

LEUKEMIA p53 HOTSPOUT MUTATION CELL PANEL

p53 is a tumor suppressor protein encoded by the TP53 gene that responds to DNA damage by regulating cell-cycle arrest, apoptosis and senescence. The Leukemia p53 Hotspot Mutation Cell Panel (ATCC® No. TCP-2070™) is composed of six select suspension cell lines derived from individuals with leukemia. The mutations have been sequenced and validated by ATCC. This panel includes p53 wild type cell lines as well as p53 mutant lines that carry hotspot mutations in one of the following codons: 175, 248 and 273. The panel is useful for anti-cancer drug targeting or reactivation of mutant p53, as well as studies related to p53 molecular mechanisms.

ATCC® No.	Name	Primary Site, Tissue	Histology	TP53 status	Zygoty	Gene mutation*	Protein Sequence*
TIB-190™	CESS	blood	acute myeloid leukemia (AML)	WT	-	-	-
CCL-240™	HL-60	blood	acute promyelocytic leukemia (APL)	NULL	homozygous	c.(del)	-
CCL-119™	CCRF-CEM	blood	acute lymphoblastic leukemia (ALL)	MUT	heterozygous	c.524G>A; c.743G>A	p.R175H; p.R248Q
CRL-2265™	CEM/C1	blood	acute lymphoblastic leukemia (ALL)	MUT	heterozygous	c.524G>A	p.R175H
CRL-2724™	KASUMI-1	blood	acute myeloid leukemia (AML)	MUT	homozygous	c.743G>A	p.R248Q
CRL-1621™	ARH-77	blood	plasma cell leukemia, carry EBV	MUT	homozygous	c.818G>A	p.R273H

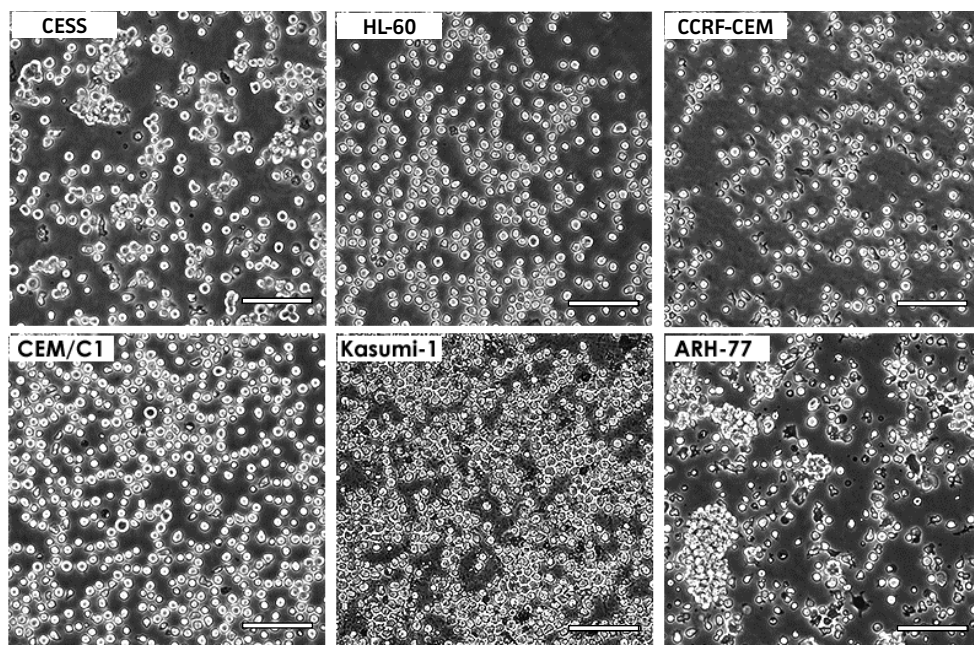


Figure 1. Cell morphology of the six cell lines in the Leukemia p53 Hotspot Mutation Cell Panel. One p53 wild-type lymphoma cell line, one p53 null cell line, and four p53 hotspot mutation leukemia cell lines were maintained in ATCC recommended culture conditions. Cell morphology was observed using a Nikon™ microscope, and images of the indicated cell lines were captured with an Olympus® digital camera.

Scale Bar = 100µm

*For a description of the sequence variation nomenclature please refer to: *den Dunnen JT and Antonarakis SE (2000), Hum. Mutat. 15:7-12.*

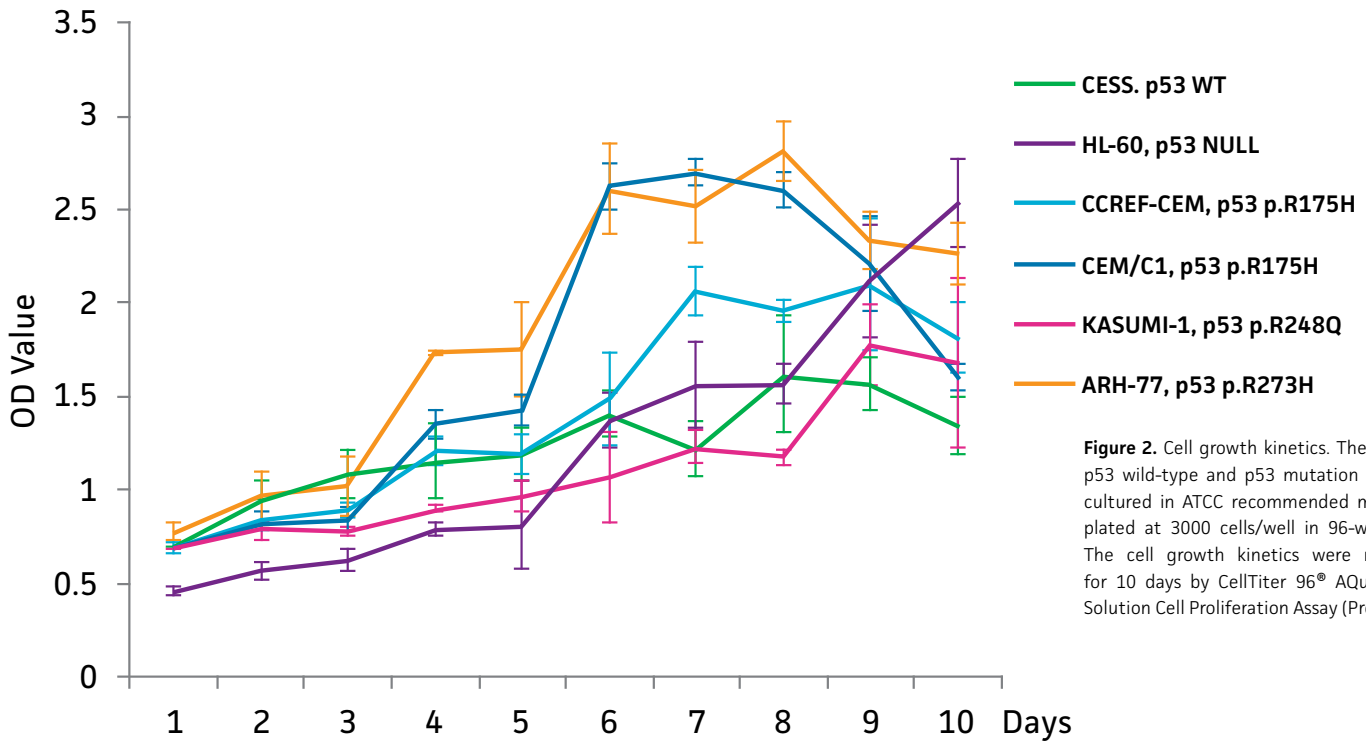


Figure 2. Cell growth kinetics. The indicated p53 wild-type and p53 mutation cells were cultured in ATCC recommended media, and plated at 3000 cells/well in 96-well plates. The cell growth kinetics were monitored for 10 days by CellTiter 96® AQueous One Solution Cell Proliferation Assay (Promega).

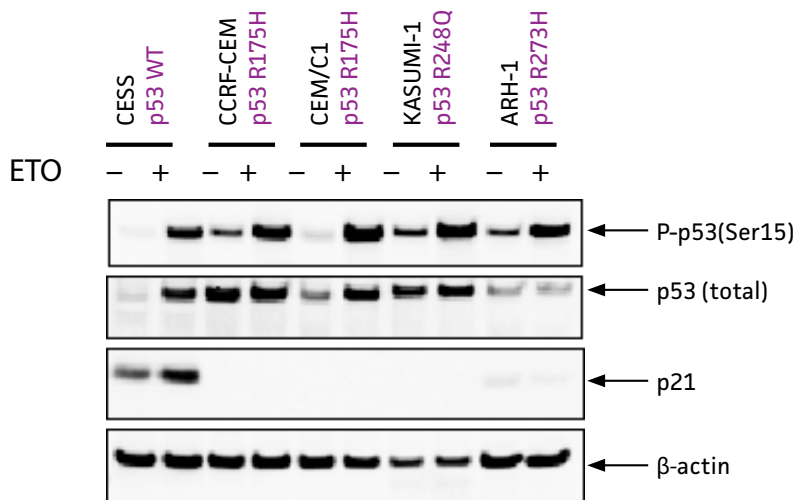


Figure 3. The indicated p53 wild-type and p53 mutation cells were treated with 20 μM etoposide (ETO) for 8 hours to induce DNA damage, or treated with DMSO as a control. Western blotting was used to examine phosphorylation of p53 at Serine 15, total protein expression of p53, and expression of p21, a downstream target of p53. β-actin protein was also examined as a control.

Testing performed for each ATCC cell line was completed on current (2012) distribution material. ATCC provides these data in good faith, but makes no warranty, express or implied, nor assumes any legal liability or responsibility for any purpose for which the data are used.

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