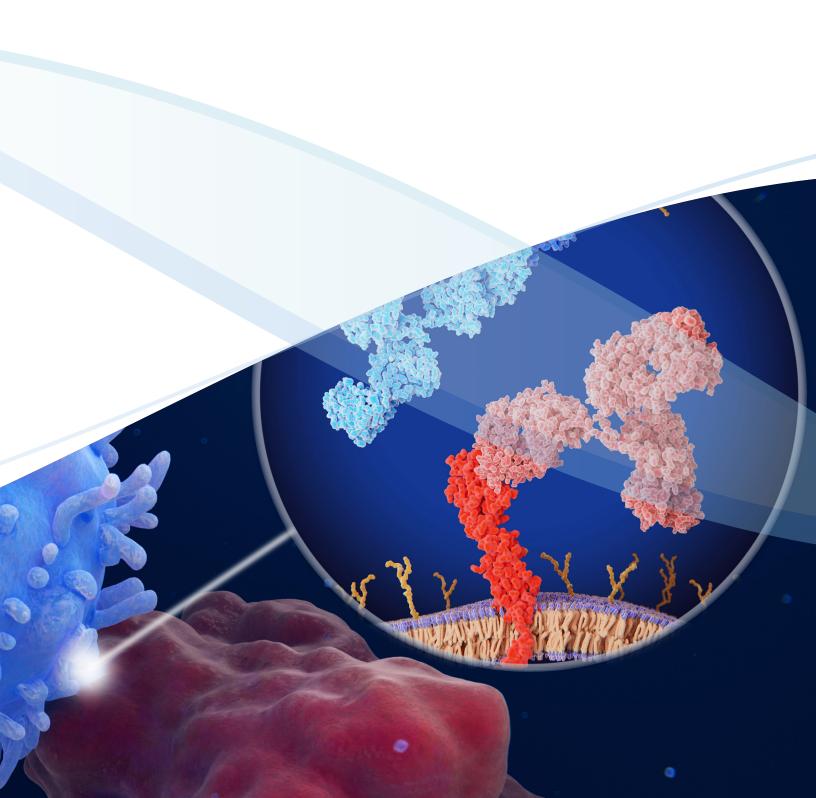


Immuno-oncology Capabilities



Immuno-oncology Tools

Cancer immunotherapy has emerged as an exciting new approach for cancer treatment, and immuno-oncology is one of the fastest growing fields in oncology. As compared to traditional cancer therapies that act directly on cancerous tumors, immuno-oncology therapy offers a unique approach that uses the body's immune system to selectively target and eradicate tumor cells. These therapies also provide long-lasting memory to the immune system, enabling it to continue fighting against cancer cells even after remission.

The development of immunomodulatory drugs and biologics dictates a clear need for human cell-based models to evaluate immune activation. To answer this need, ATCC provides a large collection of fully characterized and authenticated cell lines, human primary cells, and advanced cell models.



Page 2 Order online at www.atcc.org, call 800.638.6597, 703.365.2700, or contact your local distributor.

PRIMARY HUMAN IMMUNE CELLS

PHYSIOLOGICALLY RELEVANT MODELS OF THE IMMUNE SYSTEM

ATCC primary immunology cells are able to support complex, physiologically relevant research projects, including toxicity screening, transplantation and graft rejection, inflammation and allergy, vaccine, drug development, as well as cancer immunology studies. Our Scientists have conducted in-depth characterization of the cells in this collection. Furthermore, this collection reliably provides:

- Greater than 90% cryo-recovery
- Functional data available
- High differentiation capacity or immune activity
- Greater than 90% purity for select biomarkers
- Expansion and differentiation protocols
- Diverse pool of donors available
- Positive and negative biomarkers
- Normal cell morphology

The multipotent bone marrow and cord blood CD34+ hematopoietic stem cells within this collection give rise to either more stem cells or to common myeloid or lymphoid progenitor cells. These cells then give rise to the more differentiated components of the immune system, which may then migrate to the tissues for further specialization. Moreover, the peripheral CD14+ cells in this collection can be induced to differentiate into dendritic cells or macrophages. Finally, the mononuclear cell preparations from the bone marrow or peripheral blood include differentiated macrophages, dendritic cells, monocytes, and lymphocytes, as well as a smaller fraction of hematopoietic cells (Figure 1).

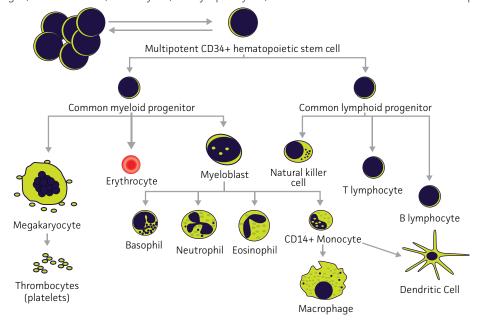


Figure 1: Differentiation of Multipotent Hematopoietic Progenitor Cells

CUSTOMIZABLE FOR ANY EXPERIMENT

The cells in this product listing have many donor options. ATCC has access to a wide range of unique donors, presenting immunologists the ability to design almost any experiment.

- Height and weight
- Age
- Ethnic and gender
- Lifestyle

- HLA and blood type
- Diet
- Family history
- Other specific parameters

Table 1: ATCC primary immune cells

Cell Type	ATCC® No.	Number of Cells/vial	Positive Biomarkers
Peripheral Blood CD14+ Monocytes	PCS-800-010 [™]	50 million	CD14, CD45
Peripheral Blood Mononuclear Cells	PCS-800-011™	25 million	CD45; Lot Specific FIO*: CD3, CD8, CD4, CD56, CD14, CD19
Bone Marrow CD34+ Cells	PCS-800-012 [™]	500,000	CD34, CD45
Bone Marrow Mononuclear Cells	PCS-800-013 [™]	25 million	CD45; Lot Specific FIO*: CD3, CD8, CD4, CD58, CD14, CD19, CD34
Cord Blood CD34+ Cells	PCS-800-014 [™]	500,000	CD34, CD45
Peripheral Blood CD4+ Helper T Cells	PCS-800-016 [™]	25 million	CD3, CD4, CD45

Page 3

Cell Type	ATCC [®] No.	Number of Cells/vial	Positive Biomarkers
Peripheral Blood CD8+ Cytotoxic T Cells	PCS-800-017 [™]	25 million	CD3, CD8, CD45
Peripheral Blood CD19+ B Cells	<u>PCS-800-018</u> ™	25 million	CD20, CD45
Peripheral Blood CD56+ Natural Killer Cells	PCS-800-019 [™]	5 million	CD45, CD56
iPSC-derived Mesenchymal Stem Cells	ACS-7010 [™]	2.5 million	CD29, CD44, CD73, CD90, CD105, CD166
iPSC-derived CD34+ Cells	<u>ACS-7020</u> ™	2.5 million	CD34, CD45
iPSC-derived Monocytes	<u>ACS-7030</u> ™	2.5 million	CD14

^{*}For information only (FIO); Lot-specific FIO is not release criteria. Check individual lots for CD-specific numbers.

REFERENCES

- 1. Clinton J, Shapiro B, Differentiation and expansion of hematopoietic precursor cells from bone marrow-derived CD34+ progenitors. Application Note, 2015.
- 2. Clinton J, Shapiro B. In vitro differentiation of macrophages and dendritic cells from primary human CD14+ monocytes. Application Note, 2015.

EXPLORE MORE AT WWW.ATCC.ORG/PRIMARYIMMUNE

CHECKPOINT LUCIFERASE REPORTER CELLS

Immune checkpoint inhibitors have been successful in treating lung, liver, breast, renal, and skin cancers. However, the complexity of immunological models and variable drug responses among different cancer types pose significant challenges in immuno-oncology. To facilitate large scale drug discovery, ATCC created tumor and immune cell lines with high endogenous expression of checkpoint inhibitory and co-stimulatory expression levels. These cell lines contain gamma interferon activation site (GAS)-response element or nuclear factor of activated T cells (NFAT)-response element upstream of the luciferase gene, which can be used to track candidate blocker efficacy. The portfolio includes clinically relevant targets such as PDL1/2, B7-H3, PD1, and CTLA-4, and can be incorporated into simple blocking assays or sophisticated co-culture cell-based drug screening assays.

Table 2: ATCC Checkpoint Luciferase Reporter Cells

Designation	ATCC [®] No.	Disease	Biomarker	Tissue of origin	Status
HCC827-GAS-Luc2	CRL-2868-GAS-LUC2™	Adenocarcinoma	PD-L1	Lung	Available
MG-63-GAS-Luc2	CRL-1427-GAS-LUC2™	Osteosarcoma	CD-155	Bone	Available
NCI-H1650-GAS-Luc2	CRL-5883-GAS-LUC2™	Adenocarcinoma	B7-H3	Lung	Available
SUP-T1 [VB]-NFAT-Luc2	CRL-1942-NFAT-LUC2™	Lymphoblastic Lymphoma	PD-1	Pleural effusion	Available
U-937 NFkB-Luc2	CRL-1593.2-NFkB-LUC2™	Histiocytic Lymphoma	SIRPA	Pleural effusion	Available
KG-1 NFkB-Luc2	CCL-246-NFkB-LUC2™	Acute myelogenous leukemia	SIGLEC10	Bone; Marrow	Available
HMC3 NFkB-Luc2	CRL-3304-NFkB-LUC2™	Embryonic Microglia Clone 3	PD-L1, SIRPA	Brain	Available

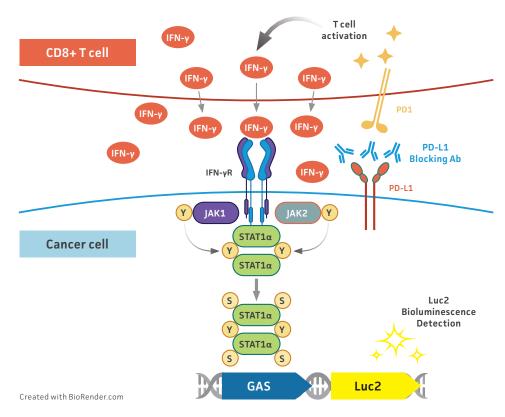


Figure 2: Mechanism of action. Luciferase signal generated by HCC827-GAS-Luc2 cells upon T cell activation through PD-L1 blockade.

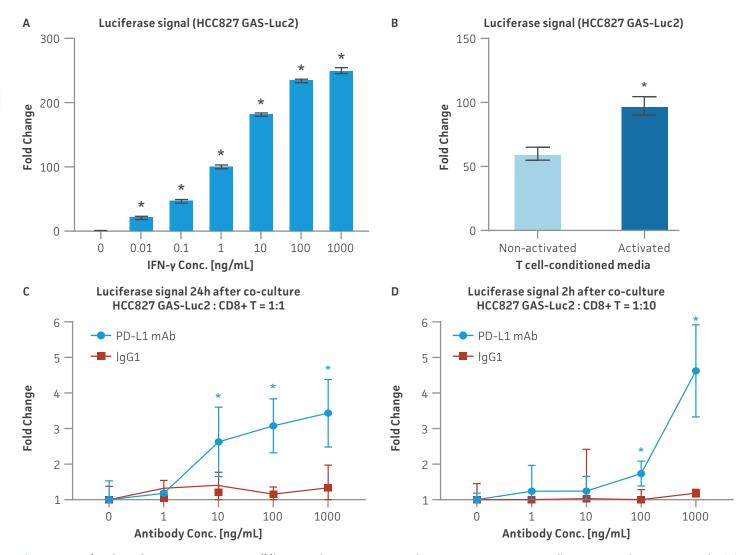


Figure 3: Evaluation of HCC827-GAS-Luc2 cell line. Luciferase expression from HCC827-GAS-Luc2 cells upon signaling activation by (A) IFN- γ stimulation (0.01 – 1,000 ng/mL), (B) conditioned-media stimulation from checkpoint matched non-activated and activated primary CD8+ T cells, and (C, D) co-culture with primary human CD8+ T cells in the presence of PD-L1 blocking antibody or isotype control IgG1 (1-1,000 ng/mL). N=3 in all experiments. *, P < 0.05.

LEARN MORE AT WWW.ATCC.ORG/IMMUNO-ONCOLOGY

CHECKPOINT MOLECULE PROFILING IN TUMOR AND IMMUNE CELLS AND APPLICATION FOR IMMUNO-ON-COLOGY DRUG SCREENING

Cancer immunotherapies have emerged as an exciting new method in treating cancer. In addition, treatments targeting immune checkpoints are promising approaches to unleash the potential of the anti-tumor immune response.

Although immune checkpoint blockades have exhibited anti-tumor effects in multiple cancer types, there are still challenges to overcome such as resistance and low response rate. Thus, there is a need for comprehensive data on the expression levels of checkpoint molecules based on cancer type, which can be utilized to guide specific treatment plans and combinations.

ATCC has complied a comprehensive data set of checkpoint molecule expression levels on a variety of tumor and immune cell lines and primary T cells. The cells that were tested demonstrate high expression levels of both checkpoint inhibitory and co-stimulatory molecules. These established cell lines can be incorporated into simple blocking assays or be integrated into co-culture testing systems. Additionally, this information provides a relevant and accessible model system for studying checkpoint molecule interactions and screening biologics as cancer immunotherapy treatments.

Table 3: Checkpoint molecule expression levels of immune cell receptors

Max

Median

	HLA typing						y checkpoint ı	molecules		
Cell Lines	ATCC® No.	HLA class I	HLA class II	PD-1	CTLA4	LAG-3	TIM-3	ВТГА	VISTA	TIGIT
Jurkat E6-1	<u>TIB-152</u> ™	+	-	45		71	43	202	2406	17
TALL-104	<u>CRL-11386</u> ™	+	-	75		159	1090	301	1051	0
MOLT-3	<u>CRL-1552</u> ™	+	-	230	71	107	42	191	377	32
НН	<u>CRL-2105</u> ™	+	+	243	24	1046	749	606	3878	1995
HuT 78	<u>TIB-161</u> ™	+	+	231	20	416	267	1114	2884	88
SUP-T1	<u>CRL-1942</u> ™	+	-	2076	219	81	20	487	1339	18
HM2	<u>HB-8587</u> ™	+	-	361	46	120	0	464	4075	221
MJ [G11]	<u>CRL-8294</u> ™	+	+	272	91	348	281	2740	1607	4727
CCRF-CEM	<u>CCL-119</u> ™	+	-	108	13	81	111	222	119	53
Primary CD8+ T cells	PCS-800-017 [™]	+	-	812	98	274	10745	623	1378	88
Primary CD4+ T cells	PCS-800-016 [™]	+	-	921	106	35	1381	756	1029	32
,	PCS-800-017 [™]									

The expression levels of established and novel inhibitory check point molecule receptors were profiled on basal immune cell lines available at ATCC by FACS analysis. HLA typing is identified by low expression (-) and high expression (+). Conditional formatting is added to the table to compare the expression of checkpoint molecules between cell lines (compare within each column). The value is calculated by subtracting the median fluorescence intensity (MFI) of the sample by the MFI of the isotype control.

Table 4: Checkpoint molecule expression levels of immune cell receptors

		HLA typing			Co	-stimulato	ry checkp	oint molec	ules			Immune cell marker	
Cell Lines	ATCC [®] No.	4-1BB	SOOI	CD30	CD28	0X40	GITR	CD226	CD4	CD8	CD4	8GD	
Jurkat E6-1	<u>TIB-152</u> ™	+	-	0	77	3463	0	0	156	11054	275	14	
TALL-104	CRL-11386™	+	-	30	501	36	14507	319	243	99	58	85358	
MOLT-3	<u>CRL-1552</u> ™	+	-	0	929	672	4353	273	303	149	143	617	
НН	<u>CRL-2105</u> ™	+	+	42	68	214676	512	1368	610	26814	29347	121	
HuT 78	<u>TIB-161</u> ™	+	+	240	1014	13216	431	3661	9674	901	7852	397	
SUP-T1	<u>CRL-1942</u> ™	+	-	0	54	1	15430	876	32	656	29250	81122	
HM2	<u>HB-8587</u> ™	+	-	0	518	531	56	854	322	736	229	5412	
MJ [G11]	<u>CRL-8294</u> ™	+	+	501	9072	51092	0	15528	37952	2987	21023	101	
CCRF-CEM	<u>CCL-119</u> ™	+	-	7	347	567	5884	163	479	342	9641	6306	
Primary CD8+ T cells	PCS-800-017 [™]	+	-	57	1567	71	607	119	720	4268	0	223247	
Primary CD4+ T cells	PCS-800-016 [™]	+	-	43	2252	862	6477	380	1040	5041	7916	21	
Median		Max											

The expression levels of established and novel co-stimulatory check point molecule receptors were profiled on basal immune cell lines available at ATCC by FACS analysis. HLA typing is identified by low expression (-) and high expression (+). Conditional formatting is added to the table to compare the expression of checkpoint molecules between cell lines (compare within each column). The value is calculated by subtracting the median fluorescence intensity (MFI) of the sample by the MFI of the isotype control.

Table 5: Checkpoint molecule expression levels of tumor cell ligands

F": with IFN	t IFNγ Iγ		HI typ					nhibitory	/ checkpoi	int molecu	le ligand	s		
			HLA class I	HLA class II	1	+	- 2	2+	ė.	+	- 4	+ +	- 5	+
Cancer	6.111	ATCC	ΙΓΑ	ILA	PD-L1	PD-L1	PD-L2	PD-L2	B7-H3	B7-H3	В7-Н4	B7-H4	HVEM	HVEM
уре	Cell lines	ATCC® catalog #										,		
	5637 HT-1197	<u>HTB-9</u> ™ CRL-1473™	+	-	52096 36857	143325 61670	49 7759	2594 0	60004 361220	52945 350756	0	0 88	1593 18982	1783 3450
Bladder	HT-1376	CRL-1473 CRL-1472™	+	_	27135	51493	1692	8578	74668	66185	0	0	365	1790
nauuei	RT4	HTB-2 TM	+	_	0	5054	52	518	143148	139442	0	42	717	160
	TCCSUP	HTB-5 TM	_	+	30543	48394	4325	9664	131058	123270	930	822	526	142
	SK-N-BE(2)	CRL-2271™	+	_	245	6837	0	258	15903	17884	156	123	262	237
Brain	U-87 MG	HTB-14 [™]	+	_	321	2990	249	246	73474	72722	338	263	4718	331
	U-87 MG-Luc2	——— HTB-14-LUC2™	+	_	15061	40367	0	0	29967	29009	1508	1374	487	706
	AU565	CRL-2351™	+	-	2428	11013	0	0	9476	8169	3514	2925	307	83:
	BT-20	HTB-19 [™]	+	_	6082	17072	886	4614	44830	44507	711	761	0	0
	DU4475	——— HTB-123™	+	_	1912	3232	1082	3774	59238	54996	1941	1317	4014	429
	HCC38	CRL-2314 [™]	+	-	13009	126059	3097	16705	220234	208819	2300	1565	6396	726
Breast	MCF7	HTB-22™	+	-	53	1802	0	0	46613	42793	4324	2944	2197	197
	MCF7-Luc2	HTB-22-LUC2™	+	-	0	3116	0	2793	56518	53829	575	936	1331	172
	MDA-MB-231	HTB-26 [™]	+	-	11359	20492	986	1880	12979	11668	149	125	456	103
	MDA-MB-468	<u>HTB-132</u> ™	+	-	221	5046	115	380	16180	16342	806	575	140	43
	T-47D	<u>HTB-133</u> ™	+	-	72	6355	0	0	32581	24851	828	594	597	70:
	HOS	CRL-1543™	-	+	13031	41473	2927	9075	60530	61277	289	305	211	55
lone	MG-63	CRL-1427™	-	+	0	7362	0	0	84745	79181	443	819	368	73
oune	Saos-2	<u>HTB-85</u> ™	+	-	6082	32705	0	0	7455	7136	332	329	897	124
	U-2 OS	<u>HTB-96</u> ™	+	-	5929	36019	290	5915	63080	64082	548	333	830	115
	Caco-2 [Caco2]	HTB-37 [™]	+	-	0	471	0	0	32201	30175	1315	1209	1900	181
olon	HCT-15	CCL-225 [™]	-	+	474	3790	35	0	12896	12520	137	94	513	94
	LoVo	CCL-229™	-	+	468	17697	0	0	20338	19572	347	346	975	248
اممما د	A-253	HTB-41™	+	-	2070	16019	123	3176	43926	41341	18	0	45	47
lead & Ieck	FaDu	HTB-43 [™]	+	-	2733	37007	205	13372	39475	31090	0	0	138	85
	FaDu-Luc2	HTB-43-LUC2 [™]	+	-	6965	29601	0	0	24921	20048	269	333	421	448
iver	C3A [HepG2/C3A]	CRL-10741 [™]	+	-	0	2114	0	2698	18098	16938	441	453	1362	268
	SK-HEP-1	HTB-52 [™]	+	+	657	8371	1201	8770	13236	13610	283	599	642	109
	A549	CCL-185 [™]	+	-	1512	9611	0	2476	34719	33139	0	0	764	75
	Calu-1	HTB-54 [™]	+	-	53834	114947	3528	10080	18438	19072	588	604	921	211
	NCI-H1650 [H-1650, H1650]	CRL-5883 [™]	+	-	3491	15369	1050	5615	127539	134041	1738	1422	263	476
	NCI-H226 [H226]	CRL-5826 [™]	-	+	49391	145367	10744	24379	73920	101793	640	767	0	67
.unq	NCI-H441 [H441]	HTB-174 [™]	+	-	13424	34487	359	1782	34363	32832	887	1044	383	829
. 5	NCI-H460 [H460]	HTB-177 [™]	+	-	7193	19574	921	2778	55359	49738	885	1089	0	74
	HCC827	CRL-2868™	+	-	9795	60468	3725	8477	41249	47178	1817	1721	879	0
	NCI-H1299	CRL-5803™	+	-	278	3436.5	0	92	37817	36030	0	0	0	0
	NCI-H1975 [H-1975, H1975]		+	-	2483	23447	490	4677	70851	62007	0	0	368	172
	NCI-H596 [H596]	HTB-178™	+	-	18669	40780	1275	3245	84320	77592	0	0	0	27
	A-375 [A375]	CRL-1619™	+	-	1255	27782	0	433	52580	40341	0	0	566	112
	A375-KRAS	CRL-1619IG-1™	+	-	40740	45361	1368	6891.5	21853	16451	0	0	1785	283
1elanoma	A375-KRAS-Luc2	CRL-1619IG-1-LUC2 TM	+	-	109294	117180	0	966	12826	13191	735	816	0	160
	RPMI-7951	HTB-66 TM	+	-	10229 1291	26724	2662	8763	65180	80081	0	0	523	164
	SH-4	CRL-7724 TM	+	-	400	12124 17538	1000.5	750	54016 26932	44759 17137	0 27	68 60	2556 236	335 118
warian	SK-MEL-24	HTB-71™	-	+		89033	718	750		11255	405			
varian	ES-2 AsPC-1	CRL-1978 [™] CRL-1682 [™]	+	+	57764 0	6325	155	5906 2800	11970 28044	26743	297	390 397	1161 1147	136 266
ancreas	PANC-1	CRL-1682 CRL-1469™	+	+	1049	0	0	2800	20419	21694	421	473	1276	97
and eds	PANC 10.05	CRL-1469 CRL-2547™	+		27818	43052	1359	4174	15027	17384	0	0	996	140
	PC-3	CRL-2547 CRL-1435™	-	+	18303	47222	346	2725	31886	29497	641	230	203	170
rostate	PC-3-Luc2	CRL-1435 CRL-1435-LUC2™	+	-	20083	30374	0	0	18686	19516	411	497	823	138
	A-431	CRL-1455-LUC2	+	_	13020	37809	1660	6635	64875	61082	996	1792	2656	512
kin	A-431-Luc2	CRL-1555 CRL-1555-LUC2™	+	_	2868	41277	688	3235	14291	12967	458	463	446	102
KIII				_	2000	416//	000	2622	14231	12307	4J0	403	440	102
terine	HEC-1-A	HTB-112™	+		0	0	0	0	23302	21501	337	373	418	44

The expression levels of established and novel checkpoint inhibitory molecule ligands were profiled on basal (-) and 100 ng/mL IFNγ-stimulated (+) tumor cell lines available at ATCC were profiled by FACS analysis. HLA typing is identified by low expression (-) and high expression (+). Conditional formatting is added to the table to compare the expression of checkpoint molecules between cell lines (compare within each column). The value is calculated by subtracting the median fluorescence intensity (MFI) of the sample by the MFI of the control isotype.

Table 6: Checkpoint molecule expression levels of tumor cell ligands.

-": without +": with IFN			HL typ	ing			Co-	stimulat	ory check	point mol	ecule liga	nds		
Cancer type	Cell lines	ATCC [®] No.	HLA class I	HLA class II	4-1BBL-	4-1BBL+	- 1-5001	+ 1-5001	CD155-	CD155+	-080-	+ 08O +	- 98C -	CD86+
урс	5637	HTB-9™	+	-	3085	2831	1322	1464	68780	85293	2092	3069	1909	1993
	HT-1197	 CRL-1473™	+	-	1001	1598	1731	2259	25101	25258	8101	6900	21270	7641
Bladder	HT-1376	CRL-1472™	+	-	0	0	3440	6322	36478	44828	4293	4179	1233	170'
	RT4	HTB-2™	+	-	2395	2961.5	5676	7754	40953	48452	883	1097	1482	195
	TCCSUP	HTB-5 [™]	-	+	3016	3758	315	366	271088	282653	3912	3573	3917	393
	SK-N-BE(2)	<u>CRL-2271</u> ™	+	-	626	528	228	240	5236	6395	452	350	923	778
Brain	U-87 MG	HTB-14 [™]	+	-	2804	3010	339	454	30877	33809	2926	2597	2080	196
	U-87 MG-Luc2	HTB-14-LUC2 [™]	+	-	1717	1370	141	219	36063	43417	1851	1491	984	753
	AU565	CRL-2351™	+	-	1289	841	633	856	37017	35953	983	1027	433	45
	BT-20	<u>HTB-19</u> ™	+	-	7297	8831	300	136	203815	235198	8916	9398	1172	124
	DU4475	HTB-123 [™]	+	-	8298	6525	0	0	36382	32343	8865	6426	2523	127
	HCC38	CRL-2314™	+	-	1912	3050	1525	1855	132767	134741	5751	4437	2143	190
Breast	MCF7	HTB-22™	+	-	4821	4165	1583	2402	23280	22977	5720	4584	2867	242
	MCF7-Luc2	HTB-22-LUC2™	+	-	3902	5935	465	1037	20258	22678	1724	5297	1215	214
	MDA-MB-231	HTB-26 [™]	+	-	531	777	14	37	38583	53188	563	428	346	23
	MDA-MB-468 T-47D	<u>HTB-132</u> ™ HTB-133™	+	-	740	769 1990	401 859	747 683	36560	43422 37651	4 75 3038	464 2166	308	29
	HOS		+	-	3140		0	000	39364 99713	124829	841	815	1620 443	132
	MG-63	CRL-1543™	-	+	1127	1210 4901	0	0	303805	268365	2894	6552	1339	
Bone	Saos-2	<u>CRL-1427</u> ™ HTB-85™	+	+	4326 2525	1975	0	0	58992	70813	1726	1733	1644	296 152
	U-2 OS	HTB-96™	+	-	2323	2660	784	778	112962	124648	2554	1174	3008	304
	Caco-2 [Caco2]	HTB-37 [™]	+	_	4255	5817	1060	661	44423	39942	6756	4849	4146	317
Colon	HCT-15	CCL-225™	_	+	369	251	0	21	33045	34475	411	140	441	33
,01011	LoVo	CCL-229™		+	1581	1647	775	1080	24870	36144	903	1271	1044	101
	A-253	HTB-41 [™]	+	-	1431	2558	3380	3887	67935	83057	3303	3051	731	98
Head &	FaDu	HTB-43™	+	_	1640	0	3643	4161	60462	62858	2728	2720	1904	195
Neck	FaDu-Luc2	HTB-43-LUC2™	+	_	1159	1591	484	557	35527	40460	1019	1334	2147	218
	C3A [HepG2/C3A]	CRL-10741™	+	_	1243	2171	394	511	54751	59271	1729	1914	1136	110
iver	SK-HEP-1	HTB-52 [™]	+	+	3066	2824	156	271	61906	75802	449	3240	383	133
	A549	CCL-185™	+	_	943	1345	2547	3209	87047	88786	719	1227	810	107
	Calu-1	HTB-54™	+	_	2993	3444	0	0	94510	114947	3240	3268	1210	125
	NCI-H1650 [H-1650, H1650]	CRL-5883™	+	_	8605	9501	0	0	353964	391949	9642	7584	1455	91
	NCI-H226 [H226]	CRL-5826™	_	+	2378	2758	3006	2629	136158	229665	2143	2477	1202	89
	NCI-H441 [H441]	HTB-174™	+	-	2762	2540	246	260	59151	73580	2841	3133	3440	325
.ung	NCI-H460 [H460]	———— HTB-177™	+	-	2375	3040	189	615	78046	86814	2342	3040	3792	322
	HCC827	 CRL-2868™	+	-	3726	3399	162	0	58497	105562	5176	7123	2222	191
	NCI-H1299	CRL-5803™	+	-	2768	3391	2961	4373	196936	184904	3765	3790	909	66
	NCI-H1975 [H-1975, H1975]	CRL-5908™	+	-	227	208	535	1455	168919	175547	3665	4409	1160	141
	NCI-H596 [H596]	<u>HTB-178</u> ™	+	-	0	0	3410.6	3890	255616	311989	5243	2880	1349	107
	A-375 [A375]	<u>CRL-1619</u> ™	+	-	0	0	755	544	30126	37903	3133	2863	1237	107
	A375-KRAS	CRL-1619IG-1 [™]	+	-	0	1852	1682	1837	105114	127213	4220	6126	2120	287
Лelanoma	A375-KRAS-Luc2	<u>CRL-1619IG-1-LUC2</u> ™	+	-	3526	3450	0	0	128469	160467	4777	5130	1723	178
neianoma	RPMI-7951	HTB-66™	+	-	0	0	1930	1297	66083	91229	883	1097	1482	195
	SH-4	<u>CRL-7724</u> ™	+	-	108	2006	1142	760	66235	65168	3429	4481	932	150
	SK-MEL-24	<u>HTB-71</u> ™	-	+	2903	3177	6613	5316	45197	75332	888	826	2945	260
)varian	ES-2	<u>CRL-1978</u> ™	+	-	2730	1971	188	0	92087	122142	1453	1620	3210	351
	AsPC-1	<u>CRL-1682</u> ™	-	+	1415	1444	310	546	32180	49052	825	1290	3033	309
ancreas	PANC-1	CRL-1469™	+	-	2031	2093	331	196	33618	34518	2265	2625	2005	187
	PANC 10.05	CRL-2547 [™]	+	-	1802	3716	847	857	40464	48360	2628	4485	1485	232
rostate	PC-3	CRL-1435™	-	+	5474	2108	0	0	91370	122713	2503	0	555	0
	PC-3-Luc2	<u>CRL-1435-LUC2</u> ™	+	-	2871	2989	217	0	57153	83352	1924	2850	3223	341
kin	A-431	CRL-1555™	+	-	2623	4203	1369	1757	130495	152286	2297	2824	1078	89
	A-431-Luc2	CRL-1555-LUC2™	+	-	845	942	0	10	39458	41452	618	709	528	57
Jterine	HEC-1-A	HTB-112™	+	_	1401	1471	199	136	46400	41305	2300	1860	628	72

The expression levels of established and novel co-stimulatory checkpoint molecule ligands were profiled on basal (-) and 100 ng/mL IFNy-stimulated (+) tumor cell lines available at ATCC were profiled by FACS analysis. HLA typing is identified by low expression (-) and high expression (+). Conditional formatting is added to the table to compare the expression of checkpoint molecules between cell lines (compare within each column). The value is calculated by subtracting the median fluorescence intensity (MFI) of the sample by the MFI of the control isotype.

CAR-T TARGET LUCIFERASE REPORTER CELL LINES

One of the bottlenecks in CAR-T therapeutic development is evaluating the biofunction of effector cells. This in vitro process involves a series of labor-intensive co-culture immunoassays. To address this challenge, we generated CAR-T Target Luciferase Reporter Cells lines that have high endogenous expression of clinically relevant cell surface tumor antigens, such as CD19, CD20, and HER2. These new immunooncology tools are comprised of both solid and liquid tumor cell lines that exhibit sensitive and stable luciferase reporter expression. These cells enable your immuno-therapeutic breakthroughs by allowing you to monitor the potency and efficacy of candidate CAR-T effector cells in your cytotoxicity and cell viability assays in real time.

Table 7: CAR-T Target Luciferase Reporter Cells

Designation	ATCC [®] No.	Disease	Target
WIL2-S-Luc2	CRL-8885-LUC2 [™]	B Cell Lymphoma	CD19
Raji-Luc2	CCL-86-LUC2™	Burkitt's Lymphoma	CD19
Daudi-Luc2	CCL-213-LUC2 TM	Burkitt's Lymphoma	CD20
Farage-Luc2	CRL-2630-LUC2 [™]	Non-Hodgkin's B Cell Lymphoma	CD20
BT-474-Luc2	HTB-20-LUC2 [™]	Breast Ductal Carcinoma	HER2

These convenient reporter-labeled cells allow you to eliminate workflows involving radioactive or fluorescent dye labeling. The cells retain high expression of both the target antigen and luciferase up to 30 population doublings. These flexible target cells can also be incorporated in other immuno-oncology applications such as ADCC and natural killer (NK) cell cytotoxicity assays.

- High expression stability of both target antigen and luciferase
- High signal-to-noise ratio (S/N)
- Physiologically relevant low E:T ratios

- High-performing, fully authenticated cell lines
- Easy-to-use reporter system
- Real-time, live-cell imaging possible

CHIMERIC ANTIGEN RECEPTOR

WIL2-S-Luc2 (ATCC® CRL-8885-LUC2™) or Raji-Luc2 (ATCC® CCL-86-LUC2™)

Human CD19 scFV CAR-T cell

Daudi-Luc2 (ATCC® CCL-213-LUC2™) or Farage-Luc2 (ATCC® CRL-2630-LUC2™) Human CD20 scFV CAR-T cell

BT474-Luc2 (ATCC® HTB-20-LUC2™)

Human Her2 scFV CAR-T cell

Figure 4: CAR-T Target Luciferase Reporter Cells. Schematic showing CAR-T target cells with expression of CD19+ WIL2-S-Luc2 and Raji-Luc2, CD20+Daudi-Luc2 and Farage-Luc2, and HER2+BT-474-Luc2 being surrounded and attacked by CD19-, CD20-, and HER2-targeting CAR-T cells, respectively.

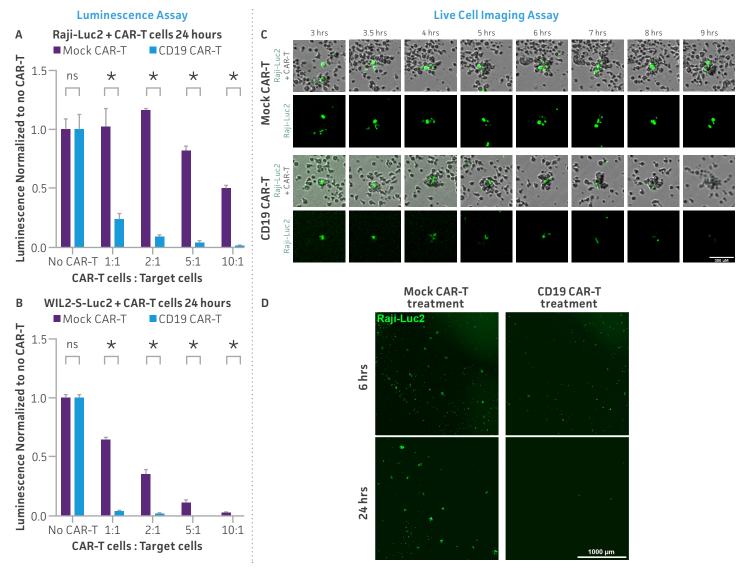


Figure 5: CAR-T Target Luciferase Reporter Cells can be incorporated into multiple CAR-T efficacy assays. (A) CD19 expressing Raji-Luc2 cells (B) or WIL2-S-Luc2 cells were used as target cells for either CD19 CAR-T or Mock CAR-T (control) effector cells from the same donor at the indicated effector to target cell ratios. A luciferase assay substrate was added, and the luminescence signal was detected. Loss of signal indicates cell death; the dose-dependent specific killing via CD19-targeting CAR-T cells was greater than the non-specific killing observed with the mock CAR-T cells. Additionally, Raji-Luc2 cells were stained with a cell labeling dye and then real-time fluorescent imaging was measured during co-culture with CD19 CAR-T effector cells. (C) Raji-Luc2 cells (Green) are surrounded by effector T cells, resulting in a decrease of fluorescence as compared to co-cultures with Mock-CAR-T cells. (D) After 6 and 24 hours of co-culture with CD19 CAR-T effector cells, we observed a decrease in the number of fluorescent cells; however, in a co-culture with Mock CAR-T cells numerous Raji-LUC2 cells were present. These results indicate that the ATCC CAR-T Target Luciferase Reporter Cells can be used to evaluate the potency of CAR-T cells in bioluminescence assays and live cell imaging in real time.

FOR MORE INFORMATION VISIT WWW.ATCC.ORG/CAR-T_TARGET

THP-1 REPORTER CELLS

The lack of stable and sensitive advanced immunology cell-based models to evaluate immune activation has hindered immune-oncology research and development for decades. To address this need, ATCC introduced luciferase reporters containing the response element of immunologically important transcription factors into the THP-1 cell line. The THP-1 LUC2 cell lines provide a means to confidently measure immune modulation for all your drug discovery and development efforts. Originating from a spontaneously immortalized human monocyte-like cell line that naturally expresses many pattern-recognition and cytokine receptors, ATCC THP-1 LUC2 cells represent the most physiologically relevant model to aid advancements in immuno-oncology and immune disorders.

Table 8: Features and Benefits of THP-1 Reporter Cells

Key Features	Key Benefits
Fully authenticated parental THP-1 cell line	No concerns about cross-contamination and misidentification, saves time and money
High signal-to-noise ratio	Clear and more intense results, straightforward data analysis
Verified, characterized stable expression	Reduced variability, reproducible results
Easy to culture, robust, and highly sensitive	Ease of use, customer convenience
Amenable to complex experimentation (combinatorial stimulation, co-culture)	Versatile and compatible with multiple platforms
High density cryopreservation	More viable cells post-thaw

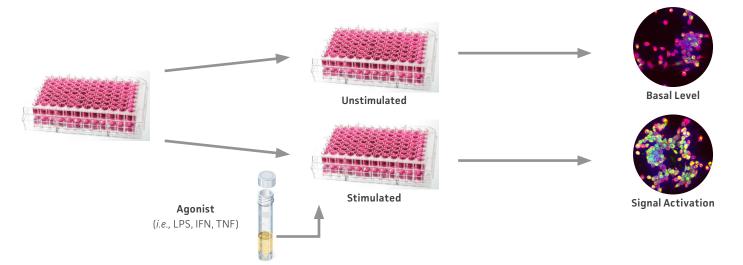


Figure 6: Quantitation of immunomodulation made easy. To use THP-1 LUC2 cells, simply seed in a 96-well plate. Stimulate the cells overnight with your compound of interest, then incubate the cells using a luciferase assay system and read the bioluminescence signals using a luminometer. Your immunomodulation data will be bigger, brighter, better.

Table 9: Available THP-1 LUC2 reporter cell lines

Response Element	ATCC No.	Signaling Pathway	Function
NFĸB	TIB-202-NFkB-LUC2™	NFĸB	Pivotal mediator of inflammatory response
GAS	TIB-202-GAS-LUC2™	JAK-STAT (Type II)	Initiates immune cell activation and recruitment
CRE	TIB-202-CRE-LUC2™	cAMP/PKA	Inflammatory mediator and phagocytosis modulator
ISRE	TIB-202-ISRE-LUC2™	JAK-STAT (Type I)	Initiates immune cell activation and recruitment
AP1	TIB-202-AP1-LUC2™	MAPK/ERK	Regulates innate and adaptive immune response
NFAT	TIB-202-NFAT-LUC2™	Calcineurin-NFAT	Mediates adaptive T and B cell activation

These high-quality cell lines are well suited to study the role of proteins involved in signaling cascades activated by immunomodulators, to optimize the MoA, pharmaceutical potency, and/or toxicological profile of leading drug candidates, and to evaluate the efficacy or toxicity of promising drug compounds in vitro assays.

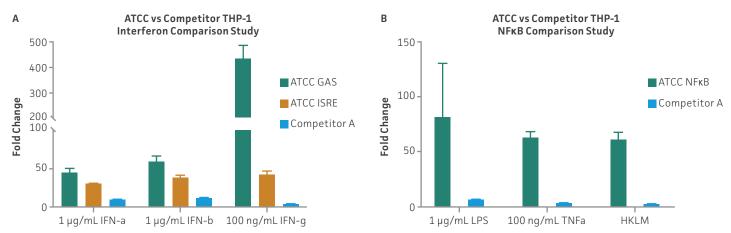


Figure 7: Comparison of luminescence and in vitro quantification of luciferase activity of THP-1 LUC2 and competitor reporter cell lines. Cells were seeded in a 96-well plate. After overnight stimulation with the appropriate interferons, bioluminescence signals were detected using a commercially available luciferase assay kit and a luminometer. Error bars show standard deviation (n=3). Panel (A) shows ATCC® THP-1 GAS-Luc2 (orange bar), THP-1 ISRE-Luc2 (yellow bar), or competitor immune regulator expression cells (green bar) stimulated with the indicated interferons and assessed for bioluminescence. Panel (B) shows ATCC® THP-1 NFkB-Luc2 (orange bar) or competitor immune regulator expression cells (green bar) treated with the indicated toll-like receptor agonists and assessed for bioluminescence intensity. In both studies, THP-1 luciferase-expressing cells exhibited enhanced bioluminescence signal compared to the competitor cells.

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LUCIFERASE CELL LINES

Imprecise in vivo animal models are a daily reality for cancer biologists. They cloud the results of biological mechanism studies and drug development work because it is often difficult to image and quantify engrafted tumors. Luciferase reporter cell lines provide a relatively simple, robust, and highly sensitive means to measure biological processes and to assess drug efficacy in animal models through bioluminescence imaging. They offer new tools for both in vitro luminescent assays and in vivo live animal bioluminescent imaging.

- Used to establish in vivo tumor models
- Quantifiable luciferase expression
- Verified Luc2 expression stability

- Derived from commonly used human and mouse cell lines
- Developed by single cell cloning
- High signal/background ratio

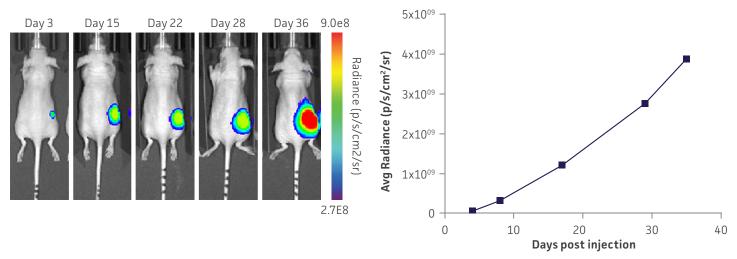


Figure 8: Luciferase-expressing reporter cell lines can be used in in vivo animal bioluminescent imaging. IDH1 Mutant U-87 Isogenic-Luc2 cells (3 x 10⁶) were injected subcutaneously into the dorsal region near the thigh of female nude mice. Tumor growth was monitored weekly using an optical bioluminescence imaging system. In vivo bioluminescence imaging demonstrated the progression of tumors, and the utility of luciferase-expressing reporter cell lines (here IDH1 Mutant U-87 Isogenic-Luc2) in xenograft animal model studies.

LEARN MORE AT WWW.ATCC.ORG/LUCIFERASE

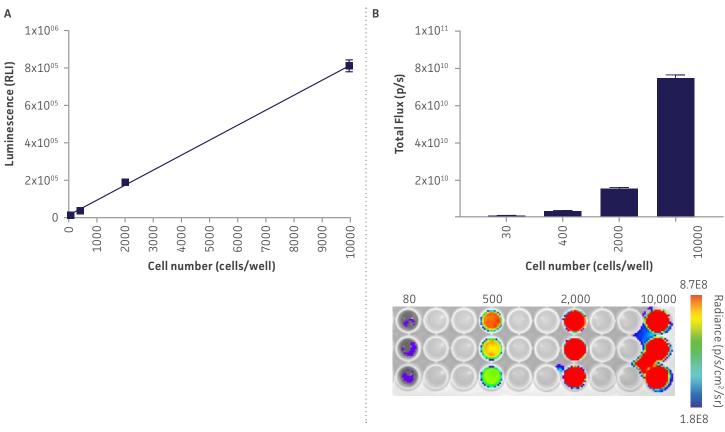


Figure 9: Luciferase-expressing reporter cells demonstrate linear, quantifiable signal in in vitro bioluminescence studies. IDH1 mutant-U-87 Isogenic-Luc2 were seeded in a 96-well plate at indicated cell numbers per well, and commercially prepared luciferase substrate preparation was added to the indicated wells. The luminescence of the plate was read within 10 minutes using a luminescence plate reader (A) and determined to have a linear correlation of bioluminescence intensity with cell numbers. (B) The plate was imaged using in vivo optical imaging system to quantify that photons emitted per cell. The resulting bioluminescence curves indicate that the luciferase-expressing reporter cells can be used to assess cell viability in live, unfixed cells.

LUCIFERASE-LABELED CELL LINES

ATCC maintains luciferase-expressing reporter cell lines derived from the most commonly used cells in molecular imaging studies. The addition of the luciferase reporter to these cell lines increases their utility by allowing for real-time imaging of the tumors.

ISOGENIC LUCIFERASE-LABELED CELL LINES

By utilizing the CRISPR/Cas9 gene editing, ATCC offers isogenic cell models harboring critical drug-resistant or -sensitive mutations that also express the luciferase reporter. These advanced models can be used in in vivo studies to identify novel, personalized treatment regimens.

Table 10: Luciferase-Labeled Human Cell Lines

ATCC® No.	Designation	Disease	Tissue
CCL-240-LUC2™	HL-60-Luc2	Leukemia	Blood
CCL-243-LUC2™	K-562-Luc2	Chronic Myelogenous Leukemia	Bone Marrow
HTB-96-LUC2™	U-2 OS-Luc2	Osteosarcoma	Bone
CRL-2003-LUC2™	TF-1-Luc2	Leukemia	Bone Marrow
CRL-2003IG-LUC2™	IDH2 R140Q mutant TF-1-Luc2	Leukemia	Bone Marrow
HTB-14-LUC2 [™]	U-87 MG-Luc2	Glioma	Brain
HTB-14IG-LUC2™	IDH1 R132H mutant U-87MG-Luc2	Glioma	Brain
HTB-22-LUC2 [™]	MCF7-Luc2	Adenocarcinoma	Breast
CCL-225-LUC2™	HCT-15-Luc2	Human Dukes' type C, colorectal adenocarcinoma	Colon
CCL-228-LUC2™	SW480-Luc2	Human Dukes' type B, colorectal adenocarcinoma	Colon
CCL-247-LUC2™	HCT 116-Luc2	Carcinoma	Colon
CCL-121-LUC2™	HT-1080-Luc2	Fibrosarcoma	Connective
CCL-185-LUC2™	A549-Luc2	Lung Carcinoma	Lung
CCL-185IG-LUC2™	EML4-ALK Fusion A549-Luc2	Lung Carcinoma	Lung
CRL-1469-LUC2™	PANC-1-Luc2	Carcinoma, Epithelioid	Pancreas
HTB-43-LUC2 [™]	FaDu-Luc2	Human Squamous Cell Carcinoma	Pharynx
<u>CRL-1435-LUC2</u> ™	PC-3-Luc2	Adenocarcinoma	Prostate
CRL-1740-LUC2 [™]	LNCaP clone FGC-Luc2	Carcinoma	Prostate
CRL-1555-LUC2™	A-431-Luc2	Carcinoma, Epidermoid	Skin
CRL-1619-LUC2™	A375-Luc2	Melanoma	Skin
<u>CRL-1619IG-1-LUC2</u> ™	KRAS G13D A375-Luc2	Melanoma	Skin
<u>CRL-1619IG-2-LUC2</u> ™	NRAS Q61K A375-Luc2	Melanoma	Skin
CRL-1739-LUC2™	AGS-Luc2	Human Gastric Adenocarcinoma	Stomach

Table 11: Luciferase-Labeled Mouse Cell Lines

ATCC® No.	Designation	Disease	Tissue
TIB-39-LUC2™	EL4-Luc2	Lymphoma	Blood
<u>CRL-2539-LUC2</u> ™	4T1-Luc2	Breast Cancer	Breast
CRL-1642-LUC2™	LL/2-Luc2	Lung Carcinoma	Lung
CRL-6323-LUC2™	B16-F1-Luc2	Melanoma	Skin
CRL-6475-LUC2™	B16-F10-Luc2	Melanoma	Skin

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HUMAN CANCER MODELS INITIATIVE

PATIENT-DERIVED CANCER MODELS

As part of our pledge to elevate biological models, ATCC is collaborating with the Human Cancer Models Initiative (HCMI) to offer scientists a wide variety of next-generation 2-D and 3-D patient-derived in vitro cancer models, including organoids and conditionally reprogrammed cells (CRCs). ATCC is committed to making available a growing collection of models generated by the HCMI, which will include both common as well as rare and understudied examples of cancer from numerous tissues. These HCMI models are valuable tools to study cancer, identify and target novel therapies, and facilitate translational cancer research.

To enhance their clinical relevance, the sequence data and patient clinical information for each model is available to the research community.

Various types of 2-D and 3-D models

- All models are human patient-derived
- Diverse genetic backgrounds
- Advanced models such as organoids
- Clinical and sequencing data available via the HCMI portal
- Models from primary, metastatic, and recurrent cancer
- Rare and pediatric cancers included
- Model-specific, easy-to-follow culturing protocols

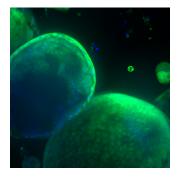
Patient-derived cancer models of the following physiological systems will be available:

- Circulatory System
- Digestive System
- Excretory System
- Integumentary System

- Musculo-skeletal System
- Nervous System
- Reproductive System
- Respiratory System

NEXT-GENERATION CANCER MODELS

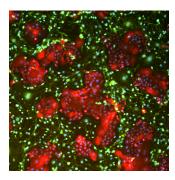
ORGANOIDS



Organoids are complex, self-organizing microtissues grown embedded within 3-D extracellular matrix. Primary patient-derived organoids have been described for various tissues, healthy and cancerous, including colon, intestine, stomach, breast, esophagus, lung, liver, prostate, and pancreas. Organoids are invaluable pre-clinical models for studying cancer and offer many advantages over existing human or non-human animal cancer models.

- May contain multiple differentiated cell types
- Exhibit cellular polarization
- Often possess a central lumen or other in vivo-like architecture
- Can remain phenotypically and genotypically stable after long term expansion

CONDITIONALLY REPROGRAMMED CELLS (CRCS) AND OTHER NON-ORGANOID MODELS

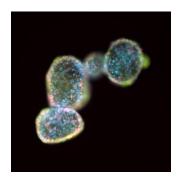


Conditional reprogramming is a cell culture technique that can be used to rapidly and efficiently establish patient-derived cell cultures from both normal and tumor cells. A major advantage of this system is that it:

- Eliminates the need for immortalization via transduction of viral or cellular genes
- Allows the expansion of a patient's tumor cells
- Reverts to differentiated phenotype in physiological culture conditions
- Makes it possible to identify the specific mutations in these cells and to screen the cells for sensitivity to drugs

In addition to CRCs, various other 2-D and 3-D model types, such as neurosphere models, are among the next-generation cancer models offered by the HCMI.

ABOUT HUMAN CANCER MODELS INITIATIVE (HCMI)



HCMI is an international consortium that is dedicated to generating novel human tumor-derived culture models with associated genomic and clinical data. The HCMI consortium comprises funding agencies and cancer model development institutions. The consortium's funding agencies include:

- National Cancer Institute (NCI)
- Cancer Research UK (CRUK)
- Hubrecht Organoid Technology (HUB)
- Wellcome Sanger Institute (WSI)

NCI-funded model development institutions include the Broad Institute and the Cold Spring Harbor Laboratory. CRUK and WSI co-fund organoid development in the United Kingdom; CRUK provides the patient samples, while WSI derives and sequences the organoid models. The foundation HUB is a Netherlands-based not-for-profit organization that derives and sequences organoid models. ATCC was selected as the sole distributor for the HCMI models. At ATCC the models are authenticated, expanded, preserved, and made available for global distribution. The HCMI model data is provided as an open source to the research community.

Tissue of Origin	Morphology	Disease	ATCC® No.
Abdominal wall	Organoid	Cancer; Metastatic	<u>PDM-523</u> ™
Ampulla Of Vater	Organoid	Cancer	<u>PDM-369</u> ™
Ampulla Of Vater	Organoid	Carcinoma	PDM-218 TM , PDM-102 TM
Bone	2-D adherent	Osteosarcoma	PDM-114 [™]
Bone; Metastatic Site: Bone	2-D adherent	Metastatic: bone cancer	<u>PDM-227</u> ™
Bone; Metastatic Site: Pleural Cavity	2-D adherent	Ewing sarcoma	<u>PDM-125</u> ™
Bone; Metastatic Site: Pleural Cavity	Mixed: adherent and suspension	Ewing sarcoma	<u>PDM-113</u> ™
Brain	2-D adherent	Glioblastoma	PDM-23 TM , PDM-109 TM , PDM-117 TM , PDM-121 TM , PDM-122 TM , PDM-132 TM , PDM-141 TM , PDM-143 TM , PDM-144 TM , PDM-147 TM , PDM-148 TM , PDM-149 TM , PDM-153 TM PDM-228 TM , PDM-235 TM , PDM-242 TM , PDM-244 TM , PDM-245 TM , PDM-297 TM , PDM-381 TM , PDM-400 TM , PDM-402 TM , PDM-403 TM , PDM-471 TM
Brain	Mixed: adherent and suspension	Glioblastoma	PDM-22 [™] , PDM-18 [™] , PDM-123 [™] , PDM-140 [™] , PDM-299 [™] , PDM-300 [™] , PDM-301 [™] , PDM-302 [™] , PDM-320 [™]
Brain	Neurosphere	Glioblastoma	<u>PDM-378</u> ™, <u>PDM-379</u> ™
Breast	Organoid	Cancer	<u>PDM-92™, PDM-195™, PDM-250™, PDM-350</u> ™, <u>PDM-520</u> ™,
Colon	Organoid	Adenocarcinoma	PDM-1 [™] , PDM-45 [™] , PDM-50 [™] , PDM-51 [™] , PDM-57 [™] , PDM-59 [™] , PDM-60 [™] , PDM-61 [™] , PDM-64 [™] , PDM-94 [™] , PDM-95 [™] , PDM-183 [™] , PDM-184 [™] , PDM-186 [™] , PDM-188 [™] , PDM-185 [™] , PDM-256 [™]
Colon	Organoid	Cancer	PDM-46 [™] , PDM-53 [™] , PDM-58 [™] , PDM-255 [™] , PDM-276 [™] , PDM-277 [™] , PDM-356 [™] , PDM-357 [™] , PDM-359 [™] , PDM-363 [™] , PDM-364 [™] , PDM-420 [™] , PDM-422 [™]
Colon	Organoid	Adenoma	<u>PDM-48</u> ™
Colon; Metastatic Site: Brain	Mixed: adherent and suspension	Adenocarcinoma	<u>PDM-104</u> [™] , <u>PDM-386</u> [™]
Colon; Metastatic Site: Colon	Organoid	Adenocarcinoma	<u>PDM-275</u> ™
Colon; Metastatic Site: Liver	Organoid	Adenocarcinoma	<u>PDM-42</u> ™
Colorectal	Organoid	Adenoma	<u>PDM-279</u> ™
Colorectal	Organoid	Cancer	<u>PDM-274</u> ™
Endometrium	Organoid	Cancer	<u>PDM-590</u> ™, <u>PDM-603</u> ™
Epithelial	2-D adherent	Epithelioid sarcoma	<u>PDM-229</u> ™, <u>PDM-236</u> ™
Esophagus	Organoid	Adenocarcinoma	PDM-68 [™] , PDM-70 [™] , PDM-71 [™] , PDM-74 [™] , PDM-76 [™] , PDM-76 [™] , PDM-78 [™] , PDM-83 [™] , PDM-124 [™] , PDM-131 [™] , PDM-159 [™]

Tissue of Origin	Morphology	Disease	ATCC® No.
Esophagus	Organoid	Cancer	PDM-65 [™] , PDM-67 [™] , PDM-72 [™] , PDM-73 [™] , https://www.atcc.org/products/pdm-7 [™] , PDM-120 [™] , PDM-225 [™] , PDM-243 [™]
Esophagus; Metastatic Site: Esophagus	Mixed: adherent and suspension	Cancer	<u>PDM-246</u> ™
Esophagus; Metastatic Site: Liver	Organoid	Cancer	PDM-119™
Esophagus; Metastatic Site: Pleural Cavity	Organoid	Cancer	<u>PDM-158</u> ™
Extrahepatic Bile Duct	Organoid	Adenocarcinoma	<u>PDM-216</u> [™] , <u>PDM-217</u> [™] , <u>PDM-220</u> [™]
Gall bladder: IPMN	Organoid	Benign neoplasm premalignant	<u>PDM-273</u> ™
Head and Neck	Organoid	Cancer	<u>PDM-441</u> ™
Kidney	2-D adherent	Wilms tumor	PDM-182™
Liver	Organoid	Cancer; Metastatic	<u>PDM-372</u> ™
Liver	Organoid	Cholangiocarcinoma	<u>PDM-219</u> ™
Lung	2-D adherent	Cancer	<u>PDM-305</u> ™
Lung	Organoid	Adenocarcinoma	<u>PDM-3</u> ™
Lung, Derived From Metastatic Site: Brain	Mixed: adherent and suspension	Cancer	<u>PDM-112</u> ™, <u>PDM-154</u> ™
Pancreas	Organoid	Adenocarcinoma	PDM-25 [™] , PDM-26 [™] , PDM-27 [™] , PDM-28 [™] , PDM-29 [™] , PDM-32 [™] , PDM-32 [™] , PDM-35 [™] , PDM-36 [™] , PDM-38 [™] , PDM-40 [™] , PDM-41 [™] , PDM-101 [™] , PDM-108 [™] , PDM-126 [™] , PDM-137 [™] , PDM-138 [™] , PDM-179 [™] , PDM-168 [™]
Pancreas	Organoid	Cancer	<u>PDM-24™, PDM-30™, PDM-37™, PDM-31</u> ™, <u>PDM-90</u> ™, <u>PDM-198™, PDM-421</u> ™, <u>PDM-423</u> ™
Pancreas: Metastatic Site: Liver	Organoid	Cancer	<u>PDM-107</u> ™, <u>PDM-288</u> ™
Pancreas: Metastatic Site: Liver	Organoid	Adenocarcinoma	<u>PDM-106</u> ™
Pancreas; Metastatic Site: Lymph Node	Organoid	Cancer	<u>PDM-223</u> ™
Pancreas; Metastatic Site: Pleural Cavity	Organoid	Adenocarcinoma	<u>PDM-170</u> ™
Rectosigmoid Junction	Organoid	Adenocarcinoma	<u>PDM-6</u> ™, <u>PDM-100</u> ™, <u>PDM-264</u> ™
Rectum	Organoid	Adenocarcinoma	<u>PDM-43</u> [™] , <u>PDM-47</u> [™] , <u>PDM-62</u> [™]
Rectum	Organoid	Adenoma	PDM-44 TM
Rectum	Organoid	Cancer	<u>PDM-97™, PDM-103™, PDM-190™, PDM-191™, PDM-254™, PDM-257™, PDM-258™, PDM-263™</u>
Rectum; Metastatic Site: Liver	Organoid	Adenocarcinoma	PDM-9 TM
Sigmoid Colon	Organoid	Adenocarcinoma	$\underline{PDM-2}^{TM}$, $\underline{PDM-4}^{TM}$, $\underline{PDM-5}^{TM}$, $\underline{PDM-7}^{TM}$, $\underline{PDM-8}^{TM}$
Skin	2-D adherent	Melanoma	PDM-282™, PDM-384™, PDM-392™, PDM-473™, PDM-478™, PDM-481™, PDM-484™, PDM-487™, PDM-487™, PDM-498™
Skin; Metastatic Site: Lymph Node	2-D adherent	Melanoma	PDM-284 [™] , PDM-285 [™] , PDM-291 [™] , PDM-293 [™] , PDM-313 [™] , PDM-319 [™] , PDM-387 [™] , PDM-388 [™]
Skin; Metastatic Site: Skin	2-D adherent	Melanoma	<u>PDM-292</u> ™, <u>PDM-389</u> ™
Small Intestine	Organoid	Adenoma	<u>PDM-368</u> ™
Small Intestine	Organoid	Cancer	<u>PDM-272</u> ™
Soft Tissue; Metastatic Site: Pleural Cavity	Mixed: adherent and suspension	Rhabdomyosarcoma	<u>PDM-129</u> ™
Soft Tissue; Metastatic Site: Soft Tissue	2-D adherent	Epithelioid sarcoma	<u>PDM-230</u> ™
Soft Tissue; Metastatic Site: Pleural Cavity	2-D adherent	Rhabdomyosarcoma	<u>PDM-238</u> ™, <u>PDM-290</u> ™
Stomach	Organoid	Adenocarcinoma	<u>PDM-136</u> [™] , <u>PDM-162</u> [™] , <u>PDM-233</u> [™]

Tissue of Origin	Morphology	Disease	ATCC® No.
Stomach	Organoid	Cancer	<u>PDM-146™, PDM-226™, PDM-296™, PDM-315™, PDM-315</u> ™,
Stomach; Metastatic Site: Lymph Node	Organoid	Adenocarcinoma	<u>PDM-135</u> ™
Stomach; Metastatic Site: Pleural Cavity	Organoid	Cancer	<u>PDM-161</u> ™
Stomach; Metastatic Site: Pleural Cavity	Organoid	Adenocarcinoma	<u>PDM-116</u> ™
Stomach; Metastatic Site: Pleural Cavity	Mixed: adherent and suspension	Cancer	<u>PDM-163</u> ™

ORGANOID GROWTH KITS

Organoids are valuable tools to study cancer, identify and target novel therapies, and facilitate translational cancer research. These these 3-D models are becoming more relevant because they are predictive of the in vivo tumor microenvironment. In efforts to simplify Organoid culture, ATCC has developed Organoid Growth Kits which are comprised of single-use supplements created to streamline media preparation. These kits contain the most costly and cumbersome supplements and reagents, reducing the time and effort required to prepare media and ensuring the successful growth of your organoids.

ATCC® No.	Growth Kit Name	Applicable Organoid ATCC® No.
ACS-7100 [™]	Organoid Growth Kit 1A	PDM-1™, PDM-2™, PDM-4™, PDM-5™, PDM-6™, PDM-7™, PDM-8™, PDM-9™, PDM-96™, PDM-96™, PDM-95™, PDM-96™, PDM-97™, PDM-100™, PDM-103™, PDM-188™, PDM-185™, PDM-186™, PDM-188™, PDM-190™, PDM-191™, PDM-254™, PDM-255™, PDM-256™, PDM-256™, PDM-256™, PDM-256™, PDM-264™, PDM-272™, PDM-273™, PDM-274™, PDM-275™, PDM-276™, PDM-277™, PDM-279™, PDM-356™, PDM-356™, PDM-356™, PDM-363™, PDM-363™, PDM-364™, PDM-372™, PDM-420™, PDM-422™
<u>ACS-7101</u> ™	Organoid Growth Kit 1B	PDM-24 [™] , PDM-25 [™] , PDM-27 [™] , PDM-28 [™] , PDM-29 [™] , PDM-30 [™] , PDM-31 [™] , PDM-31 [™] , PDM-32 [™] , PDM-33 [™] , PDM-34 [™] , PDM-35 [™] , PDM-36 [™] , PDM-36 [™] , PDM-36 [™] , PDM-101 [™] , PDM-106 [™] , PDM-107 [™] , PDM-108 [™] , PDM-116 [™] , PDM-119 [™] , PDM-120 [™] , PDM-126 [™] , PDM-126 [™] , PDM-131 [™] , PDM-135 [™] , PDM-136 [™] , PDM-137 [™] , PDM-138 [™] , PDM-146 [™] , PDM-158 [™] , PDM-159 [™] , PDM-161 [™] , PDM-162 [™] , PDM-162 [™] , PDM-170 [™] , PDM-179 [™] , PDM-198 [™] , PDM-216 [™] , PDM-217 [™] , PDM-218 [™] , PDM-219 [™] , PDM-220 [™] , PDM-221 [™] , PDM-222 [™] , PDM-223 [™] , PDM-226 [™] , PDM-233 [™] , PDM-246 [™] , PDM-288 [™] , PDM-296 [™] , PDM-315 [™] , PDM-316 [™] , PDM-368 [™] , PDM-369 [™] , PDM-421 [™] , PDM-423 [™]
ACS-7102™	Organoid Growth Kit 1C	PDM-3 [™]
<u>ACS-7103</u> ™	Organoid Growth Kit 1D	PDM-42 [™] , PDM-43 [™] , PDM-44 [™] , PDM-45 [™] , PDM-46 [™] , PDM-46 [™] , PDM-47 [™] , PDM-48 [™] , PDM-50 [™] , PDM-51 [™] , PDM-53 [™] , PDM-53 [™] , PDM-53 [™] , PDM-63 [™] , PDM-68 [™]
<u>ACS-7104</u> ™	Organoid Growth Kit 1E	<u>PDM-65™, PDM-67™, PDM-70™, PDM-71™, PDM-72™, PDM-73™, PDM-74™, PDM-76™, PDM-76™, PDM-773™, PDM-78™, PDM-78™, PDM-78™, PDM-78™, PDM-83™, PDM-243™</u>
ACS-7105 TM	Organoid Growth Kit 1F	<u>PDM-92</u> ™, <u>PDM-195</u> ™, <u>PDM-250</u> ™, <u>PDM-350</u> ™, <u>PDM-520</u> ™, <u>PDM-523</u> ™
<u>ACS-7106</u> ™	Organoid Growth Kit 1G	<u>PDM-37</u> ™, <u>PDM-102</u> ™

TUMOR/NORMAL MATCHED CELL LINE PAIRS

Tumor-derived cell lines matched to either normal or metastatic cell lines obtained from the same patient provide a valuable resource for cancer studies. The availability of such models allows researchers to analyze cancer-specific mutations, monitor the behavior and chemical sensitivity of tumor lines based on their normal counterparts, and develop drugs or therapies to target specific cancers or cancer mutations.

Table 1: Tumor and normal cell lines from the same individual

Cancer type	Tissue source	Name	ATCC® No.	Tissue source	Name	ATCC® No.
Primary site of disease				Normal pairing		
Adenocarcinoma	Lung	NCI-H1395	<u>CRL-5868</u> ™	Peripheral Blood	NCI-BL1395	<u>CRL-5957</u> ™
Adenocarcinoma	Lung	NCI-H1437	<u>CRL-5872</u> ™	Peripheral Blood	NCI-BL1437	<u>CRL-5958</u> ™
Adenocarcinoma	Lung	NCI-H2009	<u>CRL-5911</u> ™	Peripheral Blood	NCI-BL2009	<u>CRL-5961</u> ™
Adenocarcinoma	Lung, lymph node (metastasis)	NCI-H2087	<u>CRL-5922</u> ™	Peripheral Blood	NCI-BL2087	<u>CRL-5965</u> ™
Adenocarcinoma	Lung, pleural effusion	NCI-H2122	<u>CRL-5985</u> ™	Peripheral Blood	NCI-BL2122	<u>CRL-5967</u> ™
Basal Cell Carcinoma	Skin	TE 354.T	<u>CRL-7762</u> ™	Skin	TE 353.Sk	<u>CRL-7761</u> ™
Benign Osteoid Osteoma	Bone	Hs 919.T	<u>CRL-7672</u> ™	Skin	Hs 919.Sk	<u>CRL-7671</u> ™
Carcinoma	Mammary gland; breast	Hs 605.T	<u>CRL-7365</u> ™	Skin	Hs 605.Sk	<u>CRL-7364</u> ™
Carcinoma	Mammary gland; breast	Hs 854.T	CRL-7590™	Skin	Hs 854.Sk	<u>CRL-7589</u> ™
Ductal Carcinoma	Mammary gland; breast	HCC1008	<u>CRL-2320</u> ™	Peripheral Blood	HCC1007 BL	CRL-2319 [™]
Ductal Carcinoma	Mammary gland; breast	HCC1954	<u>CRL-2338</u> ™	Peripheral Blood	HCC1954 BL	<u>CRL-2339</u> ™
Ductal Carcinoma	Mammary gland; breast	Hs 578T	<u>HTB-126</u> ™	Mammary Gland; Breast	Hs 578Bst	HTB-125 [™]
Malignant Melanoma	Skin	COLO 829	<u>CRL-1974</u> ™	Peripheral Blood	COLO 829BL	<u>CRL-1980</u> ™
Melanoma	Skin	Hs 895.T	<u>CRL-7637</u> ™	Skin	Hs 895.Sk	<u>CRL-7636</u> ™
Mesothelioma	Lung, pleural effusion	NCI-H2052	<u>CRL-5915</u> ™	Peripheral Blood	NCI-BL2052	<u>CRL-5963</u> ™
Neuroendocrine Carcinoma	Lung, pleural effusion	NCI-H1770	<u>CRL-5893</u> ™	Peripheral Blood	NCI-BL1770	<u>CRL-5960</u> ™
Osteosarcoma	Bone	Hs 704.T	<u>CRL-7444</u> ™	Skin	Hs 704.Sk	<u>CRL-7443</u> ™
Osteosarcoma	Bone	Hs 707(A).T	<u>CRL-7448</u> ™	Skin	Hs 707(B).Ep	<u>CRL-7449</u> ™
Osteosarcoma	Bone	Hs 888.T	CRL-7622™	Lung	Hs888Lu	CCL-211™
Osteosarcoma	Bone	Hs 889.T	<u>CRL-7626</u> ™	Skin	Hs 889.Sk	<u>CRL-7625</u> ™
Osteosarcoma	Bone	Hs 890.T	<u>CRL-7628</u> ™	Skin	Hs 890.Sk	<u>CRL-7627</u> ™
Pagetoid Sarcoma	Skin	Hs 925.T	<u>CRL-7677</u> ™	Skin	Hs 925.Sk	<u>CRL-7676</u> ™
Primary Ductal Carcinoma	Mammary gland; breast	HCC38	<u>CRL-2314</u> ™	Peripheral Blood	HCC38 BL	<u>CRL-2346</u> ™
Primary Ductal Carcinoma	Mammary gland; breast	HCC1143	<u>CRL-2321</u> ™	Peripheral Blood	HCC1143 BL	<u>CRL-2362</u> ™
Primary Ductal Carcinoma	Mammary gland; breast	HCC1187	<u>CRL-2322</u> ™	Peripheral Blood	HCC1187 BL	<u>CRL-2323</u> ™
Primary Ductal Carcinoma	Mammary gland; breast	HCC1395	<u>CRL-2324</u> ™	Peripheral Blood	HCC1395 BL	<u>CRL-2325</u> ™
Primary Ductal Carcinoma	Mammary gland; breast	HCC1599	<u>CRL-2331</u> ™	Peripheral Blood	HCC1599 BL	<u>CRL-2332</u> ™
Primary Ductal Carcinoma	Mammary gland; breast	HCC1937	<u>CRL-2336</u> ™	Peripheral Blood	HCC1937 BL	<u>CRL-2337</u> ™
Primary Ductal Carcinoma	Mammary gland; breast	HCC2157	<u>CRL-2340</u> ™	Peripheral Blood	HCC2157 BL	<u>CRL-2341</u> ™
Primary Ductal Carcinoma	Mammary gland; breast	HCC2218	<u>CRL-2343</u> ™	Peripheral Blood	HCC2218 BL	CRL-2363 [™]
Scirrhous Adenocarcinoma	Mammary gland; breast	Hs 742.T	<u>CRL-7482</u> ™	Skin	Hs 742.Sk	<u>CRL-7481</u> ™
Small Cell Lung Cancer; Carcinoma	Lung	NCI-H1672	<u>CRL-5886</u> ™	Peripheral Blood	NCI-BL1672	<u>CRL-5959</u> ™
Non-Small Cell Lung Cancer; Carcinoma	Lung, lymph node (metastasis)	NCI-H2126	<u>CCL-256</u> ™	Peripheral Blood	NCI-BL2126	CCL-256.1 [™]
Small Cell Lung Cancer; Carcinoma	Lung	NCI-H1184	<u>CRL-5858</u> ™	Peripheral Blood	NCI-BL1184	<u>CRL-5949</u> ™
Small Cell Lung Cancer; Carcinoma	Lung	NCI-H2171	<u>CRL-5929</u> ™	Peripheral Blood	NCI-BL2171	<u>CRL-5969</u> ™
Small Cell Lung Cancer; Carcinoma	Lung	NCI-H2195	<u>CRL-5931</u> ™	Peripheral Blood	NCI-BL2195	<u>CRL-5956</u> ™
Small Cell Lung Cancer; Carcinoma	Bone marrow (metastasis)	NCI-H2107	<u>CRL-5983</u> ™	Peripheral Blood	NCI-BL2107	<u>CRL-5966</u> ™

Table 1: Tumor and normal cell lines from the same individual (continued)

Cancer type	Tissue source	Name	ATCC® No.	Tissue source	Name	ATCC® No.
Primary site of disease				Normal pairing		
Small Cell Lung Cancer; Carcinoma	Lung, pleural effusion	NCI-H128	<u>HTB-120</u> ™	Peripheral Blood	NCI-BL128	<u>CRL-5947</u> ™
Small Cell Lung Cancer; Carcinoma	Bone marrow (metastasis)	NCI-H209	<u>HTB-172</u> ™	Peripheral Blood	NCI-BL209	<u>CRL-5948</u> ™
Transitional Cell Carcinoma	Ureter	Hs 789.T	<u>CRL-7886</u> ™	Skin	Hs 789.Sk	<u>CRL-7518</u> ™

Table 2: Tumor and normal reference cell lines for detecting somatic mutations

Cancer type	Tissue source	Name	ATCC [®] No.	Tissue source	Name	ATCC® No.
Primary site of disease				Normal pairing		
Primary Ductal Carcinoma	Mammary gland; breast	HCC1395	SC-CRL-2324™	Peripheral Blood	HCC1395 BL	SC-CRL-2325™

Table 3: Primary and metastatic cell lines from the same individual

Cancer type	Tissue source	Name	ATCC® No.	Tissue source	Name	ATCC [®] No.
Primary site of disease				Metastatic pairin	ıg	
Colorectal Adenocarcinoma	Colon	SW480	CCL-228™	Lymph Node	SW620	<u>CCL-227</u> ™
Melanoma	Skin	Hs 688(A).T	<u>CRL-7425</u> ™	Lymph Node	Hs 688(B).T	<u>CRL-7426</u> ™

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