

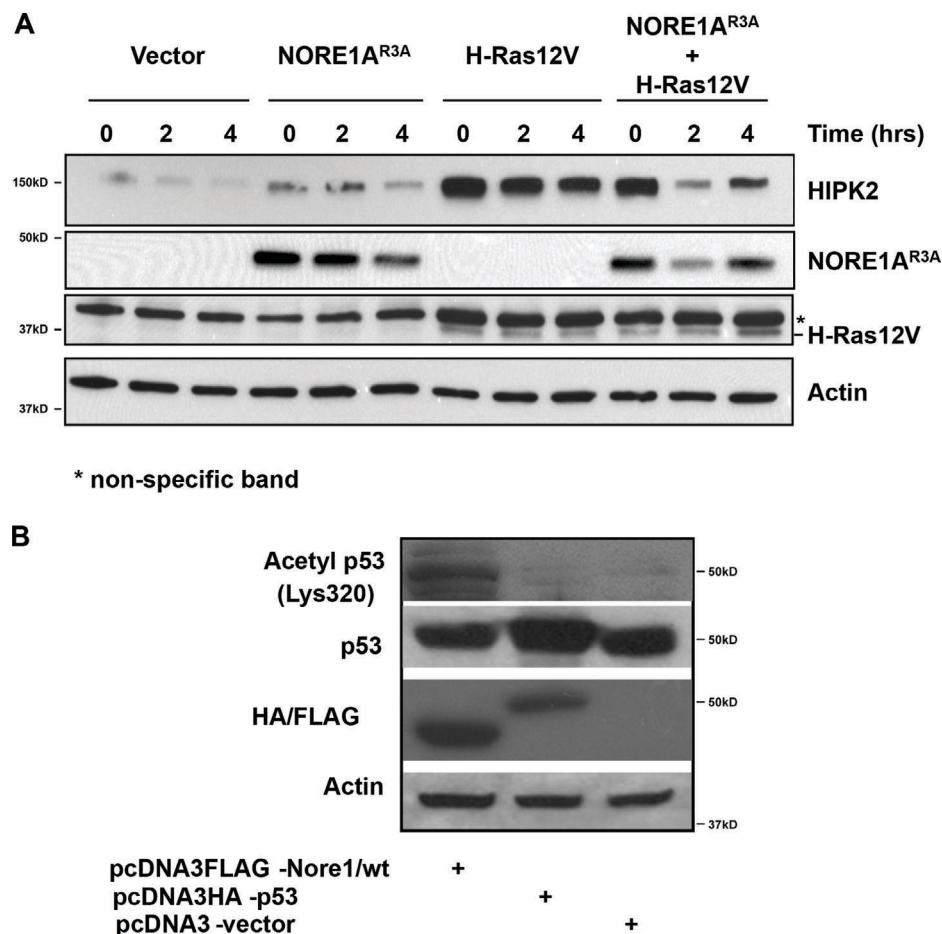
Donninger et al., <http://www.jcb.org/cgi/content/full/jcb.201408087/DC1>

Figure S1. NORE1A^{R3A} fails to stabilize HIPK2. (a) HEK-293 cells were transfected with HIPK2, NORE1A and activated H-Ras expression constructs. The cells were treated with cyclohexamide and lysed at the indicated time points. Levels of protein were determined by Western blot analysis. (b) HEK293 cells were transiently transfected with vector, NORE1A or p53 expression plasmids. The cell lysates were then Western blotted for the total levels of each protein and the degree of p53 acetylation at residue K320 using a specific antibody (Cell Signaling Technology). In the presence of NORE1A, endogenous p53 exhibited enhanced K320 acetylation.

Table S1. Description of cell lines used

CELL LINE	RAS Status	P53 status	HIPK2 status	NORE1A Status	REMARKS
A549 human lung tumor cell line	mutant	WT	+	-	Can be induced to undergo senescence by NORE1A transfection.
MEFs	WT	WT	+	+	Primary murine cells with senescence machinery intact
HBEC-3KT Human lung	WT	WT	+	+	Human lung epithelial cells immortalized without the use of viral oncogenes.
HEK-293 cells Human kidney	WT	WT	+	+	Human embryonic kidney cells. Excellent cell line for transient transfection experiments due to ease of transfection and relatively high levels of expression.
HEK-293T Human kidney	WT	inhibited	+	+	As above but expressing SV40 LT which compromises p53. Higher levels of expression from transiently transfected plasmids.
COS-7 Monkey kidney	WT	WT	?	?	Express high levels of protein from transiently transfected plasmids. Possess large, flat nuclei, ideal for visualizing fluorescent nuclear proteins.
HuH6 Human hepatocellular carcinoma	WT (MET*)	WT	+	+	Express both NORE1A and HIPK2. Constitutively phosphorylated MET receptor may activate the WT Ras pathway.
HepG2 Human hepatocellular carcinoma	mutant	WT	+	+	Contain mutant Ras, express both NORE1A and HIPK2. Can be used for endogenous co-IP.

Table S2. The clinicopathological features of HCC patient samples

Variables	Features	
	HCCB ^a	HCCP ^b
No. of patients	20	20
Male	15	16
Female	5	4
Age (mean ± SD)	71.8 ± 8.9	73.4 ± 10.0
Etiology		
HBV	9	10
HCV	7	6
Ethanol	4	4
Cirrhosis		
+	16	15
-	4	5
Tumor size		
>5 cm	11	14
<5 cm	9	6
Edmondson and Steiner grade		
II	8	3
III	5	8
IV	7	9
Alpha-fetoprotein secretion		
>300 ng/ml of serum	12	10
<300 ng/ml of serum	8	10

^aHCCB, HCC with better outcome/longer survival (survival >3 yr after partial liver resection)^bHCCP, HCC with poorer outcome/shorter survival (survival <3 yr after partial liver resection)