Table S1. Patient information

Patient	Diagnosis	Mutations/cytogenetics	p53 staining	
1	Diamond Blackfan anemia	No RPS19 mutation	Weak	
2	Diamond Blackfan anemia	No testing done	Weak	
3 (Panel C)	Diamond Blackfan anemia	No testing done	Strong	
4	Diamond Blackfan anemia	RPS19 splice site mutation	Weak	
5	Diamond Blackfan anemia	No mutation in RPS19, RPS24,	Weak	
		RPL5, RPL9, RPL11, RPL14,		
		RPL35a, RPL36, RPS7, RPS10,		
		RPS15, RPS17, RPS26, RPS27A		
6 (Panel D)	Diamond Blackfan anemia	No RPS19 mutation	Strong	
7	Diamond Blackfan anemia	No testing done Weak		
8	Diamond Blackfan anemia	No testing done Weak		
9 (Panel E)	MDS with del(5q)	46,XY,del(5)(q22q34)[2]/46,XY[18] Strong, focal		
10 (Panel F)	MDS with del(5q)	46,XX,del(5q)(q13q33)[5]/45,sl,dic(17 Strong		
		:20)(p11.2;q11.2)[9]/45,sdl1,t(4:15)(q		
		25;q21[3]/46,XX,[3]		
11	MDS with del(5q)	46,XY,del(5)(q15q31)[17]/46,XY[3] Negative		
12	MDS with del(5q)	46,XY,del(5)(q12q35)[6]/46,XY[cp6] Negative		

Table S2. shRNA sequences

Gene Name	Gene symbol	RefSeqID		shRNA sequence
RPS14	RPS14	NM_005617	shRNA #1	5'-CCGAGATGAATCCTCACCATA-3'
			shRNA #2	5'-GCTATGTTGGCTGCCCAGGAT-3'
RPS19	RPS19	NM_001022	shRNA #1	5'-CTACGATGAGAACTGGTTCTA-3'
			shRNA #2	5'-GCTTGCTCCCTACGATGAGAA-3'

Table S3. Primer sequences

Gene Name	Gene symbol	RefSeqID	Primer sequences	
RPS14	RPS14	NM_005617	Forward	5'CTCAGGTGGCTGAAGGAGAG3'
			Reverse	5'GCAGCCAACATAGCAGCATA3'
RPS19	RPS19	NM_001022	Forward	5'AGACGTGAACCAGCAGGAGT3'
			Reverse	5'TTCTCTGACGTCCCCATAG3'
<i>p</i> 53	TP53	NM_000546	Forward	5'GTTCCGAGAGCTGAATGAGG3'
			Reverse	5'TCTGAGTCAGGCCCTTCTGT3'



Figure S1. Efficacy of *RPS14* and *RPS19* shRNAs. Quantitative RT-PCR and western blots for the expression of RPS14 and RPS19 gene and protein after their knockdown using indicated shRNAs. RT-PCR was performed in primary human CD34+ bone marrow cells (CD34+) and the westerns were performed in HEL (human erythroleukemia) cells. * denotes p < .05, and ** denotes p < .01.



Figure S2. Efficacy of *RPS14* **and** *RPS19* **shRNAs.** Quantitative RT-PCR for the expression of the indicated genes was performed in primary human CD34+ bone marrow cells (CD34+). * denotes p < .05, and ** denotes p < .01.

В

Α



Figure S3. *RPS14* and *RPS19* shRNAs increase apoptosis of primary human bone marrow CD34+ cells. Apoptotic cells were analyzed by flow cytometry after annexin V staining. * denotes p < .05, and ** denotes p < .01. Results shown for each experiment are representative of three independent experiments performed in triplicate (mean ± SEM);



Figure S4. Activation of p53 in erythroid cells. (A) Protein levels for p53 were analyzed by intracellular flow cytometry in cells expressing control (*luciferase*), *RPS14*, or *RPS19* shRNAs. Erythroid cell stage was determined by staining for the erythroid markers CD71 and GlyA. Expression of CD71 precedes expression of GlyA. (B) Protein levels for p53 by intracellular flow cytometry in cells expressing control (*luciferase*), *RPS19* shRNAs. Early myeloid cell stages were determined by staining for the myeloid markers CD33, CD13 and CD45. * denotes p < .05, and ** denotes p < .01.



IP: HDM2



Figure S5. Pa rtial knockdown of *RPS14* **using shRNA#2 leads to binding of RPL11 to HDM2**, consistent with the results of RPS14 shRNA #1 shown in Figure 3. Immunoprecipitation from A549 cell lysates were performed using anti-HDM2 or normal rabbit IgG antibodies. Western blots show the levels of HDM2 and RPL11 in the immunoprecipitates.



Figure S6. Pif ithrin-*α* **treatment of CD34+ cells rescues the apoptotic phenotype.** After infection of CD34+ cells with either control shRNA against *luciferase* gene or against *RPS14* and *RPS19*, cells were treated with different concentrations of PFT-a (a p53 inhibitor). After 96 hours, apoptotic cells were assessed by flow cytometry using annexin V antibody. Results shown for each experiment are representative of three independent experiments performed in triplicate (mean ± SEM);