

Pat.	FAB	Age	Sex	Sample	Cytogenetics	Molecular genetics	Outcome
1	M1	77	M	PBMC	C.K.	N.A.	Dead
2	M2	62	F	PBMC	N.K.	FLT3-ITD+ NPM1+	Chemo A&W 108 mo.
3	M2	42	F	PBMC	N.K.	FLT3-ITD+ NPM1+	Allo-SCT A&W 15 mo.
4	M2	53	M	PBMC	N.K.	N.D.	Allo-SCT Relapse 2 mo.
5	M4	61	M	PBMC	Inv (16)	N.D.	Chemo A&W 24 mo.
6	M4	23	M	PBMC	t(16;16), -21q	N.D.	Allo-SCT A&W 15 mo.
7	M4	77	M	PBMC	N.K.	FLT3-ITD+	Dead
8	M4	60	F	PBMC	N.K.	NPM1+	Chemo A&W 23 mo.
9	M4	48	F	PBMC	N.K.	NPM1+	Chemo Dead 1 mo.
10	M5	67	F	PBMC	46,XX,t(9;11) (p21;q23)	N.D.	Relapse
11	M1	67	M	BM	N.K.	N.A.	Dead 3 mo.
12	M1	21	F	BM	46,XX,i(17q)	N.A.	Allo-SCT CR
13	M1	63	F	BM	C.K.	N.A.	Not known
14	M1	69	F	BM	C.K.	N.A.	Not known
15	M1	63	F	BM	N.K.	N.A.	Dead 3 mo.
16	M2	65	M	BM	N.K.	N.A.	Dead 2 mo.
17	M2	66	M	BM	N.A.	N.A.	Not known
18	M2	65	M	BM	N.K.	CEBPA+	Dead
19	M2	39	M	BM	46,XX del7, t(8;21)	N.D.	Allo-SCT Dead (GvHD)
20	M2	75	M	BM	N.K.	N.D.	Dead 1 mo.
21	M4	73	M	BM	C.K.	N.A.	Dead 3 mo.
22	M4	64	M	BM	N.K.	N.A.	C.R.
23	M4	69	M	BM	N.K.	N.A.	Chemo A&W 60 mo.
24	M4	77	F	BM		N.D.	Dead
25	M4	77	F	BM	N.K.	FLT3-ITD+ NPM1+	Dead 1 mo.
26	M5b	34	F	BM	N.K.	N.A.	Dead 3 mo.

Supplemental Table 1. Patient Characteristics. N.A. -Not Available; C.K. -Complex Karyotype; N.K. -Normal Karyotype; N.D. -None Detected; A&W -Alive and Well; C.R. -Complete Remission; GvHD -Graft versus Host Disease.

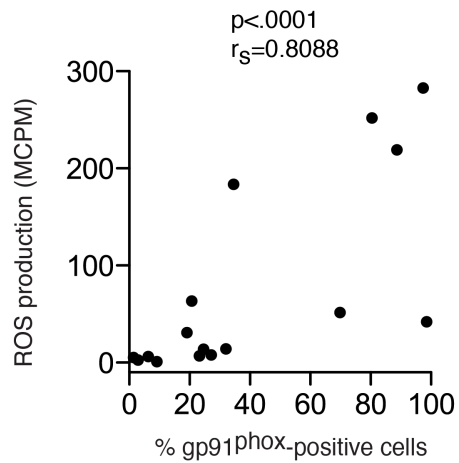


Figure S1. Correlation of gp91^{phox}/NADPH oxidase expressing cells and ROS production in leukemic BM. Unsorted BM cells (*Patients 11-26*) recovered at diagnosis from untreated patients with FAB-M1 AML (n=5), FAB-M2 AML (n=5), and FAB-M4/M5 AML (n=6) were stimulated with PMA and assayed for ROS production (ordinate) and frequency (%) of cells expressing gp91^{phox} (abscissa).

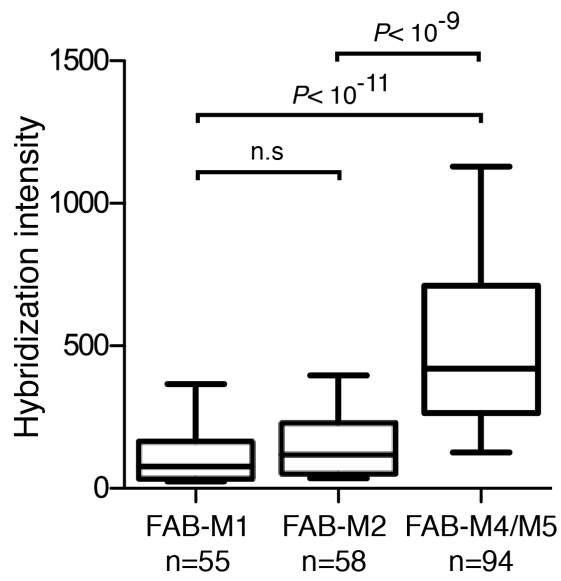


Figure S2. Gp91^{phox} mRNA levels in untreated AML patients. Analysis of microarray data from 207 patients with untreated AML.

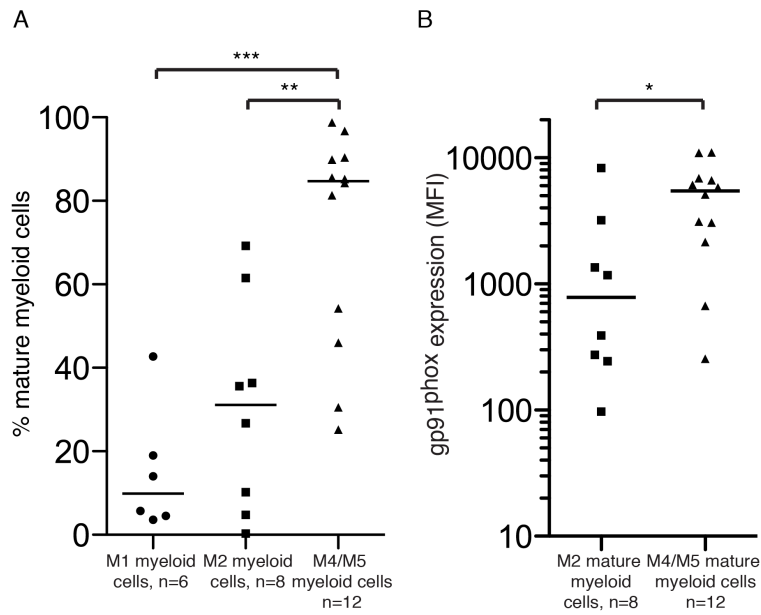


Figure S3. Expression of gp91^{phox} by mature cells in FAB-M1, FAB-M2, and FAB-M4/M5 AML.

Data points indicated % mature myeloid cells (defined as CD33⁺ with expression of CD14⁺ and/or CD15⁺ and analyzed by FACS) in peripheral blood or BM of untreated patients from AML patients with indicated FAB classes; left). The right part of the figure shows the median fluorescence intensity of gp91^{phox} in similarly gated mature cells from BM or peripheral blood of the patients with FAB-M2 or FAB-M4/M5 AML.

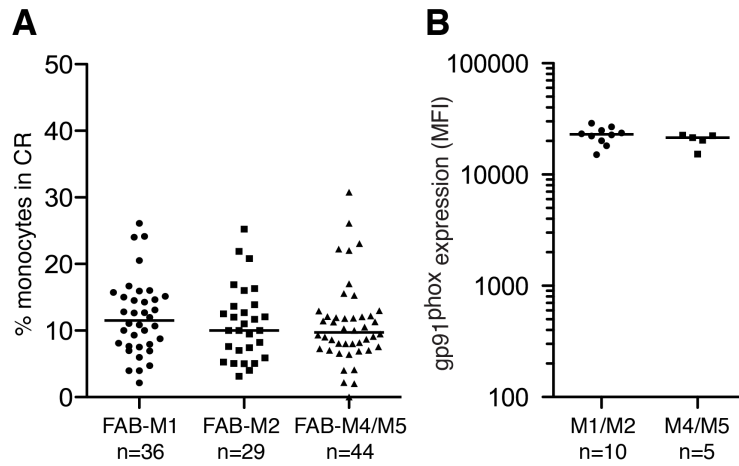


Figure S4. No differences between monocytes in AML patients in CR. (A) Blood counts from 109 patients in CR were analyzed in regard of proportions of circulating monocytes. (B) Analysis of gp91^{phox} expression on CD14⁺ monocytes from 15 patients in CR1 revealed no statistical differences.

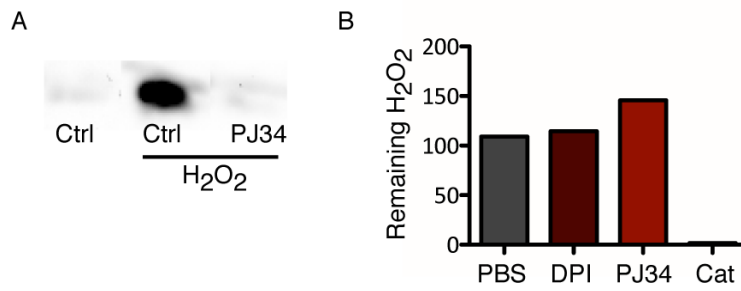


Figure S5. PJ34 inhibit PAR formation in lymphocytes. (A) PAR detection by western blot. Lymphocytes where exposed to hydrogen peroxide (500 μ M) and PAR formation analyzed by western blot after 20 min in the presence or absence of PARP-1 inhibitor PJ34. (B) Remaining H₂O₂ was measured after incubation with PBS, DPI (3 μ M), PJ34 (0.5 μ M) or catalase (200U/ml). Only catalase hade H₂O₂ scavenging properties.