SUPPLEMENTAL MATERIALS

Table S1. CLL Patient Demographics and Clinical Characteristics (n=35)

CLL Patient	Sex	Age	Months Since Diagnosis	Prior Treatment	CD38	ZAP70	ABT-737 Sensitivity*	Vinblastine Sensitivity**
11	F	48	4	No	-	-	very res	res
13	F	55	unknown	No	-	-	very res	res
26	M	53	84	No	-	-	very res	res
1	M	67	13	No	-	-	very res	ND
2	F	60	120	No	ND	ND	very res	ND
7	F	62	48	No	-	-	res	res
10	F	85	144	No	+	ND	res	res
12	M	67	0	No	+	+	res	res
14	F	72	48	Yes	-	-	res	res
16	M	66	120	Yes	-	-	res	res
17	M	57	0	No	+	-	res	res
42	M	86	24	No	-	-	res	res
47	M	40	0	No	-	-	res	res
23	M	67	12	No	+	+	res	sens
18	M	68	120	Yes	-	-	sens	res
20	F	63	72	No	-	ND	sens	res
27	M	76	36	No	-	+	sens	res
32	M	83	42	No	+	-	sens	res
40	F	64	180	No	-	-	sens	res
48	F	76	168	No	ND	ND	sens	res
49	M	69	48	No	-	-	sens	res
50	F	83	168	No	ND	ND	sens	res
8	M	52	60	No	-	-	sens	sens
9	F	71	0	No	-	-	sens	sens
19	F	59	1	No	+	+	sens	sens
25	F	69	36	No	-	-	sens	sens
31	M	67	unknown	No	+	+	sens	sens
33	F	65	36	No	+/-	+	sens	sens
39	M	49	15	No	-	+	sens	sens
44	F	76	12	No	-	+	sens	sens
45	M	80	168	Yes	ND	ND	sens	sens
3	M	51	44	No	-	-	sens	ND
4	F	82	156	No	+	+	sens	ND
5	F	67	108	No	+	-	sens	ND
6	F	63	84	Yes	ND	ND	sens	ND

* ABT-737 sensitivity: very resistant = >50% survival at 0.01 μM and >40% survival at 0.1 μM; resistant = >50% survival at 0.01 μM; sensitive = ≤50% survival at 0.01 μM (see Figure 1A).
** Vinblastine sensitivity: resistant = >50% survival at 1 μM; sensitive = ≤50% survival at 1 μM (see Figure 1 B).
ND = not determined.

FIGURE LEGEND

Figure S1. JNK is not required for apoptosis induced by the combination of vinblastine plus ABT-737. NB4 cells were incubated with 10 μM JNK Inhibitor VIII for 1 h prior to the addition of 0.1 μM ABT-737, 0.1 μM dinaciclib and/or 2 μM vinblastine for 6 h. Cell lysates were harvested for protein expression. Dinaciclib plus vinblastine was used as a positive control for JNK-dependent apoptosis as previously published (2). Dinaciclib, through inhibition of CDK9, prevents transcription thereby preventing expression of Mcl-1. The lack of P-c-Jun in dinaciclib-treated cells is due to concomitant inhibition of transcription of c-Jun.