEK and PI3K inhibition is necessary for maximal reduction in cell proliferation.

A. *KRAS* mutant NSCLC cell lines were treated with varying concentrations of AZD6244 and GDC-0941 in a 2x2 dose matrix for 72 hours and cell proliferation was determined by CellTiter-Glo viability assay. Values shown are % viability relative to vehicle control.

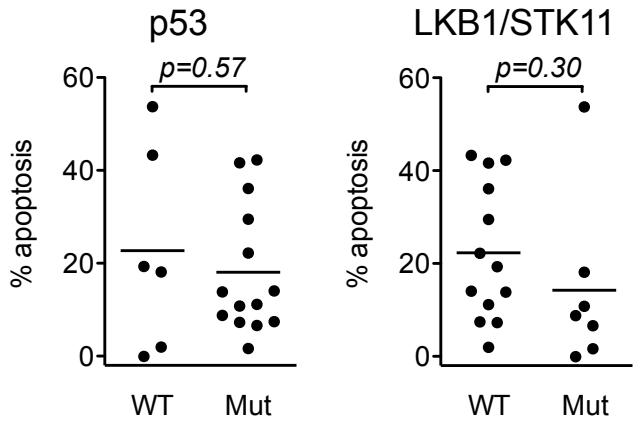
B. Cells were treated with varying concentrations of AZD6244 or GDC-0941 for 24 hours and harvested for western blot analysis using the indicated antibodies.

C. Cells were treated with varying equimolar concentrations of AZD6244/GDC-0941 for 24 hours and harvested for western blot analysis using the indicated antibodies.

D. Cells were treated with 1 mM AZD6244, GDC-0941 or combination for up to 96 hours and cell proliferation was determined at the indicated time points. Data are normalized to cell viability at the time of initial drug treatment.

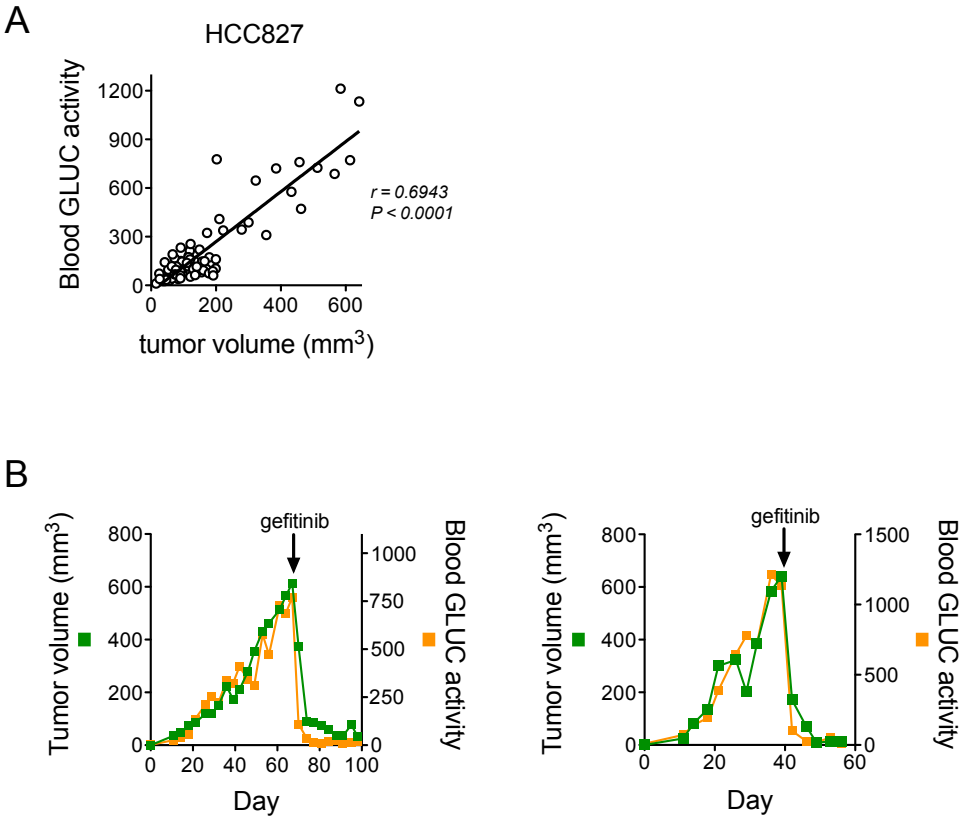


Supplemental Figure 4. Apoptotic reponse of *KRAS* mutant NSCLC cell lines in response to MEKi/PI3Ki.
A. Representative PI/annexin flow cytometry plots showing differential apoptotic response induced by AZD6244/GDC-0941, corresponding to data shown in Figure 1A.
B. Cell lines were treated with 1 μM AZD6244, GDC-0941 or combination for 72 hours and apoptosis determined by annexin staining and flow cytometry. Data shown are mean and standard error of 3 independent experiments.
C. A427 cells were treated with AZD6244/GDC-0941 as above in the absence or presence of the pan-caspase inhibitor QVD-Oph and caspase-3/7 activity determined. The mean and standard error of triplicate measurements are shown.



Supplemental Figure 5. Mutational status of TP53 or STK11/LKB1 does not correlate with apoptotic response to MEKi/PI3Ki.

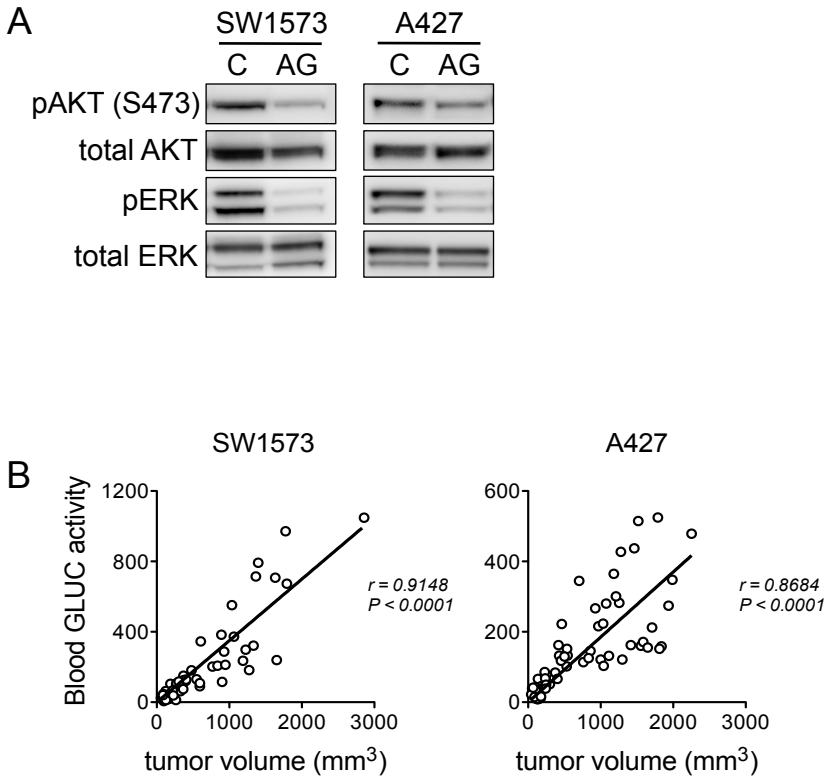
Apoptosis was determined by annexin staining and flow cytometry; data values are from Figure 1A. Mutational status of cells lines shown in Table S1.



Supplemental Figure 6. Secreted Gaussia luciferase allows for precise quantitation of tumor growth and treatment response.

A. HCC827 cells were engineered to express Gaussia luciferase (GLUC) and subcutaneous HCC827-GLUC xenografts were established in nude mice.Tail bleeds were performed and secreted luciferase in whole blood was measured by luminescence. Correlation plots of blood GLUC levels vs tumor volume for untreated animals. Spearman correlation coefficients are shown.

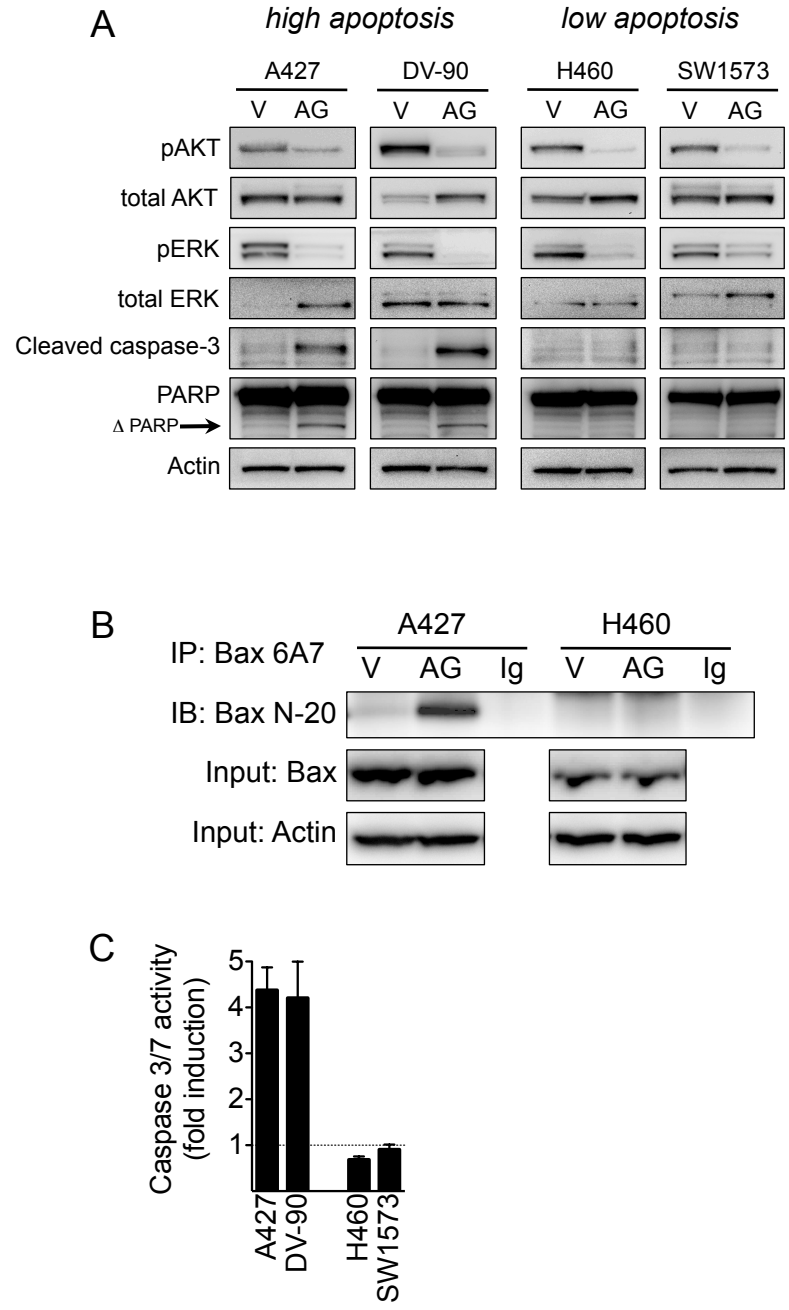
B. Tumor measurements compared with blood GLUC levels in two different mice before and after treatment with the EGFR inhibitor gefitinib (arrow).



Supplemental Figure 7. Tumor response of *KRAS* NSCLC xenografts to MEKi/PI3Ki

A. Decreased pAKT and pERK in SW1573 xenografts following treatment with GDC-0941 and AZD6244. Tumors were harvested 3 hrs after third dose of AZD6244 and GDC-0941 (AG) and subjected to western blot analyses with the indicated antibodies. (C = control, untreated tumor)

B. Tail bleeds were performed on mice bearing SW1573-GLUC and A427-GLUC xenografts and secreted luciferase in whole blood was measured by luminescence. Correlation plots of blood GLUC levels vs tumor volume for untreated animals. Spearman correlation coefficients are shown.

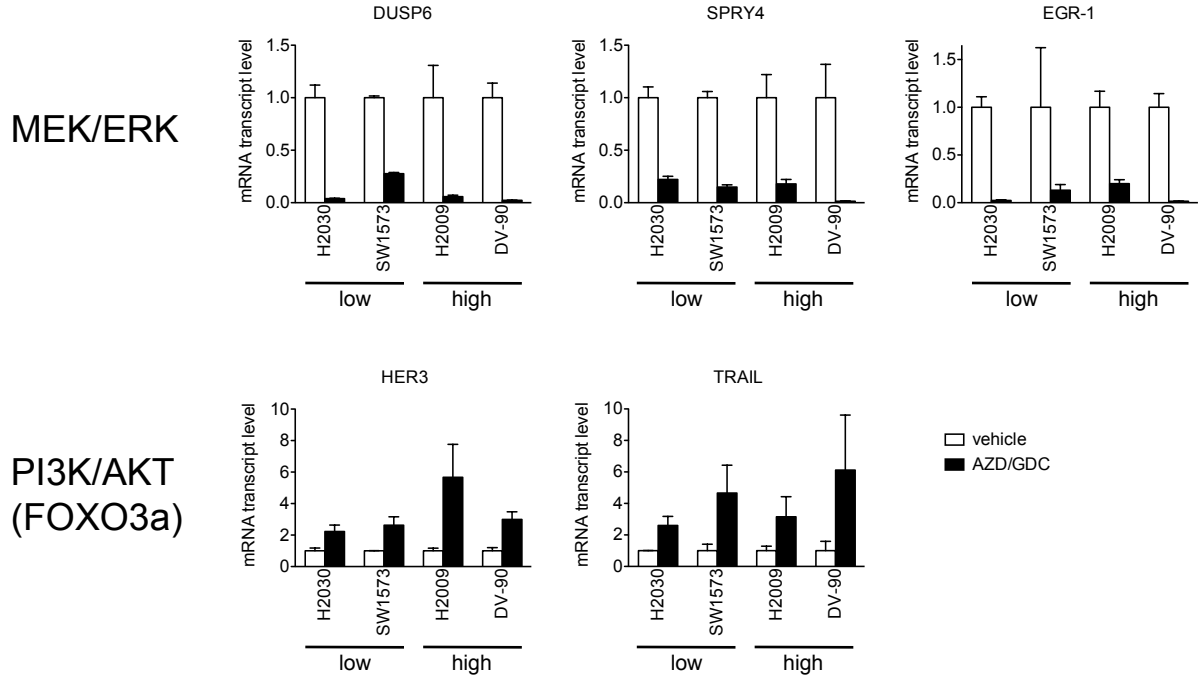


Supplemental Figure 8. Apoptosis induced by MEKi/PI3Ki is BAX and caspase-3 dependent

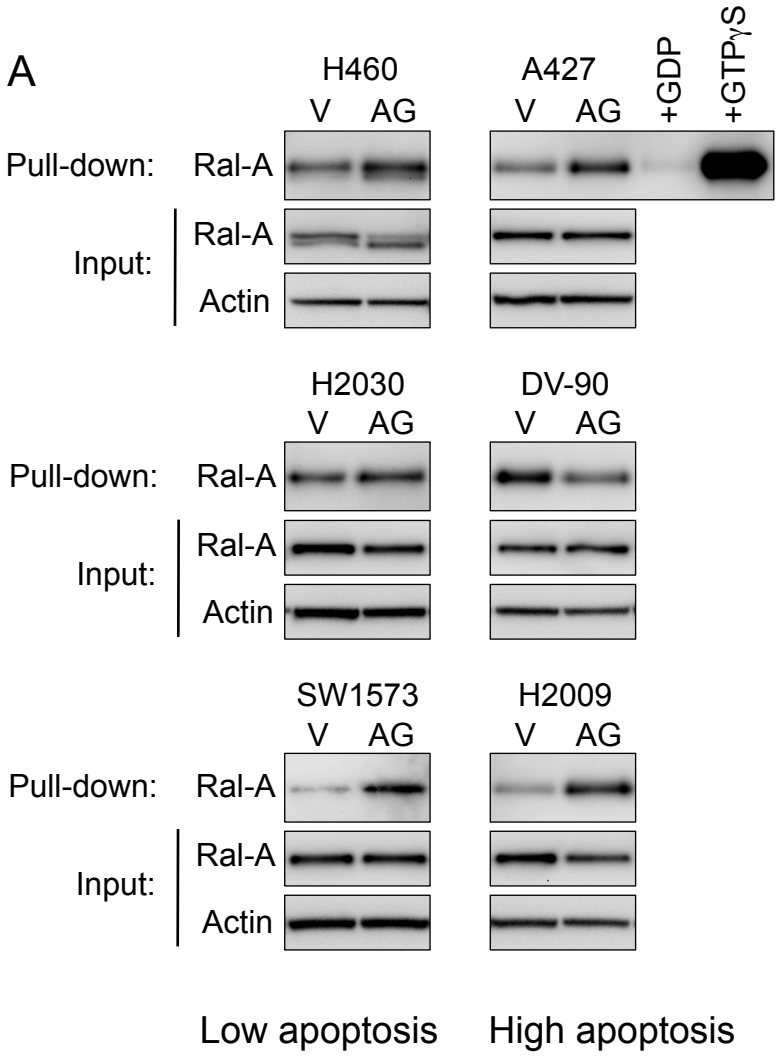
A. Cell lines were treated with 1 μM AZD6244/GDC-0941 (AG) or vehicle (V) for 24 hours and protein lystates were analyzed by western blot analyses using the indicated antibodies.

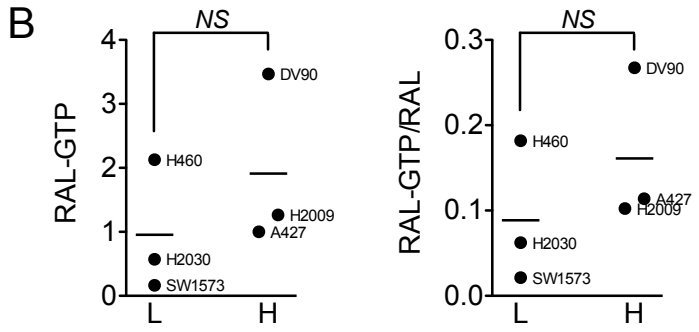
B. Cell lines were treated with 1 μM AZD6244/GDC-0941 for 24 hours. Activated BAX was immunoprecipitated with the conformation specific 6A7 antibody, and western blot analysis was performed with the N-20 antibody. (Ig, isotype control).

C. Cell lines were treated with 1 μM AZD6244/GDC-0941 for 48 hours and caspase-3/7 activity was determined. Data displayed are relative caspase activation compared with vehicle treated control. The mean and standard error of 3-5 independent experiments are shown.



Supplemental Figure 9. Modulation of MEK/ERK and PI3Ki/AKT transcriptional output does not correlate with apoptotic sensitivity of *KRAS* mutant NSCLC cancer cells.
Cell lines with low (H2030, SW1573) or high (H2009, DV-90) apoptotic response were treated with 1 μM AZD6244/GDC-0941(black bars) or vehicle (open bars) for 24 hours and mRNA levels were determined by quantitative RT-PCR (relative to GAPDH expression). Data are mean and error of duplicate samples, normalized to mRNA level of vehicle treated cells.

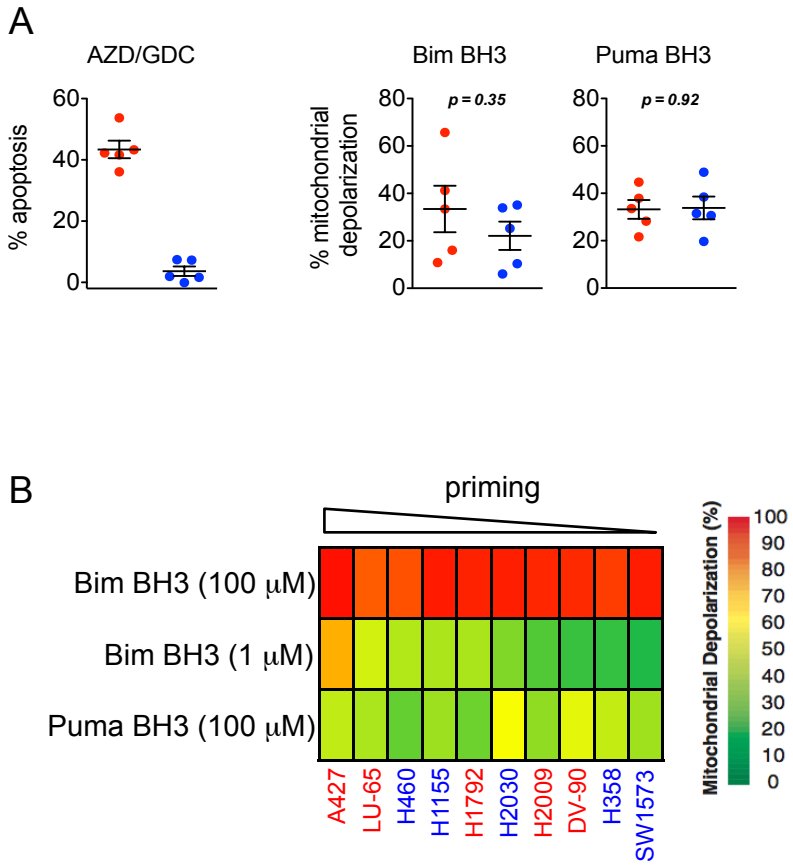




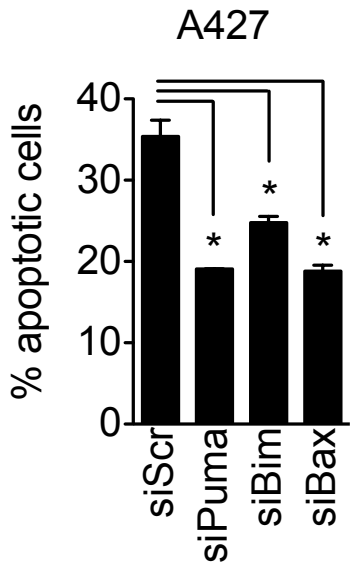
Supplemental Figure 10. Modulation of RalGDS signaling does not correlate with apoptotic sensitivity of *KRAS* mutant NSCLC cell lines.

A. Ral-GTP pull-down assay. Cell lines were treated with 1 mM AZD6244/GDC-0941 for 24 hours and Ral-GTP pull-down was performed. For positive and negative controls, lysates were incubated with GTPγS or GDP prior to pull-down, respectively.

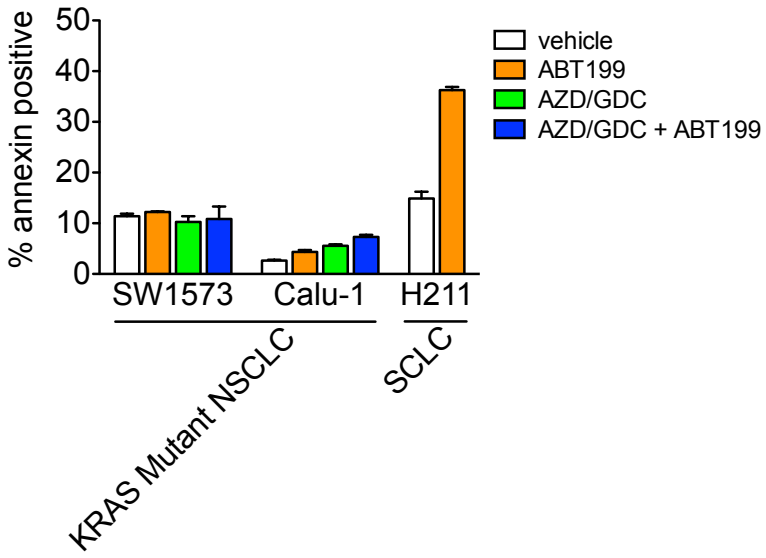
B. On left, Ral-GTP levels in pull-downs for untreated cells were quantified (from western blot) and normalized to actin. On right, Ral-GTP levels are expressed as a percentage of total Ral in cell lysates from untreated cells.



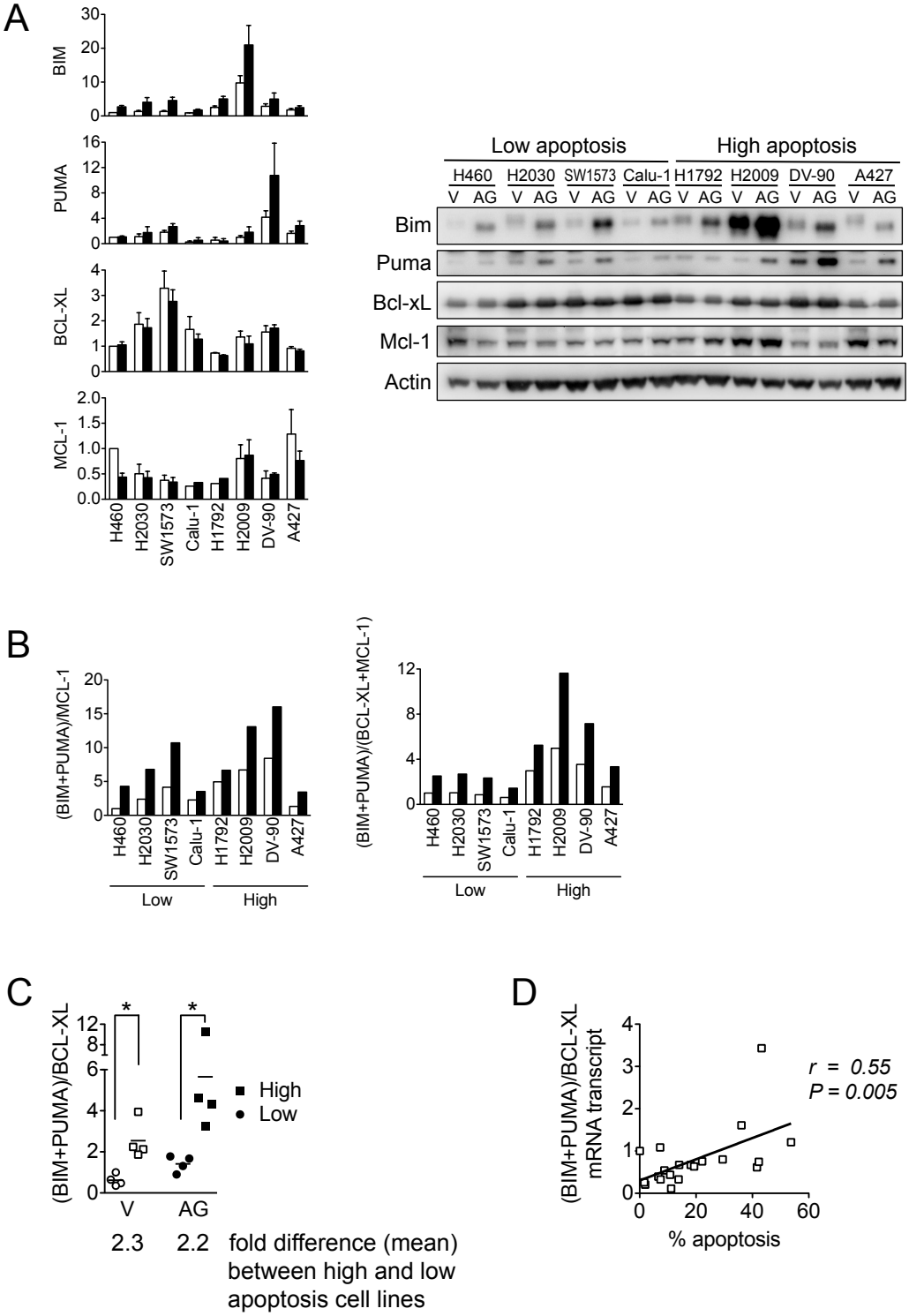
Supplemental Figure 11. Sensitivity to MEKi/PI3Ki does not correlate with BH3 priming.
A. BH3 profiling was performed on five *KRAS* mutant NSCLC lines that undergo apoptosis in response to AZD6244/GDC-0941 (red) and five that have no apoptotic response (blue). Mitochondrial depolarization induced by BIM (1 μM) or PUMA (100 μM) BH3 peptides are measures of baseline apoptotic priming of cells and were not different between the two groups.
B. Heat map showing individual depolarization values for each cell line in response to BIM (1 μM) and PUMA (100 μM) BH3 peptides. The BIM (100 μM) BH3 peptide served as a positive control for expression of the apoptosis effectors BAX and/or BAK.



Supplemental Figure 12. siRNA mediated knockdown of PUMA and BIM protects from apoptosis induced by MEKi/PI3Ki.
A427 cells were transfected with siRNA targeting PUMA, BIM and BAX and the apoptotic response to 1 μM AZD6244/GDC-0941 was determined by annexin staining and flow cytometry. Data shown are mean and standard error of duplicate experiments. Asterisks indicate significant difference ($p<0.05$) compared with scrambled siRNA control.



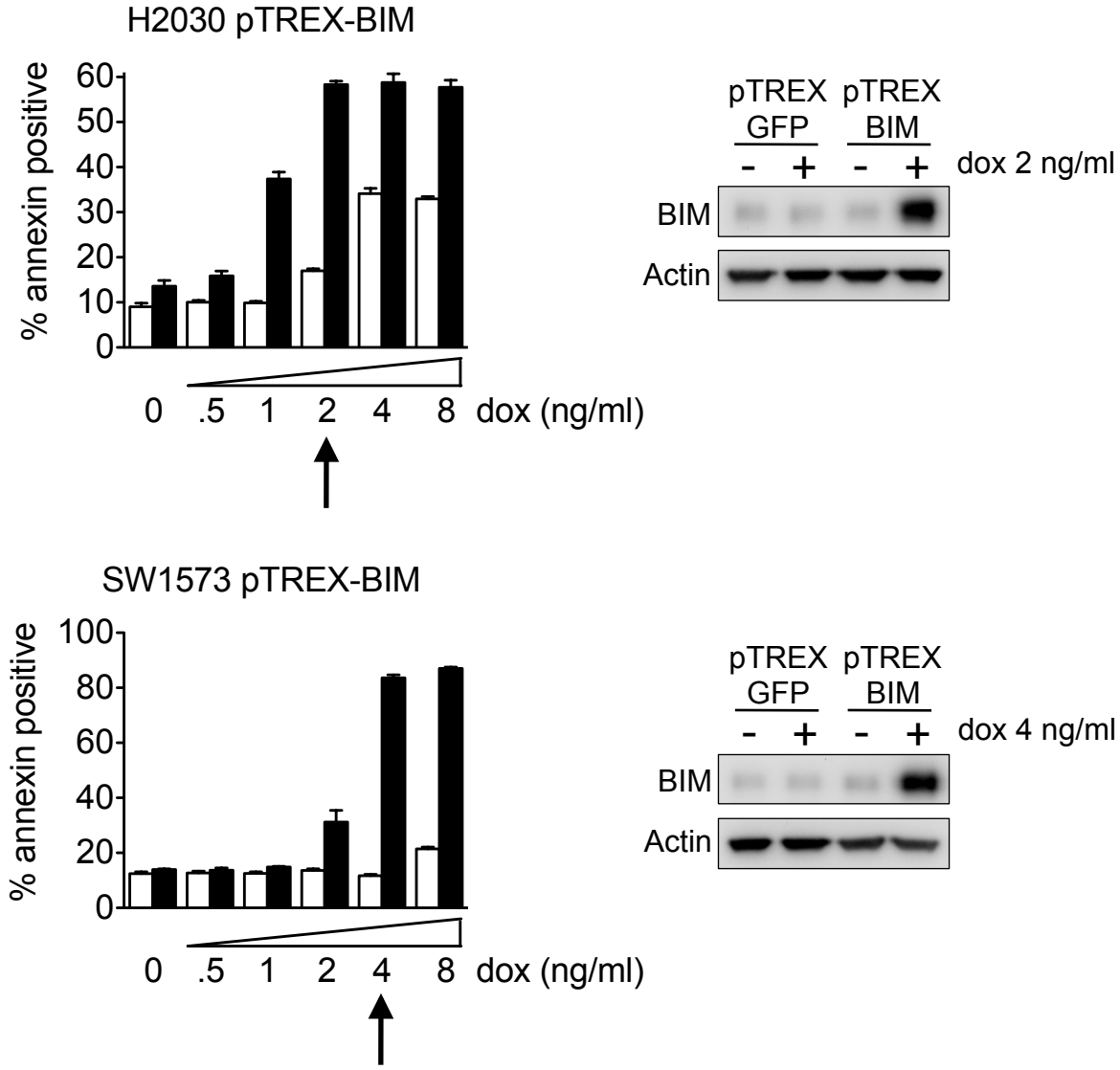
Supplemental Figure 13. Inhibition of BCL-2 alone does not restore apoptotic response in insensitive *KRAS* mutant NSCLC cell lines.
KRAS mutant SW1573 and Calu-1 NSCLC cell lines were treated with 1 μM AZD6244, GDC-0941, ABT199 or combination for 72 hours and apoptotic response determined. As a positive control for ABT-199 activity, the small cell lung cancer cell line H211 was treated with ABT-199 alone. Data shown are mean and standard error of triplicate samples.



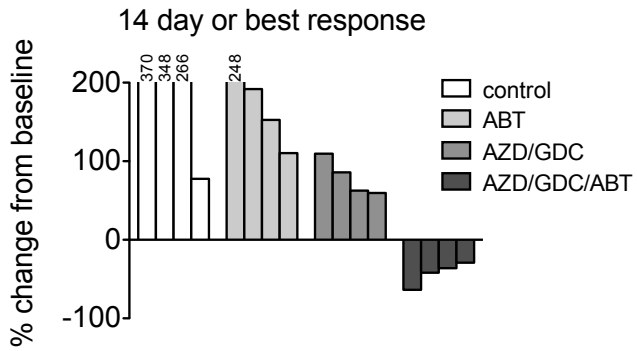
Supplemental Figure 14. Individual protein expression levels of BCL-2 family members do not correlate with sensitivity to MEKi/PI3Ki.

A-C. Cell lines were treated with 1 μM AZD6244/GDC-0941 (AG, black bars) or vehicle (V, open bars) for 24 hours and harvested for western blotting. Protein expression levels were quantified from western blots and normalized to vehicle treated H460 cells. Each data point is mean of three independent experiments.

D. mRNA was extracted from untreated cells and transcript levels were determined by quantitative RT-PCR. Spearman correlation coefficient is shown.

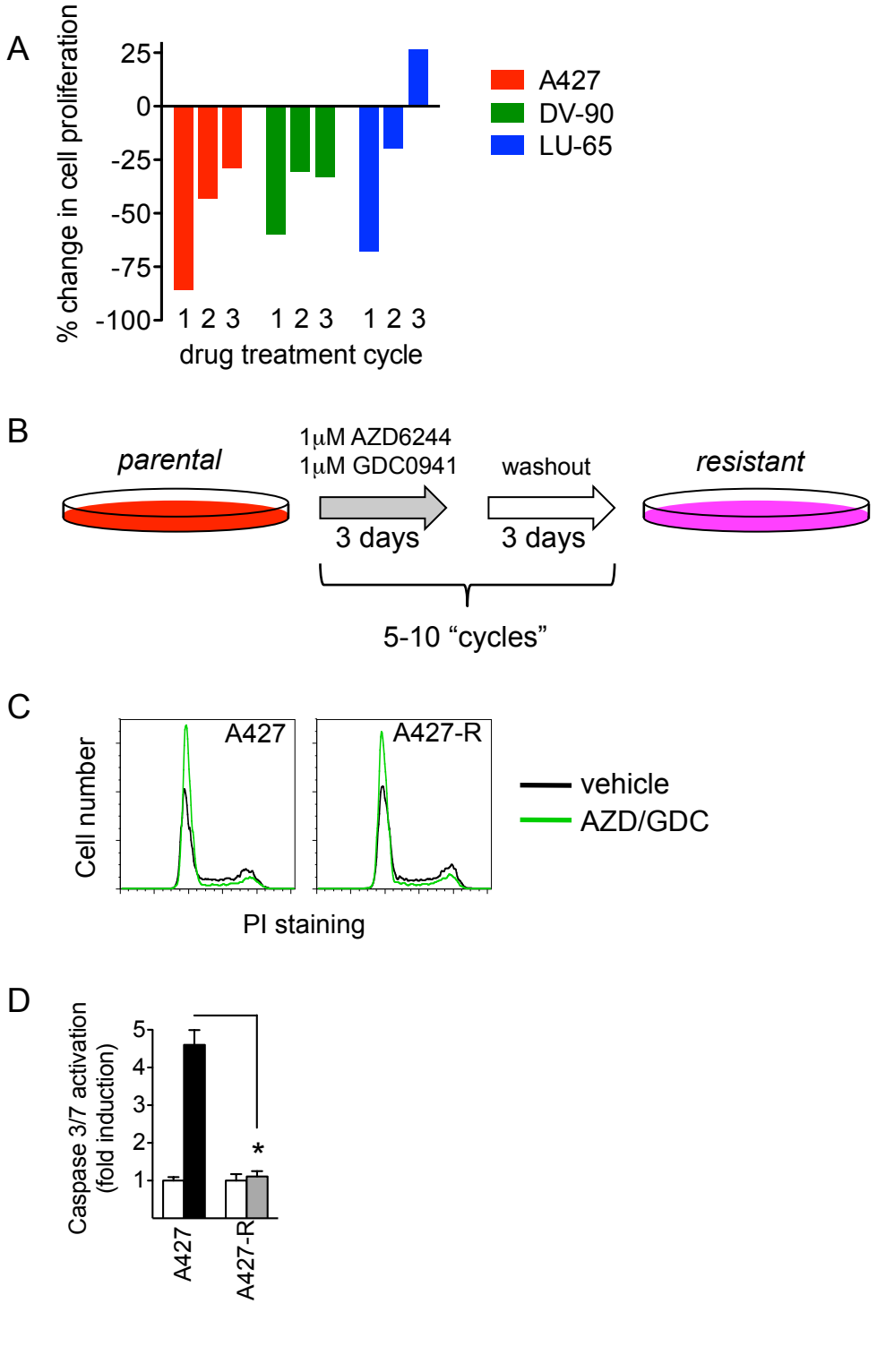


Supplemental Figure 15. Inducible ectopic expression of BIM
Cells expressing BIM under control of a tetracycline inducible promoter (pTREX) were treated with the indicated concentration of doxycycline for 72 hours in the presence of 1 μM AZD6244/GDC-0941 or vehicle and the apoptotic response was determined by annexin staining and flow cytometry. Data shown are mean and standard error of triplicate samples. Arrows indicate doxycycline concentrations used in subsequent studies. Western blots show BIM expression of corresponding cells after induction with doxycycline.



Supplemental Figure 16. Restoration of apoptosis by ABT-263 leads to MEKi/PI3Ki-induced regression in vivo.

Waterfall plots of 14 day or best response of mice bearing SW1573 xenografts treated with AZD6244/GDC-0941, ABT-263 or combination corresponding to Figure 3E.



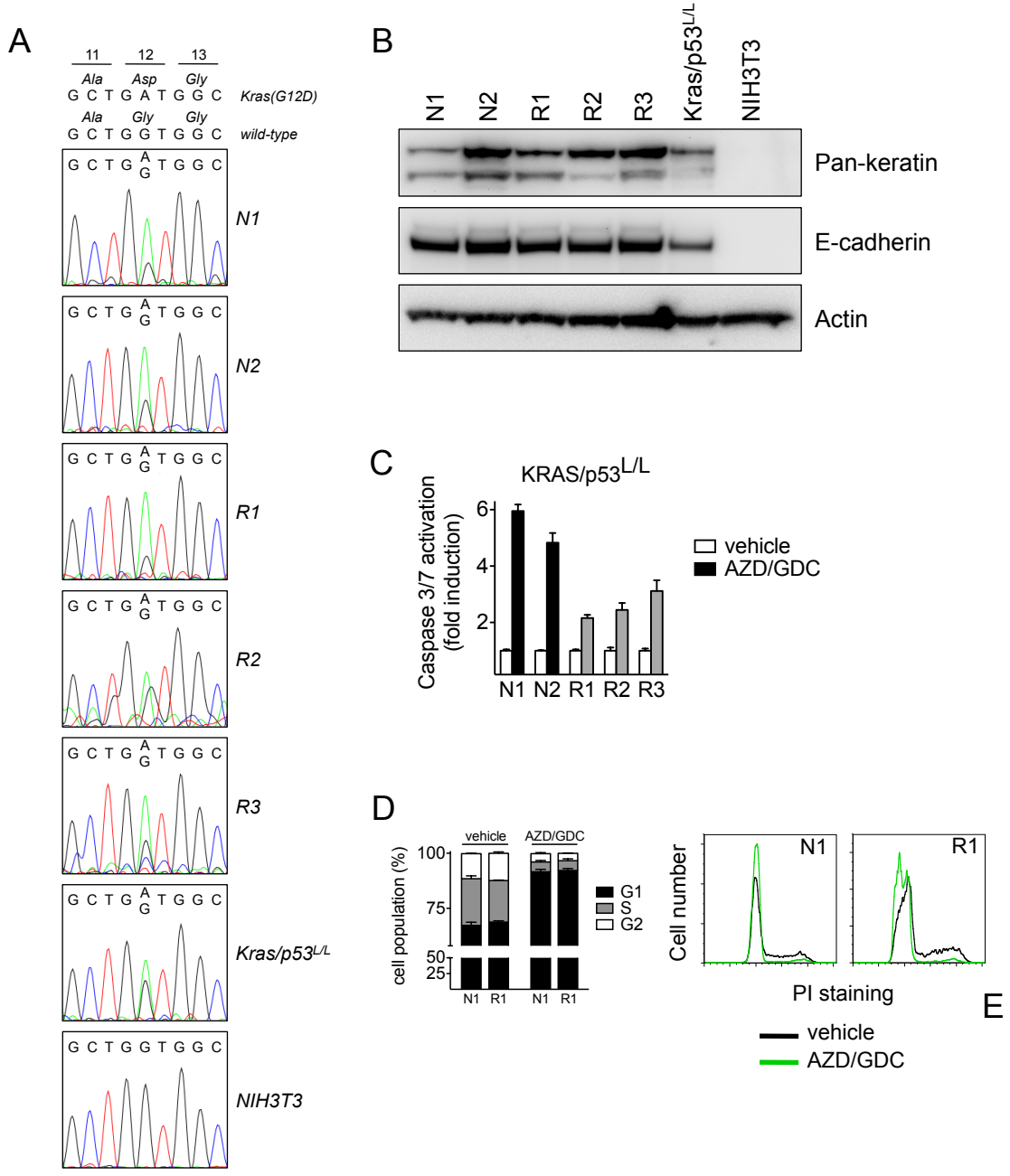
Supplemental Figure 17. In vitro derived MEKi/PI3Ki resistant cells

A. Cell lines were seeded into multiple replicate 96-well plates and treated with 1 μM AZD6244/GDC-0941 for 72 hours (cycle 1). Cell proliferation of replicate plates were determined before and after drug treatment and % change in proliferation was calculated relative to pre-treatment values. For additional replicate plates, drug was removed after 72 hours and surviving cell populations were allowed to expand for 72 hours, followed by repeat drug exposure (cycle 2, etc.). With each consecutive drug exposure ("cycle"), the percent decrease in cell viablity was diminished.

B. Schematic of protocol used to generate resistant A427 and DV-90 cell lines.

C. A427 and A427-R cells were treated with 1 μM AZD6244/GDC-0941 for 24 hours, stained with propidium iodide and cell cycle populations analyzed by flow cytometry. Representative cell cycle flow plots are shown (right), with green indicating cells treated with AZD6244/GDC-0941 and black indicating vehicle treated cells.

D. Resistant cells were treated with 1 μM AZD6244/GDC-0941 for 48 hours and caspase-3/7 activity was determined.



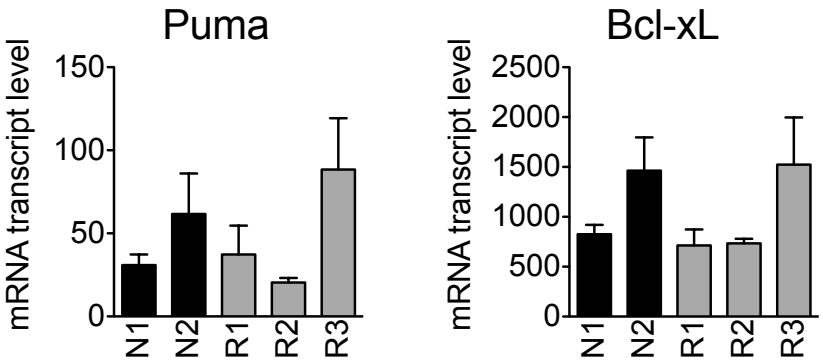
Supplemental Figure 18. Cell lines derived from resistant *Kras p53$^{L/L}$* tumors

A. Cell lines derived from *Kras p53$^{L/L}$* tumors express mutant *Kras (G12D)*. The region encompassing the G12D mutant allele was amplified by PCR from total RNA and the presence of the mutation (c.35G>A) confirmed by Sanger sequencing. *Kras p53$^{L/L}$* untreated tumor nodule and cell lines are heterozygous at this locus, whereas the NIH3T3 mouse fibroblast cell line is wild-type.

B. Cell lines derived from resistant *Kras p53$^{L/L}$* tumors retain epithelial phenotype. Lysates from untreated *Kras p53$^{L/L}$* tumor nodules and resistant cell lines were analyzed for the expression of cytokeratins and E-cadherin by western blotting. In contrast, the mesenchymal mouse fibroblast cell line NIH3T3 does not express these epithelial markers.

C. Cell lines from resistant tumors have reduced apoptotic response to MEKi/PI3Ki. Cells were treated with 1 μM AZD6244/GDC-0941 for 48 hours and caspase-3/7 activation was assessed. Data shown are mean and error of triplicate samples.

D. Cell lines derived from resistant tumors undergo cell cycle arrest in response to MEKi/PI3K. Cells were treated with 1 μM AZD6244/GDC-0941 for 24 hours, stained with propidium iodide and cell cycle populations analyzed by flow cytometry. Representative cell cycle flow plots are shown, with green indicating cells treated with AZD6244/GDC-0941 and black indicating vehicle treated cells.



Supplemental Figure 19. PUMA and BIM mRNA levels do not differ between cell lines derived from treatment naïve and resistant *Kras p53*$^{L/L}$ tumors

PUMA and BCL-XL mRNA expression levels were determined by quantitative RT-PCR. Data shown are mean and error of 3 independent experiments.

Supplemental Table 1. Mutational status of *KRAS* mutant NSCLC cell lines. Mutational status of cell lines as reported by the Sanger Cancer Cell Line Project (http://cancer.sanger.ac.uk/cancergenome/projects/cell_lines/) and the UMD TP53 Mutation Database (http://p53.fr/)

	KRAS	PIK3CA	PTEN	TP53	STK11	SMARCA4	CDKN2A	SMAD4	NF1	NF2	CTNNB1	RB1	MSH6	RUNX1
H460	Q61H	E545K			Q37*		del							
H2030	G12C			G262V	E317*	del								
SW1573	G12C	K111E					del	del		del	S33F			
H1155	Q61H		R233*	R273H									R911*	
H358	G12C			del										
H23	G12C			M246I	W332*	K1566_E1567>N*								
Calu-1	G12C			del										
H1734	G12C			R273L	M51fs							S127I		
SKLU1	G12D			H193R			del							
H441	G12V			R158L										
COR-L23	G12V			del			E26*							S450fs
A549	G12S				Q37*	Q729fs*4	del							
LU99A	G12C	T1025A					del							
H1573	G12A	PIK3R1 del		R248L		E1399*								
SW900	G12V			Q167*			del		del					
LU65	G12C			E11Q			H66R					S82*		
H2009	G12A			R273L								del		
H1792	G12C			intron 672+1G>A[&]										
DV90	G13D													
A427	G12D				del	del	del				T41A			

[&]Alters exon 6/intron 6 splice donor site

Supplemental Table 2. Knockdown efficiency of pLKO shRNA hairpins used in lentiviral screen. A549 cells were infected with 1:5 lentiviral stock, selected with puromycin for 72 hours and lysates collected. Percent knockdown relative to shGFP control was determined by western blotting. Values for % knockdown of RNA are those reported by The RNAi Consortium (Broad Institute).

Gene	TRC identifier	Sequence	% knockdown (western blot)	% knockdown (RNA - Broad)
BAD	TRCN0000033454	GACGAGTTTGTGGACTCCTTT	76	80
BAK	TRCN0000033466	TGGTACGAAGATTCTTCAAAT	87	66
BAX	TRCN0000033471	GCCGGAACTGATCAGAACCAT	70	90
BCL-2	TRCN0000040071	GTGATGAAGTACATCCATTAT	25	85
BCL-W	TRCN0000033507	CAGAAGGGTTATGTCTGTGGA	>95	NR
BCL-XL	TRCN0000033500	GTGGAACTCTATGGGAACAAT	95	99
BID	TRCN0000062709	CTTTCACACAACAGTGAATTT	60	80
BIK	TRCN0000033527	CTTCGATTCTTTGGAATGCAT	53	NR
BIM	TRCN0000001051 TRCN0000001054	ATGGTTATCTTACGACTGTTA AGCCGAAGACCACCCACGAAT	87 62	67 50
BFL-1/A1	TRCN0000033495	GAAGGTATTCTCATCAAGAAA	65	
BMF	TRCN0000130959	CGAAAGCTTCAGTGCATTGCA	49	NR
HRK	TRCN0000033557	GCTAGGCGACGAGCTGCACCA	74	NR
MCL-1	TRCN0000005518	GCTTCGGAAACTGGACATCAA	63	NR
NOXA	TRCN0000151311	GCTTCTGTTCAGATGATCTTT	50	NR
PUMA	TRCN0000033612	GAGGGTCCTGTACAATCTCAT	>95	81

NR = not reported

Supplemental Materials and Methods

Cell line, antibodies, reagents

H460, H2030, H358, H23, Calu-1, H1734, H441, COR-L23, A549, LU-99A, SW900, LU-65, H1792, DV-90 and HCC827 cells were maintained in RPMI supplemented with 5% FBS. SW1573, H1155, SK-LU-1, H1573, H2009 and A427 were maintained in DMEM/F12 with 5% FBS. The N1, N2, R1, R2, R3 cell lines are and were maintained in RPMI with 10% FBS.
For western blotting, the following antibodies were used: BCL-XL, BIM, PUMA, cleaved caspase-3, pan-keratin, pERK1/2 (T202/204), ERK1/2, p-AKT (T308), p-AKT (S473), p-S6 (S240/244), p-S6 (S235/236) (Cell Signaling); BAX (N-20), BAX (6A7), MCL-1, AKT1/2/3, PARP (Santa Cruz); E-cadherin (BD Biosciences). BIM (Hs_BCL2L11_5), BAX (Hs-BAX_10), PUMA (Hs_BBC3_2) siRNA was from Qiagen.

Caspase 3/7 activity assay

Cells were seeded into 96 well plates 24 hours prior to addition of drug. Drugs were added to cells for 48 hours and cell viability and caspase 3/7 activation were determined by Caspase 3/7 activity values were normalized to viability. The 48 hour time point was chosen as the point of optimal caspase-3/7 activation relative to cell viability.

Cell cycle analysis

Cells were seeded 24 hours prior to experiment to give a confluency of 30-50%. Drugs were added for 24 hours and cells harvested, stained with propidium iodide and analyzed by flow

cytometry. Cell cycle sub-populations were calculated using the Dean-Jett model with FloJo software.

BAX activation assay

Cells lysed in CHAPS lysis buffer (20 mM Tris pH 7.5, 137 mM NaCl, 1% CHAPS, 1 mM EDTA, 1 mM EGTA, 5 mM $MgCl_2$) for 10 min on ice, spun for 10 min at 15000 rpm and the supernatant collected. Lysates (500 μg total protein) were incubated with 1 μg Anti-BAX 6A7 antibody or with 1 μg Rabbit IgG in the presence of 30 μl of Protein G-Dynabeads (Invitrogen) overnight at 4° C. Beads were washed 3 times in lysis buffer, eluted with 2X SDS sample buffer and analyzed by western blotting.

Ral-GTP pull down assay

Cells were assayed with the Ral Activation Assay Kit (Millipore) according to the manufacturers instructions. Briefly, cells were washed with PBS, lysed with 1 X Ral Activation Assay Buffer (RAB). Following centrifugation, the supernatant was collected. For positive and negative control pull downs, extracts were treated with GTPgS or GDP, respectively. Extracts were incubated with Ral BP1 agarose beads, washed with RAB and eluted with SDS sample buffer. Elutions were normalized based on lysate protein quantification and the amount of Ral-A was analyzed by western blotting.

Quantitative RT-PCR

Cells were seeded 24 hours prior to give a confluency of 50%. Cells were treated with drugs for 24 hours and RNA was extracted using the RNeasy Kit (Qiagen). For lung tumor nodules from

Kras and Kras p53L/L mice, RNA was extracted using Trizol reagent (Invitrogen) followed by RNeasy Kit. cDNA was prepared from 500 ng total RNA with the First Strand Synthesis Kit (Invitrogen) using oligo-dT primers. Quantitative PCR was performed using FastStart Sybr Green (Roche) on a Lightcycler 480. Sequence of primers used are listed in Supplementary Materials and Methods. mRNA expression relative to actin or GAPDH were calculated using the Delta-Delta threshold cycle (Ct) method as previously described (14). Human primers used: BCL-XL F 5'-agccttggatccaggagaa-3', R 5'-agcggttgaagcgttcct-3'; BIM F 5'-gatccttccagtgggtatttctctt-3', R 5'-actgagatagtggttgaaggcctgg-3'; MCL-1 F 5'-aagccaatgggcaggtct-3', R 5'-tgtccagtttccgaagcat-3'; PUMA F 5'-gacctcaacgcacagtacga-3', R 5'-gagattgtacaggaccctcca-3'; Actin F 5'-ctgtgctatccctgtacgcctc-3', R 5'-catgatggagttgaaggtagtttcgt-3'; GAPDH F 5'-aacagcgacacccatcctc-3', R 5'-cataccaggaaatgagcttgacaa-3'; DUSP6 F 5'-cgactggaacgagaatacgg-3', R 5'- ttggaacttactgaagccacct-3'; SPRY4 F 5'- ccccggcttcaggattta-3', R 5'-ctgcaaaccgctcaatacag-3'; EGR-1 F 5'-agccctacgagcacctgac-3', R 5'- ggtttggctggggtaactg-3'; TRAIL F 5'-cctcagagagtagcagctcaca-3', R 5'-cagagccttttcattcttgga-3'; HER3 F 5'-ctgatcaccggcctcaat-3', R 5'-ggaagacattgagcttctctgg-3'. Mouse primers used: BIM F 5'-ggagacgagttcaacgaaactt-3', R 5'-aacagttgtaagataaccatttgagg-3'; BCL-XL F 5'-tgaccacctagagccttgga-3', R 5'-tgttcccgtagagatccacaa-3'; Actin F 5'-ctaaggccaaccgtgaaaag-3', R 5'-accagaggcatacagggaca-3'.

Cleaved caspase-3 IHC

Formalin fixed paraffin embedded xenograft tumors were sectioned. Slides were de-paraffinized, re-hydrated through xylene and alcohol washes. Antigen retrieval was performed. Slides were blocked in goat serum and Avidin D. Slides were incubated with cleaved caspase-3 antibody

(Cell Signaling) overnight at 4^{o}C. Slides were blocked with biotin, incubated with rabbit biotinylated secondary antibody for 1 hour, washed in PBS, incubated with an Avidin D, horseradish peroxidase, and developed with DAB coloring reagent. Slides were counter-stained in hemotoxylin.

Supplemental Table 1. Mutational status of *KRAS* mutant NSCLC cell lines. Mutational status of cell lines as reported by the Sanger Cancer Cell Line Project (http://cancer.sanger.ac.uk/cancergenome/projects/cell_lines/) and the UMD TP53 Mutation Database (http://p53.fr/)

	KRAS	PIK3CA	PTEN	TP53	STK11	SMARCA4	CDKN2A	SMAD4	NF1	NF2	CTNNB1	RB1	MSH6	RUNX1
H460	Q61H	E545K			Q37*		del							
H2030	G12C			G262V	E317*	del								
SW1573	G12C	K111E					del	del		del	S33F			
H1155	Q61H		R233*	R273H									R911*	
H358	G12C			del										
H23	G12C			M246I	W332*	K1566_E1567>N*								
Calu-1	G12C			del										
H1734	G12C			R273L	M51fs							S127I		
SKLU1	G12D			H193R			del							
H441	G12V			R158L										
COR-L23	G12V			del			E26*							S450fs
A549	G12S				Q37*	Q729fs*4	del							
LU99A	G12C	T1025A					del							
H1573	G12A	PIK3R1 del		R248L		E1399*								
SW900	G12V			Q167*			del		del					
LU65	G12C			E11Q			H66R					S82*		
H2009	G12A			R273L								del		
H1792	G12C			intron 672+1G>A[&]										
DV90	G13D													
A427	G12D				del	del	del				T41A			

[&]Alters exon 6/intron 6 splice donor site

Supplemental Table 2. Knockdown efficiency of pLKO shRNA hairpins used in lentiviral screen. A549 cells were infected with 1:5 lentiviral stock, selected with puromycin for 72 hours and lysates collected. Percent knockdown relative to shGFP control was determined by western blotting. Values for % knockdown of RNA are those reported by The RNAi Consortium (Broad Institute).

Gene	TRC identifier	Sequence	% knockdown (western blot)	% knockdown (RNA - Broad)
BAD	TRCN0000033454	GACGAGTTTGTGGACTCCTTT	76	80
BAK	TRCN0000033466	TGGTACGAAGATTCTTCAAAT	87	66
BAX	TRCN0000033471	GCCGGAACTGATCAGAACCAT	70	90
BCL-2	TRCN0000040071	GTGATGAAGTACATCCATTAT	25	85
BCL-W	TRCN0000033507	CAGAAGGGTTATGTCTGTGGA	>95	NR
BCL-XL	TRCN0000033500	GTGGAACTCTATGGGAACAAT	95	99
BID	TRCN0000062709	CTTTCACACAACAGTGAATTT	60	80
BIK	TRCN0000033527	CTTCGATTCTTTGGAATGCAT	53	NR
BIM	TRCN0000001051 TRCN0000001054	ATGGTTATCTTACGACTGTTA AGCCGAAGACCACCCACGAAT	87 62	67 50
BFL-1/A1	TRCN0000033495	GAAGGTATTCTCATCAAGAAA	65	
BMF	TRCN0000130959	CGAAAGCTTCAGTGCATTGCA	49	NR
HRK	TRCN0000033557	GCTAGGCGACGAGCTGCACCA	74	NR
MCL-1	TRCN0000005518	GCTTCGGAAACTGGACATCAA	63	NR
NOXA	TRCN0000151311	GCTTCTGTTCAGATGATCTTT	50	NR
PUMA	TRCN0000033612	GAGGGTCCTGTACAATCTCAT	>95	81

NR = not reported