

# 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.



### 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.<sup>1,2</sup> 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
- DietFamily history
- Other specific parameters

#### Table 1: ATCC primary immune cells

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

Cell Туре	ATCC <sup>®</sup> No.	Number of Cells/vial	Positive Biomarkers
Peripheral Blood CD8+ Cytotoxic T Cells	<u>PCS-800-017</u> ™	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	<u>PCS-800-019</u> ™	5 million	CD45, CD56
iPSC-derived Mesenchymal Stem Cells	<u>ACS-7010</u> ™	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.

Designation	ATCC <sup>®</sup> No	Disease	Piomarkor	Tissue of origin	Status
Designation	ATCC NO.	Disease	Diomarker	rissue or origin	Status
HCC827-GAS-Luc2	CRL-2868-GAS-LUC2 <sup>™</sup>	Adenocarcinoma	PD-L1	Lung	Available
MG-63-GAS-Luc2	CRL-1427-GAS-LUC2™	Osteosarcoma	CD-155	Bone	Available
NCI-H1650-GAS-Luc2	<u>CRL-5883-GAS-LUC2</u> ™	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	<u>CRL-1593.2-NFkB-LUC2</u> ™	Histiocytic Lymphoma	SIRPA	Pleural effusion	Available
KG-1 NFkB-Luc2	<u>CCL-246-NFkB-LUC2™</u>	Acute myelogenous leukemia	SIGLEC10	Bone; Marrow	Available
HMC3 NFkB-Luc2	<u>CRL-3304-NFkB-LUC2</u> ™	Embryonic Microglia Clone 3	PD-L1, SIRPA	Brain	Available

#### Table 2: ATCC Checkpoint Luciferase Reporter Cells



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





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### 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

		HLA typing				Inhibitory	y checkpoint ı	nolecules		
Cell Lines	ATCC <sup>®</sup> 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		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
HH	<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	<u>PCS-800-017</u> ™	+	-	812	98	274	10745	623	1378	88
Primary CD4+ T cells	<u>PCS-800-016</u> ™	+	-	921	106	35	1381	756	1029	32

#### Median

Max

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:	<b>Checkpoint mol</b>	ecule expressio	n levels of immu	ne cell receptors

		HLA typing Co-stimulatory checkpoint molecules							Immune cell marker			
Cell Lines	ATCC <sup>®</sup> No.	4-1BB	ICOS	CD30	CD28	0X40	GITR	CD226	CD4	CD8	CD4	CD8
Jurkat E6-1	<u>TIB-152</u> ™	+	-	0	77	3463	0		156	11054	275	14
TALL-104	<u>CRL-11386</u> ™	+	-	30	501	36	14507	319	243	99		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	<u>PCS-800-017</u> ™	+	-	57	1567	71	607	119	720	4268	0	223247
Primary CD4+ T cells	<u>PCS-800-016</u> ™	+	-	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

"-": withou	t IFNγ		HL	A				Inhihitory	, chocknoi	int molocy	loligand	c		
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			lass	lass	÷	÷	ż	5+		÷	- 4	+ +	- V	+ V
Cancer	6 U.U.		ILAG	ILAG	- F	-i-	5		1-H	1-H	H-7	H-7	IVEN	IVEN
type	Cell lines	ATCC <sup>®</sup> catalog #	-	-	<b>C</b>	142225	<b>L</b>	2504	C0004	E 2045		0	1502	1700
	5637 HT-1197	<u>HIB-9</u> CRI-1473™	++	_	36857	61670	49 7759	2594	361220	350756	0	88	18982	34508
Bladder	HT-1376	CRL-1472™	+	_	27135	51493	1692	8578	74668	66185	0	0	365	1790
	RT4	HTB-2 <sup>™</sup>	+	-	0	5054	52	518	143148	139442	0	42	717	1602
	TCCSUP	<u>HTB-5</u> ™	-	+	30543	48394	4325	9664	131058	123270	930	822	526	1422
	SK-N-BE(2)	<u>CRL-2271</u> ™	+	-	245	6837	0	258	15903	17884	156	123	262	237
Brain	U-87 MG	HTB-14 <sup>™</sup>	+	-	321	2990	249	246	73474	72722	338	263	4718	3312
	U-87 MG-Luc2	HIB-14-LUC2"	+	-	15061	40367	0	0	29967	29009	1508	1374	487	706
	RT-20	<u>URL-2551</u> HTR_19™	+	_	6082	17072	886	461/	9470 ///830	o109 4/1507	711	761	0	0
	DU4475	HTB-123 <sup>™</sup>	+	_	1912	3232	1082	3774	59238	54996	1941	1317	4014	4293
	HCC38	CRL-2314™	+	-	13009	126059	3097	16705	220234	208819	2300	1565	6396	7267
Breast	MCF7	<u>HTB-22</u> ™	+	-	53	1802	0	0	46613	42793	4324	2944	2197	1972
	MCF7-Luc2	HTB-22-LUC2 <sup>™</sup>	+	-	0	3116	0	2793	56518	53829	575	936	1331	1723
	MDA-MB-231	<u>HTB-26</u> ™	+	-	11359	20492	986	1880	12979	11668	149	125	456	1031
	MDA-MB-468	<u>HTB-132</u> ™	+	-	221	5046	115	380	16180	16342	806	575	140	438
	T-47D	<u>HTB-133</u> ™	+	-	72	6355	0	0	32581	24851	828	594	597	703
	HUS MG C2	<u>CRL-1543</u> <sup></sup>	-	+	13031	41473	2927	9075	60530	61277	289	305	211	552
Bone	MG-03 Sans-2	<u>URL-1427</u> HTB-85™	-	+	6082	32705	0	0	84/45 7/155	79181	332	329	308 897	730 12//
	U-2 0S	<u>HTB-96</u> ™	+	_	5929	36019	290	5915	63080	64082	548	333	830	1152
	Caco-2 [Caco2]	HTB-37™	+	-	0	471	0	0	32201	30175	1315	1209	1900	1817
Colon	HCT-15	<u>CCL-225</u> ™	-	+	474	3790	35	0	12896	12520	137	94	513	947
	LoVo	<u>CCL-229</u> ™	-	+	468	17697	0	0	20338	19572	347	346	975	2481
Hood C	A-253	<u>HTB-41</u> ™	+	-	2070	16019	123	3176	43926	41341	18	0	45	477
Neck	FaDu	<u>HTB-43</u> ™	+	-	2733	37007	205	13372	39475	31090	0	0	138	855
	FaDu-Luc2	HTB-43-LUC2™	+	-	6965	29601	0	0	24921	20048	269	333	421	448
Liver		<u>CRL-10741</u> <sup>™</sup>	+	-	0	2114	1201	2698	18098	16938	441	453	1362	2682
	SK-HEP-1 Δ5/19	<u>HIB-52</u> CCL-185™	+	+	1512	9611	1201	8770 2476	3/719	33139	283	0	042 764	752
	Calu-1	<u>CCC-105</u> HTB-54™	+	_	53834	114947	3528	10080	18438	19072	588	604	921	2119
	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	672
Lung	NCI-H441 [H441]	<u>HTB-174</u> ™	+	-	13424	34487	359	1782	34363	32832	887	1044	383	829
Lung	NCI-H460 [H460]	<u>HTB-177</u> ™	+	-	7193	19574	921	2778	55359	49738	885	1089	0	742
	HCC827	<u>CRL-2868</u> ™	+	-	9795	60468	3725	8477	41249	47178	1817	1721	879	0
	NCI-H1299	<u>CRL-5803</u> ™	+	-	278	3436.5	0	92	37817	36030	0	0	0	0
	NCI-H1975 [H-1975, H1975]	<u>URL-5908</u> ™	+	-	2483	23447	490	4677	70851	62007	0	0	368	1729
	A_375 [A375]	<u>HIB-1/8</u> CRL-1619™	+	-	1255	40780	1275	3245	84320 52580	//3/1	0	0	566	275 1127
	A 375-KRAS	<u>CRL-1619IG-1</u> ™	+	_	40740	45361	1368	6891.5	21853	16451	0	0	1785	2838
	A375-KRAS-Luc2	CRL-1619IG-1-LUC2™	+	_	109294	117180	0	966	12826	13191	735	816	0	60
Melanoma	RPMI-7951	HTB-66 <sup>™</sup>	+	-	10229	26724	2662	8763	65180	80081	0	0	523	1646
	SH-4	<u>CRL-7724</u> ™	+	-	1291	12124	0	0	54016	44759	0	68	2556	3350
	SK-MEL-24	<u>HTB-71</u> ™	-	+	400	17538	1000.5	750	26932	17137	27	60	236	1187
Ovarian	ES-2	<u>CRL-1978</u> ™	+	-	57764	89033	718	5906	11970	11255	405	390	1161	1368
-	AsPC-1	<u>CRL-1682</u> ™	-	+	0	6325	155	2800	28044	26743	297	397	1147	2666
Pancreas	PANC-1	<u>CRL-1469</u> "	+	-	1049	0	1250	0	20419	21694	421	473	1276	976
	PANC 10.00	CRL-2047	+	-	18303	43052	3/18	41/4	31886	29/07	6/1	230	203	170/
Prostate	PC-3-Luc2	<u>CRL-1435-LUC2™</u>	+	- -	20083	30374	0	0	18686	19516	411	497	823	1387
	A-431	CRL-1555™	+	-	13020	37809	1660	6635	64875	61082	996	1792	2656	5120
Skin	A-431-Luc2	CRL-1555-LUC2™	+	-	2868	41277	688	3235	14291	12967	458	463	446	1021
Uterine	HEC-1-A	<u>HTB-112</u> ™	+	-	0	0	0	0	23302	21501	337	373	418	449

#### Median

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.

Max

-0

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

"-": withou	"-": without IFNγ HLA													
	• Y			=				Semarate	ory encert		cure rigu			
			lass	class	÷	5 5	4	÷	ż	+ 2	1.1	+	1.1	+
Cancer			ΓĂ	ΓĂ	-186	-18	COS-	SOS-	D15	D15	D80	D80	D86	D86
type	Cell lines	ATCC®No.	Ξ	Ξ	4	4	<u> </u>	2	0	0	0	0	0	0
	5637 UT 1107	HIB-9 <sup>™</sup>	+	-	3085	2831	1322	2250	68780 25101	85293	2092	3069	1909	1993
Bladder	HT-11376	CRL-1475 CRL-1/72™	+	-	1001	1099	3440	6322	25101	20200	6101	6900	1233	1707
Diauuei	RT4	<u>URL-1472</u> HTB-2™	+	_	2395	2961.5	5676	7754	40953	48452	883	1097	1482	1954
	TCCSUP	HTB-5™	_	+	3016	3758	315	366	271088	282653	3912	3573	3917	3933
	SK-N-BE(2)	 CRL-2271™	+	-	626	528	228	240	5236	6395	452	350	923	778
Brain	U-87 MG	<u>HTB-14</u> ™	+	-	2804	3010	339	454	30877	33809	2926	2597	2080	1968
	U-87 MG-Luc2	HTB-14-LUC2 <sup>™</sup>	+	-	1717	1370	141	219	36063	43417	1851	1491	984	753
	AU565	<u>CRL-2351</u> ™	+	-	1289	841	633	856	37017	35953	983	1027	433	454
	BT-20	<u>HTB-19</u> ™	+	-	7297	8831	300	136	203815	235198	8916	9398	1172	1244
	DU4475	<u>HTB-123</u> ™	+	-	8298	6525	0	0	36382	32343	8865	6426	2523	1278
	HCC38	<u>CRL-2314</u> ™	+	-	1912	3050	1525	1855	132767	134741	5751	4437	2143	1906
Breast	MCF7	<u>HTB-22</u> ™	+	-	4821	4165	1583	2402	23280	22977	5720	4584	2867	2424
	MCF7-Luc2	HTB-22-LUC2™	+	-	3902	5935	465	1037	20258	22678	1724	5297	1215	2149
	MDA-MB-231	HIB-26 ····	+	-	531	777	14	37	38583	53188	563	428	346	234
	MDA-MB-468	HIB-132 <sup>™</sup>	+	-	740	1000	401	/4/ 692	36560	43422	4/5	464 2166	1620	1225
	HOS	CRL_15/3™	Ŧ	-	1127	1210	0	083	99713	12/829	2038 8/1	2100	1020	400
	MG-63	<u>CRI-1427™</u>	_	+	4326	4901	0	0	303805	268365	2894	6552	1339	2968
Bone	Saos-2	HTB-85 <sup>™</sup>	+	_	2525	1975	0	0	58992	70813	1726	1733	1644	1525
	U-2 OS	HTB-96 <sup>™</sup>	+	_	2321	2660	784	778	112962	124648	2554	1174	3008	3045
	Caco-2 [Caco2]	HTB-37™	+	-	4255	5817	1060	661	44423	39942	6756	4849	4146	3170
Colon	HCT-15	CCL-225™	-	+	369	251	0	21	33045	34475	411	140	441	335
	LoVo	<u>CCL-229</u> ™	-	+	1581	1647	775	1080	24870	36144	903	1271	1044	1010
Lload C	A-253	<u>HTB-41</u> ™	+	-	1431	2558	3380	3887	67935	83057	3303	3051	731	985
Neck	FaDu	<u>HTB-43</u> ™	+	-	1640	0	3643	4161	60462	62858	2728	2720	1904	1951
	FaDu-Luc2	HTB-43-LUC2 <sup>™</sup>	+	-	1159	1591	484	557	35527	40460	1019	1334	2147	2183
Liver	C3A [HepG2/C3A]	<u>CRL-10741</u> ™	+	-	1243	2171	394	511	54751	59271	1729	1914	1136	1100
	SK-HEP-1	<u>HTB-52</u> ™	+	+	3066	2824	156	271	61906	75802	449	3240	383	1339
	A549	<u>CCL-185</u> ""	+	-	943	1345	2547	3209	87047	88786	719	1227	810	1078
		<u>HIB-54</u> <sup>™</sup>	+	-	2993	3444	0	0	94510	114947	3240	3268	1210	1254
	NCI-H1000 [H-1000, H1000]	CPL 5926M	+	-	2002	2750	2006	2620	126150	220665	21/2	7584	1202	916
	NCI-H441 [H441]	<u>URL-3820</u> HTR_17/™	-	-	2762	25/0	2/16	260	59151	73580	2143	2477	3440	3250
Lung	NCI-H460 [H460]	<u>HTB-177</u> ™	+	_	2375	3040	189	615	78046	86814	2342	3040	3792	3223
	HCC827	CRL-2868™	+	-	3726	3399	162	0	58497	105562	5176	7123	2222	1917
	NCI-H1299	CRL-5803™	+	-	2768	3391	2961	4373	196936	184904	3765	3790	909	662
	NCI-H1975 [H-1975, H1975]	CRL-5908™	+	-	227	208	535	1455	168919	175547	3665	4409	1160	1412
	NCI-H596 [H596]	<u>HTB-178</u> ™	+	-	0	0	3410.6	3890	255616	311989	5243	2880	1349	1078
	A-375 [A375]	<u>CRL-1619</u> ™	+	-	0	0	755	544	30126	37903	3133	2863	1237	1077
	A375-KRAS	<u>CRL-1619IG-1</u> ™	+	-	0	1852	1682	1837	105114	127213	4220	6126	2120	2878
Melanoma	A375-KRAS-Luc2	<u>CRL-1619IG-1-LUC2</u> ™	+	-	3526	3450	0	0	128469	160467	4777	5130	1723	1784
Melanoma	RPMI-7951	<u>HTB-66</u> ™	+	-	0	0	1930	1297	66083	91229	883	1097	1482	1954
	SH-4	<u>CRL-7724</u> ™	+	-	108	2006	1142	760	66235	65168	3429	4481	932	1507
	SK-MEL-24	<u>HTB-71</u> ™	-	+	2903	3177	6613	5316	45197	75332	888	826	2945	2605
Ovarian	ES-2	<u>CRL-1978</u> ™	+	-	2730	1971	188	0	92087	122142	1453	1620	3210	3510
Demostra	ASPL-1	<u>CRL-1682</u> <sup>M</sup>	-	+	1415	1444	310	546	32180	49052	825	1290	3033	3095
Pancreas		CRL-1469	+	-	2031	2093	331	196	33618	34518	2265	2625	2005	18/8
	PANU 10.00	CDL 1/25M	+	-	1802	3710	847	1 68	40464	40300	2020	4485	1485	2323
Prostate	PC-3-Luc2	<u>UKL-1435</u> CRL-1/(35-111C2™	- 	+	2871	2080	217	0	57152	122/13	2003	2850	2000	3/12
	Δ-431	CRI-1555™	+	-	2623	4203	1369	1757	130/95	152286	2297	282/	1078	803
Skin	A-431-Luc2	CRL-1555-LUC.2™	+	_	845	942	0	10	39458	41452	618	709	528	573
Uterine	HEC-1-A	HTB-112 <sup>™</sup>	+	-	1401	1471	199	136	46400	41305	2300	1860	628	722

#### Median

The expression levels of established and novel co-stimulatory checkpoint molecule ligands were profiled on basal (-) and 100 ng/mL IFNγstimulated (+) 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.

#### SEE THE DATA AT WWW.ATCC.ORG/IMMUNO-ONCOLOGY

Max

# **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 immuno-oncology 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 <sup>®</sup> No.	Disease	Target
WIL2-S-Luc2	<u>CRL-8885-LUC2</u> ™	B Cell Lymphoma	CD19
Raji-Luc2	<u>CCL-86-LUC2</u> ™	Burkitt's Lymphoma	CD19
Daudi-Luc2	<u>CCL-213-LUC2</u> ™	Burkitt's Lymphoma	CD20
Farage-Luc2	<u>CRL-2630-LUC2</u> ™	Non-Hodgkin's B Cell Lymphoma	CD20
BT-474-Luc2	<u>HTB-20-LUC2</u> ™	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
  - :Tratios F

#### CHIMERIC ANTIGEN RECEPTOR



Human CD19 scFV CAR-T cell

Daudi-Luc2 (ATCC<sup>®</sup> CCL-213-LUC2<sup>™</sup>) or Farage-Luc2 (ATCC<sup>®</sup> CRL-2630-LUC2<sup>™</sup>)



High-performing, fully authenticated cell lines

- Easy-to-use reporter system
- Real-time, live-cell imaging possible

#### BT474-Luc2 (ATCC<sup>®</sup> HTB-20-LUC2<sup>™</sup>)



Created with BioRender.com

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; 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 mono-cyte-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
Unsti	mulated Basal Level
Agonist (i.e., LPS, IFN, TNF)	aulated Signal Activation

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
ΝϜκΒ	<u>TIB-202-NFκB-LUC2</u> ™	NFκB	Pivotal mediator of inflammatory response
GAS	TIB-202-GAS-LUC2 <sup>™</sup>	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 <sup>™</sup>	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<sup>®</sup> <u>THP-1 GAS-Luc2</u> (orange bar), <u>THP-1 ISRE-Luc2</u> (yellow bar), or competitor immune regulator expression cells (green bar) stimulated with the indicated interferons and assessed for bioluminescence. Panel (B) shows ATCC<sup>®</sup> <u>THP-1 NFkB-Luc2</u> (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 10<sup>6</sup>) 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.

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

#### Table 11: Luciferase-Labeled Mouse Cell Lines

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

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

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

Musculo-skeletal System

Reproductive System

Nervous System

Respiratory System

- 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.

- Models from primary, metastatic, and recurrent cancer
- Rare and pediatric cancers included
- Model-specific, easy-to-follow culturing protocols

#### 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 <sup>®</sup> No.
Abdominal wall	Organoid	Cancer; Metastatic	<u>PDM-523</u> ™
Ampulla Of Vater	Organoid	Cancer	<u>PDM-369</u> ™
Ampulla Of Vater	Organoid	Carcinoma	<u>PDM-218</u> <sup>™</sup> , <u>PDM-102</u> <sup>™</sup>
Bone	2-D adherent	Osteosarcoma	<u>PDM-114</u> ™
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 <sup>TM</sup> , PDM-109 <sup>TM</sup> , PDM-117 <sup>TM</sup> , PDM-121 <sup>TM</sup> , PDM-122 <sup>TM</sup> , PDM-132 <sup>TM</sup> , PDM-141 <sup>TM</sup> , PDM-143 <sup>TM</sup> , PDM-144 <sup>TM</sup> , PDM-147 <sup>TM</sup> , PDM-148 <sup>TM</sup> , PDM-149 <sup>TM</sup> , PDM-153 <sup>TM</sup> PDM-228 <sup>TM</sup> , PDM-235 <sup>TM</sup> , PDM-242 <sup>TM</sup> , PDM-244 <sup>TM</sup> , PDM-245 <sup>TM</sup> , PDM-297 <sup>TM</sup> , PDM-381 <sup>TM</sup> , PDM-400 <sup>TM</sup> , PDM-402 <sup>TM</sup> , PDM-403 <sup>TM</sup> , PDM-471 <sup>TM</sup>
Brain	Mixed: adherent and suspension	Glioblastoma	<u>PDM-22<sup>™</sup></u> , <u>PDM-18<sup>™</sup></u> , <u>PDM-123<sup>™</sup></u> , <u>PDM-140<sup>™</sup></u> , <u>PDM-299</u> <sup>™</sup> , <u>PDM-300</u> <sup>™</sup> , <u>PDM-301</u> <sup>™</sup> , <u>PDM-302</u> <sup>™</sup>
Brain	Neurosphere	Glioblastoma	<u>PDM-378</u> ™, <u>PDM-379</u> ™
Breast	Organoid	Cancer	<u>PDM-92™, PDM-195</u> ™, <u>PDM-250</u> ™, <u>PDM-350</u> ™, <u>PDM-520</u> ™
Colon	Organoid	Adenocarcinoma	<u>PDM-1<sup>™</sup>, PDM-45<sup>™</sup>, PDM-50<sup>™</sup>, PDM-51<sup>™</sup>, PDM-57<sup>™</sup>, PDM-59<sup>™</sup>, PDM-60<sup>™</sup>, PDM-61<sup>™</sup>, PDM-64<sup>™</sup>, PDM-94<sup>™</sup>, PDM-95<sup>™</sup>, PDM-183<sup>™</sup>, PDM-184<sup>™</sup>, PDM-185<sup>™</sup>, PDM-185<sup>™</sup>, PDM-185<sup>™</sup>, PDM-185<sup>™</sup>, PDM-256<sup>™</sup>, PDM-185<sup>™</sup>, PDM-189<sup>™</sup>, PDM-256<sup>™</sup>, PDM-185<sup>™</sup>, PDM-189<sup>™</sup>, PDM-256<sup>™</sup>, PDM-185<sup>™</sup>, PDM-</u>
Colon	Organoid	Cancer	PDM-46 <sup>™</sup> , PDM-53 <sup>™</sup> , PDM-58 <sup>™</sup> , PDM-255 <sup>™</sup> , PDM-276 <sup>™</sup> ,   PDM-277 <sup>™</sup> , PDM-354 <sup>™</sup> PDM-356 <sup>™</sup> , PDM-357 <sup>™</sup> ,   PDM-359 <sup>™</sup> , PDM-363 <sup>™</sup> , PDM-364 <sup>™</sup> , PDM-410 <sup>™</sup> ,   PDM-415 <sup>™</sup> , PDM-417 <sup>™</sup> , PDM-418 <sup>™</sup> , PDM-419 <sup>™</sup> ,   PDM-420 <sup>™</sup> , PDM-422 <sup>™</sup>
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> <sup>™</sup> , <u>PDM-603</u> <sup>™</sup>
Epithelial	2-D adherent	Epithelioid sarcoma	<u>PDM-229</u> ™, <u>PDM-236</u> ™
Esophagus	Organoid	Adenocarcinoma	<u>PDM-68<sup>™</sup>, PDM-70<sup>™</sup>, PDM-71<sup>™</sup>, PDM-74<sup>™</sup>, PDM-76<sup>™</sup>, PDM-76<sup>™</sup>, PDM-77<sup>™</sup>, PDM-131<sup>™</sup>, PDM-124<sup>™</sup>, PDM-131<sup>™</sup>, PDM-150<sup>™</sup></u>

Tissue of Origin	Morphology	Disease	ATCC <sup>®</sup> No.
Esophagus	Organoid	Cancer	PDM-65 <sup>™</sup> , <u>PDM-67<sup>™</sup>, PDM-72<sup>™</sup>, PDM-73<sup>™</sup>, https://</u> www.atcc.org/products/pdm-7 <sup>™</sup> , <u>PDM-120</u> <sup>™</sup> , <u>PDM-225</u> <sup>™</sup> , <u>PDM-243</u> <sup>™</sup>
Esophagus; Metastatic Site: Esophagus	Mixed: adherent and suspension	Cancer	<u>PDM-246</u> <sup>™</sup>
Esophagus; Metastatic Site: Liver	Organoid	Cancer	<u>PDM-119</u> ™
Esophagus; Metastatic Site: Pleural Cavity	Organoid	Cancer	<u>PDM-158</u> ™
Extrahepatic Bile Duct	Organoid	Adenocarcinoma	<u>PDM-216</u> <sup>™</sup> , <u>PDM-217</u> <sup>™</sup> , <u>PDM-220</u> <sup>™</sup>
Gall bladder: IPMN	Organoid	Benign neoplasm premalignant	<u>PDM-273</u> ™
Head and Neck	Organoid	Cancer	PDM-441 <sup>™</sup>
Kidney	2-D adherent	Wilms tumor	<u>PDM-182</u> ™
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 <sup>™</sup> , PDM-26 <sup>™</sup> , PDM-27 <sup>™</sup> , PDM-28 <sup>™</sup> , PDM-29 <sup>™</sup> , PDM-32 <sup>™</sup> , PDM-33 <sup>™</sup> , PDM-34 <sup>™</sup> , PDM-35 <sup>™</sup> , PDM-36 <sup>™</sup> , PDM-38 <sup>™</sup> , PDM-39 <sup>™</sup> , PDM-40 <sup>™</sup> , PDM-41 <sup>™</sup> , PDM-101 <sup>™</sup> , PDM-108 <sup>™</sup> , PDM-126 <sup>™</sup> , PDM-137 <sup>™</sup> , PDM-138 <sup>™</sup> , PDM-179 <sup>™</sup> , PDM-168 <sup>™</sup>
Pancreas	Organoid	Cancer	<u>PDM-24<sup>™</sup>, PDM-30<sup>™</sup>, PDM-37<sup>™</sup>, PDM-31</u> <sup>™</sup> , <u>PDM-90<sup>™</sup>, PDM-198</u> <sup>™</sup> , <u>PDM-421</u> <sup>™</sup> , <u>PDM-423<sup>™</sup></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> <sup>™</sup> , <u>PDM-100</u> <sup>™</sup> , <u>PDM-264</u> <sup>™</sup>
Rectum	Organoid	Adenocarcinoma	<u>PDM-43</u> ™, <u>PDM-47</u> ™, <u>PDM-62</u> ™
Rectum	Organoid	Adenoma	<u>PDM-44</u> ™
Rectum	Organoid	Cancer	<u>PDM-97</u> <sup>™</sup> , <u>PDM-103</u> <sup>™</sup> , <u>PDM-190</u> <sup>™</sup> , <u>PDM-191</u> <sup>™</sup> , <u>PDM-254</u> <sup>™</sup> , <u>PDM-257</u> <sup>™</sup> , <u>PDM-258</u> <sup>™</sup> , <u>PDM-263</u> <sup>™</sup>
Rectum; Metastatic Site: Liver	Organoid	Adenocarcinoma	<u>PDM-9</u> ™
Sigmoid Colon	Organoid	Adenocarcinoma	<u>PDM-2</u> <sup>™</sup> , <u>PDM-4</u> <sup>™</sup> , <u>PDM-5</u> <sup>™</sup> , <u>PDM-7</u> <sup>™</sup> , <u>PDM-8</u> <sup>™</sup>
Skin	2-D adherent	Melanoma	<u>PDM-282™, PDM-384™, PDM-392™, PDM-473™,</u> <u>PDM-478™, PDM-481</u> ™, <u>PDM-484</u> ™, <u>PDM-487</u> ™, <u>PDM-494</u> ™, <u>PDM-498</u> ™
Skin; Metastatic Site: Lymph Node	2-D adherent	Melanoma	<u>PDM-284<sup>™</sup>, PDM-285<sup>™</sup>, PDM-291<sup>™</sup>, PDM-293<sup>™</sup>, PDM-293<sup>™</sup>, PDM-313<sup>™</sup>, PDM-319<sup>™</sup>, PDM-387<sup>™</sup>, PDM-388<sup>™</sup></u>
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™, PDM-162™, PDM-233</u> ™

Tissue of Origin	Morphology	Disease	ATCC <sup>®</sup> No.
Stomach	Organoid	Cancer	<u>PDM-146<sup>™</sup>, PDM-226<sup>™</sup>, PDM-296<sup>™</sup>, PDM-315<sup>™</sup>, PDM-315<sup>™</sup>, PDM-316</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 <sup>®</sup> No.	Growth Kit Name	Applicable Organoid ATCC <sup>®</sup> No.
<u>ACS-7100</u> ™	Organoid Growth Kit 1A	PDM-1 <sup>™</sup> , PDM-2 <sup>™</sup> , PDM-5 <sup>™</sup> , PDM-5 <sup>™</sup> , PDM-7 <sup>™</sup> , PDM-8 <sup>™</sup> , PDM-9 <sup>™</sup> , PDM-94 <sup>™</sup> , PDM-95 <sup>™</sup> , PDM-95 <sup>™</sup> , PDM-96 <sup>™</sup> , PDM-188 <sup>™</sup> , PDM-188 <sup>™</sup> , PDM-189 <sup>™</sup> , PDM-190 <sup>™</sup> , PDM-191 <sup>™</sup> , PDM-254 <sup>™</sup> , PDM-255 <sup>™</sup> , PDM-256 <sup>™</sup> , PDM-256 <sup>™</sup> , PDM-256 <sup>™</sup> , PDM-263 <sup>™</sup> , PDM-264 <sup>™</sup> , PDM-272 <sup>™</sup> , PDM-273 <sup>™</sup> , PDM-274 <sup>™</sup> , PDM-275 <sup>™</sup> , PDM-276 <sup>™</sup> , PDM-276 <sup>™</sup> , PDM-276 <sup>™</sup> , PDM-272 <sup>™</sup> , PDM-272 <sup>™</sup> , PDM-272 <sup>™</sup> , PDM-272 <sup>™</sup> , PDM-264 <sup>™</sup> , PDM-264 <sup>™</sup> , PDM-364 <sup>™</sup> , PDM-364 <sup>™</sup> , PDM-410 <sup>™</sup> , PDM-415 <sup>™</sup> , PDM-415 <sup>™</sup> , PDM-4110 <sup>™</sup> , PDM-420 <sup>™</sup> , PDM-422 <sup>™</sup>
<u>ACS-7101</u> ™	Organoid Growth Kit 1B	PDM-24 <sup>™</sup> , PDM-25 <sup>™</sup> , PDM-27 <sup>™</sup> , PDM-28 <sup>™</sup> , PDM-29 <sup>™</sup> , PDM-30 <sup>™</sup> , PDM-31 <sup>™</sup> , PDM-32 <sup>™</sup> , PDM-33 <sup>™</sup> , PDM-34 <sup>™</sup> , PDM-35 <sup>™</sup> , PDM-36 <sup>™</sup> , PDM-36 <sup>™</sup> , PDM-36 <sup>™</sup> , PDM-36 <sup>™</sup> , PDM-108 <sup>™</sup> , PDM-108 <sup>™</sup> , PDM-116 <sup>™</sup> , PDM-119 <sup>™</sup> , PDM-120 <sup>™</sup> , PDM-124 <sup>™</sup> , PDM-126 <sup>™</sup> , PDM-131 <sup>™</sup> , PDM-135 <sup>™</sup> , PDM-136 <sup>™</sup> , PDM-138 <sup>™</sup> , PDM-146 <sup>™</sup> , PDM-158 <sup>™</sup> , PDM-159 <sup>™</sup> , PDM-161 <sup>™</sup> , PDM-162 <sup>™</sup> , PDM-162 <sup>™</sup> , PDM-168 <sup>™</sup> , PDM-170 <sup>™</sup> , PDM-179 <sup>™</sup> , PDM-198 <sup>™</sup> , PDM-216 <sup>™</sup> , PDM-217 <sup>™</sup> , PDM-218 <sup>™</sup> , PDM-218 <sup>™</sup> , PDM-220 <sup>™</sup> , PDM-220 <sup>™</sup> , PDM-222 <sup>™</sup> , PDM-223 <sup>™</sup> , PDM-226 <sup>™</sup> , PDM-233 <sup>™</sup> , PDM-246 <sup>™</sup> , PDM-288 <sup>™</sup> , PDM-296 <sup>™</sup> , PDM-315 <sup>™</sup> , PDM-316 <sup>™</sup> , PDM-368 <sup>™</sup> , PDM-368 <sup>™</sup> , PDM-423 <sup>™</sup>
<u>ACS-7102</u> ™	Organoid Growth Kit 1C	PDM-3™
<u>ACS-7103</u> ™	Organoid Growth Kit 1D	<u>PDM-42<sup>™</sup>, PDM-43<sup>™</sup>, PDM-44<sup>™</sup>, PDM-45<sup>™</sup>, PDM-46<sup>™</sup>, PDM-46<sup>™</sup>, PDM-48<sup>™</sup>, PDM-50<sup>™</sup>, PDM-51<sup>™</sup>, PDM-53<sup>™</sup>, PDM-57<sup>™</sup>, PDM-58<sup>™</sup>, PDM-58<sup>™</sup>, PDM-58<sup>™</sup>, PDM-68<sup>™</sup>, PDM-68<sup>™</sup></u>
<u>ACS-7104</u> ™	Organoid Growth Kit 1E	<u>PDM-65™, PDM-67™, PDM-70</u> ™, <u>PDM-71™, PDM-72™, PDM-73</u> ™, <u>PDM-74</u> ™, <u>PDM-76</u> ™, <u>PDM-77</u> ™, <u>PDM-78</u> ™, <u>PDM-79</u> ™, <u>PDM-83</u> ™, <u>PDM-243</u> ™
<u>ACS-7105</u> ™	Organoid Growth Kit 1F	<u>PDM-92™, PDM-195™, PDM-250™, PDM-350™, PDM-520™, PDM-523</u> ™
ACS-7106 <sup>™</sup>	Organoid Growth Kit 1G	PDM-37™, PDM-102™

# 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.

Cancer type	Tissue source	Name	ATCC <sup>®</sup> No.	Tissue source	Name	ATCC <sup>®</sup> 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	<u>CRL-2319</u> ™
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	<u>HTB-125</u> ™
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	<u>CRL-7622</u> ™	Lung	Hs888Lu	<u>CCL-211</u> ™
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	<u>CRL-2363</u> ™
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	<u>CCL-256.1</u> ™
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

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

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

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

Cancer type	Tissue source	Name	ATCC <sup>®</sup> No.	Tissue source	Name	ATCC <sup>®</sup> No.
Primary site of disease				Metastatic pairing	9	
Colorectal Adenocarcinoma	Colon	SW480	<u>CCL-228</u> ™	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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