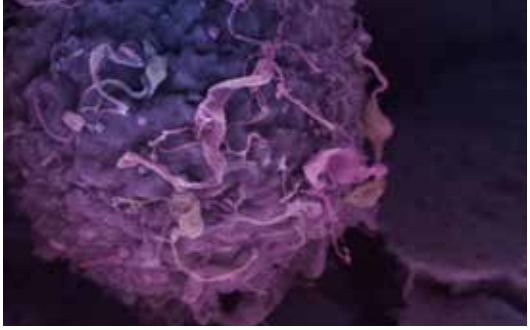

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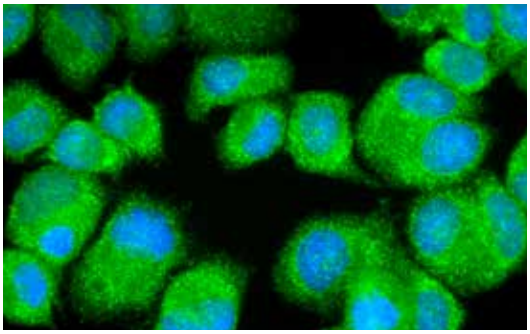
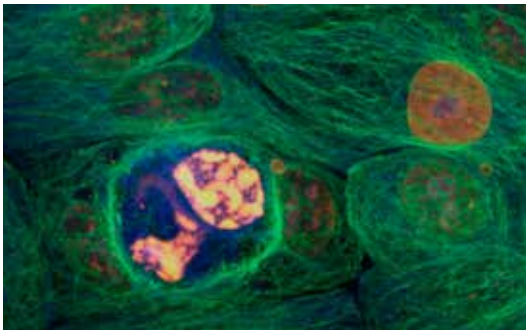
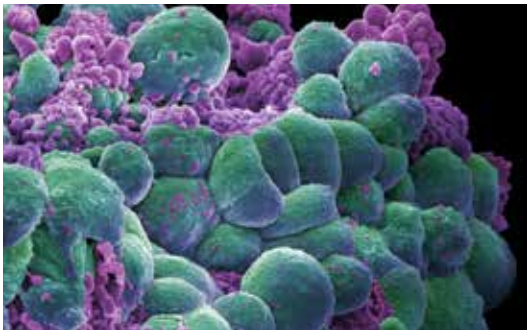
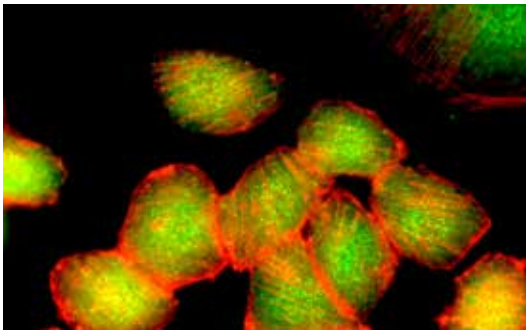
Breast Cancer Resource Book

BREAST CANCER RESOURCE BOOK

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ATCC is the premier global biological materials resource and standards organization

ATCC is an innovative global partner to the scientific community for authenticated biomaterials, quality standards, and custom services. With the world's largest and most diverse collection of human, animal, and plant cell lines, as well as molecular genomic tools, microorganisms, and biological products, ATCC is the most respected and trusted resource for the worldwide research community. Since its founding in 1925, ATCC associates share in its mission to acquire, authenticate, preserve, develop, and distribute biological materials and information to advance better science.

ATCC and breast cancer

According to the American Cancer Society, breast cancer is the leading cause of death among all female cancer patients. Because of its scope in morbidity and mortality, breast cancer represents an area of intense study for scientists and research clinicians. ATCC has extensive resources of normal and tumor breast tissue cells, as well as culture reagents and diagnostic standards, available for the pursuit of front-line breast cancer research.

INTRODUCTION

Worldwide, breast cancer accounts for approximately 23% of all cancer diagnoses in women, and represents the leading cause of cancer deaths among all female cancer patients[1]. Thus, breast cancer affects a large portion of the global population and constitutes a substantial public health burden. Consequently, it has generated a considerable amount of research interest.

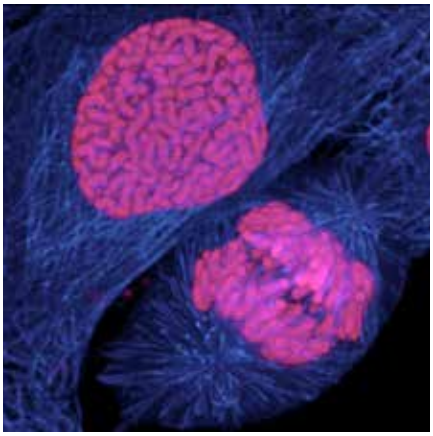
Breast cancers encompass a heterogeneous array of tumor types that are classified according to their histological and molecular characteristics. Currently, breast cancers are sorted into one of at least four subtypes, although classification methods are being refined as molecular profiling techniques improve. Each subtype is associated with a different prognosis and course of treatment (see Table below). Therefore, to generate the most effective treatment options, investigators need *in vitro* research tools that represent the heterogeneity of breast cancers *in vivo*.

This resource book will describe the extensive collection of cell lines, primary cells, and associated reagents that ATCC offers to support breast cancer research. This resource book was created to assist researchers in the study of this complicated disease. We hope you will find it a helpful resource for planning and getting your experiments up and running. For updates to our breast cancer portfolio, visit www.atcc.org/BreastCancerPanel.

Breast Cancer Classifications

Subtype	Immunoprofile	Characteristics[2 3]
Luminal A	ER+, PR+/-, HER2-	Low expression of proliferation marker Ki67 Responsive to hormone therapy Often responsive to chemotherapy
Luminal B	ER+, PR+/-, HER2+	High expression of proliferation marker Ki67 Usually responsive to hormone therapy Variably responsive to chemotherapy Variably responsive to HER2 antibody therapies (i.e. trastusumab)
Basal (Triple Negative)	ER-, PR-, HER2-	High expression of proliferation marker Ki67 Expression of EGFR+ Variable expression of basal cell marker cytokeratin 5/6 Not responsive to hormone therapy, but often responsive to chemotherapy
HER2 amplified	ER-, PR-, HER2+	High expression of proliferation marker Ki67 Often responsive to HER2 antibody therapies (i.e. trastusumab) and chemotherapy

TUMOR CELL LINES



Tumor cell lines have formed the cornerstone of breast cancer research since the early 1970s, when the MCF-7 line and the MD Anderson series were first established[2]. Since that time, the number of tumor cell lines available to breast cancer researchers has exploded and their contribution to our understanding of this complex disease cannot be overstated. In fact, some of the most effective treatment options available today exist because researchers were able to tease out the disease mechanism using an *in vitro* culture system and subsequently design targeted therapeutics.

Tumor cell lines have become even more powerful research tools with the advent of next-generation sequencing technology. The availability of this technology has led to the formation of several large-scale sequencing initiatives which have generated a vast amount of actionable data. One such initiative is the Cancer Cell Line Encyclopedia (CCLE). The CCLE is a collaborative effort between Novartis and the Broad Institute that has released mutation data for 1,651 genes representing nearly 1,000 cell lines. The CCLE research group used this data set to compare the copy number, expression pattern, and mutation frequency of tumor cell lines with primary tumors and showed that tumor cell lines are representative of their *in vivo* counterparts. Additionally, they used the sequencing data to predict that tumor cell lines harboring particular mutations are sensitive to specific classes of drugs[4].

Thus, tumor cell lines are an invaluable resource not only for understanding disease mechanisms, but also for drug design and discovery. The following pages contain a description of the ways our cell lines are organized. Please see the appendix for a full listing of ATCC breast cancer cell line, as well as for lists of cell lines that have been organized according to gene mutation or that are part of a paired tumor/normal set.

Breast Cancer Cell Lines by Gene

Tumor cell lines become more powerful tools for cancer research and drug discovery when the genetic abnormalities that drive their phenotype are known. To aid in the selection of cell lines and tumor cell panels, information regarding four of the major genes associated with breast cancer, obtained from the Catalogue of Somatic Mutations in Cancer (Wellcome Trust Sanger Institute, UK), is detailed below. As an additional resource, the appendix of this resource book provides information about the specific gene mutation, predicted protein sequence, zygosity, and tumor histology of each breast cancer cell line.

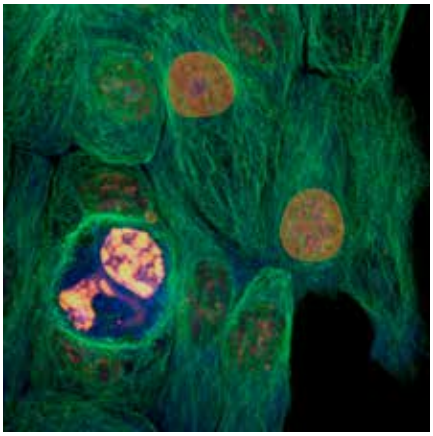
- **CDKN2A** - Cyclin-dependent kinase inhibitor 2A (CDKN2A) is a tumor suppressor gene that encodes at least three different splice variants, two of which can induce arrest at the G1 phase of the cell cycle by inhibiting the CDK4 kinase. Germ-line mutations in this gene are associated with the development of several types of malignancy, including breast cancer[5].
- **PIK3CA** - The phosphatidylinositol-4, 5-bisphophate 3-kinase, catalytic subunit alpha (PIK3CA) gene encodes the p110α catalytic subunit of class I phosphatidylinositol 3-kinases (PI3K). PI3K operates as part of the PI3K/AKT/mTOR pathway to mediate cell proliferation, survival, migration, and vesicular trafficking. Mutations in this gene are frequently associated with breast cancer and are considered a positive prognostic factor[6,7].
- **PTEN** - Phosphatase and tensin homolog (PTEN) is a tumor suppressor gene that encodes for a lipid phosphatase[8]. PTEN primarily exerts its anti-tumorigenic effect by inhibiting the activation of PI3K. However, PTEN may also help regulate genomic stability, cell cycle progression, differentiation, and gene expression[9]. Mutations in this gene are commonly associated with a variety of cancers, including prostate, brain, and breast[8].
- **TP53** - The TP53 gene encodes the tumor suppressor protein p53, which plays a significant role in regulating the cellular response to DNA damage and other cytotoxic stresses. In addition, it plays an important regulatory role in cellular functions including cell cycle arrest, DNA repair, genome stability, apoptosis, cell differentiation, and angiogenesis. Mutations in TP53 are commonly associated with human malignancies and can be found in a significant proportion of human cancers from an array of tissue sources, including breast[10].

See the appendix for a list of the breast cancer cell lines arranged by gene mutation.

Paired Tumor/Normal Cell Lines

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 compare tumor lines to their normal or near normal counterparts.

TUMOR CELL PANELS



The value of individual tumor cell lines is further enhanced by organizing them into groups or "panels" according to their histological or molecular characteristics. Such panels can be used for predicting the cellular response of a particular class of disease to novel therapeutics in a controlled, experimental setting.

ATCC has developed an extensive collection of breast cancer related Tumor Cell Panels to complement our wide array of individual tumor cell lines. Each ATCC Tumor Cell Panel includes low-passage, authenticated tumor cell lines, which have been annotated with genetic mutation data (from the Catalogue of Somatic Mutations in Cancer database, Wellcome Trust Sanger Institute, UK), and collected together in ways that best represent the specific features of this heterogeneous disease. The panels are described below to aid you in selecting the one that best suits your research needs.

The **Comprehensive Breast Cancer Cell Panel (ATCC® No. 30-4500K™)** is a comprehensive set of 45 breast cancer cell lines derived from ATCC master seed stocks. Each panel features a CD containing signed certificates of analyses and product sheets for each individual cell line.

The **Triple Negative Breast Cancer Cell Panels (ATCC® No. TCP-1001™, TCP-1002™, TCP-1003™)** are arranged according to their classification into the following subtypes: (1) basal-like, which includes subtypes basal-like 1 and 2 (BL1 and BL2) and immunomodulatory (IM); (2) mesenchymal-like, which includes the mesenchymal (M) and mesenchymal stem-like (MSL) groups; and, (3) the luminal androgen receptor (LAR) subtype. **Triple-Negative Breast Cancer Panel 1; Basal-like Morphology (ATCC® TCP-1001™)** is composed of nine triple negative breast tumor cell lines that share a basal-like morphology. **Triple-Negative Breast Cancer Panel 2; Mesenchymal & Luminal Morphology (ATCC® TCP-1002™)** is composed of six triple negative breast tumor cell lines that share a mesenchymal-like morphology or a LAR subtype. **Triple Negative Breast Cancer Panel 3 (ATCC® TCP-1003™)** is composed of all the items in Triple Negative Breast Cancer Panel 1 and Triple Negative Breast Cancer Panel 2, plus two triple negative breast cancer cell lines with an unclassified morphology.

The **Breast Cancer Biomarkers Cell Panel (ATCC® No. TCP-1004™)** is comprised of seven cell lines and takes the Tumor Cell Panel concept to the next level by including published biomarker data for each culture in a convenient, printable format. This panel puts biomarker information at the researcher's fingertips, so they can reach a deeper understanding of the mechanisms behind the development and progression of breast cancer.

The **Breast Cancer p53 Hotspot Mutation Cell Panel (ATCC® No. TCP-2010™)** is comprised of eight select cell lines. This cell line panel is designed to help investigators unravel the relationship between TP53 (p53) gene mutations and oncogenesis. ATCC sequenced the p53-mutant cell lines in our collection and arranged them based on their precise mutational profiles. This panel combines cell lines that harbor mutations at different p53 hotspots with appropriate control lines that are either wild-type or null for p53 expression. Additionally, the p53-mutant cell lines included in these panels have mutations that result in translated proteins that are either unable to bind DNA, or that are structurally altered. Thus, this panel allows researchers to perform mechanistic assays at both the gene and protein level.

The **Breast Cancer Mouse Model Cell Panel (ATCC® No. TCP-1005™)** is composed of eight immortalized mouse mammary epithelial cell lines that stably overexpress MEK1 activated mutant (MEKDD), EGFR2/Neu, Myc, or Ha-Ras. The cell lines in this panel have been used successfully to generate mouse models of breast cancer for studying metastasis and EGFR-MEK signaling, oncogenes in cell transformation, and for testing anti-cancer compounds[11-14].

See the appendix for all of the breast cancer cell lines included in each Breast Cancer Tumor Cell Panel

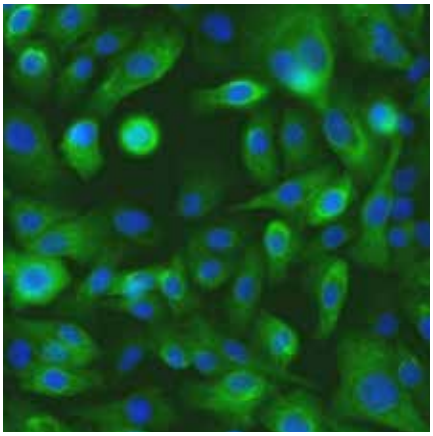
CELL AUTHENTICATION

The value of tumor cell lines continues to grow as large-scale, cell-line sequencing initiatives release massive amount of genomic information that can be used to annotate and associate them with a particular class of tumor. However, the immeasurable potential of annotated tumor cell lines is undermined by the ease with which they can become corrupted by intra- or interspecies contamination.

All ATCC tumor cell lines are rigorously tested to confirm their identity and to rule out both intra- and interspecies contamination. These tests include morphology, karyotyping, cytochrome C oxidase I (COI) gene analysis (a PCR-based assay to detect interspecies contamination), and short tandem repeat (STR) profiling.

hTERT IMMORTALIZED CELLS

ATCC offers a wide variety of cells that have been immortalized through the forced expression of the hTERT component of the telomerase gene. Expression of hTERT allows human cells to maintain the telomere ends of chromosomes and repress replicative senescence. Analysis of numerous hTERT immortalized cell lines indicates these cells have a stable karyotype and retain many of the physiological characteristics of the primary cell, including normal phenotypic marker expression. Additionally, they exhibit normal p53 cell cycle checkpoint control, are non-malignant, contact inhibited, and anchorage dependent. Moreover, they retain normal growth responses to serum and mitogens, require growth factors for proliferation, and do not show changes associated with transformation, such as tumorigenicity or growth in soft agar[15].



ATCC® No. CRL-4010™ stained with a monoclonal pan-cytokeratin antibody (green) and Hoechst dye (blue)

The **human mammary epithelium hTERT-HME1 [ME16C] (ATCC® No. CRL-4010™)** cell line was derived from normal primary mammary epithelial cells. These cells were infected with a retroviral pBabepuro+hTERT vector and cultured in complete growth medium containing puromycin until stable clones were selected[16]. hTERT-HME1 cells have served as normal controls in several studies that sought to unravel the molecular mechanism of breast cancer pathogenesis. For example, Zhang and colleagues used this cell line to show that the BRMS1 gene is expressed in normal cells, but not in metastatic cancer cells. They went on to show that this gene sensitizes breast cancer cells to ATP-induced growth inhibition and apoptosis[17]. In another study, Lee and colleagues used hTERT-HME1 cells to show that certain Caveolin-1 mutations contribute to the pathogenesis of breast cancer by acting in a dominant-negative manner. Thus, hTERT-HME1 cells have contributed to mechanistic studies that have helped drive basic research and that have the potential to inform rational therapeutic design[18].

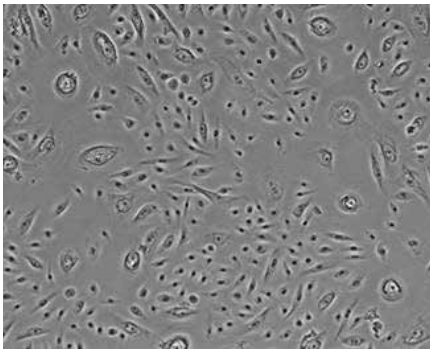
Spectrum of Tools for Cell-based Research

Cell Type	Tissue Phenotype	Genotypic Stability	Proliferative Capacity	Ease-of-Use
Primary Cell Solution	++++	++++	+	++
hTERT Immortalized Cell Lines	+++	+++	++++	++++
Continuous Cell Lines	++	+	++++	++++

ATCC Cell Biologists offer this table for guidance in the cell selection process. In the table, the indicated biological attributes of ATCC cell types were scored on an increasing scale of + to ++++. The scoring is subjective, based on years of collective experience, and does not represent a comprehensive study.

PRIMARY CELLS

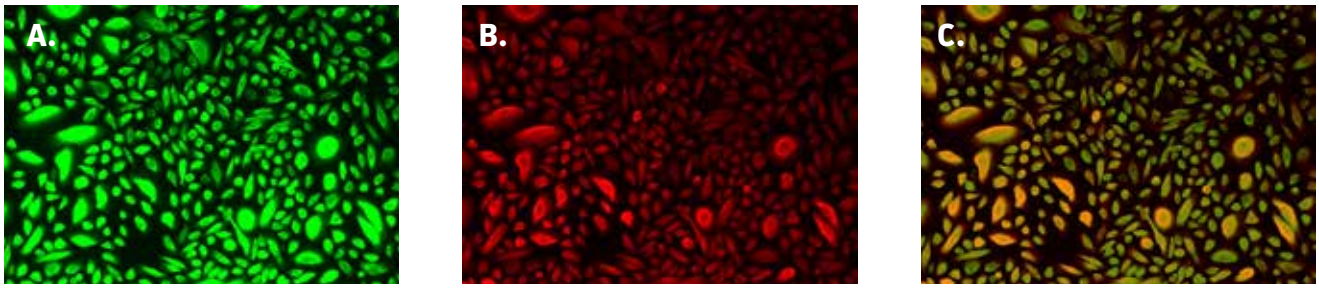
The human mammary gland is a complex tissue composed of milk-producing luminal epithelial cells surrounded by contractile myoepithelial cells; the vast majority of breast cancers originate in these structures. Studies have suggested that continuous cell lines tend towards lineage-restricted profiles that fail to mimic the cellular heterogeneity of either the normal or cancerous mammary gland[19]. Thus, cultures derived directly from the tissue may provide a more representative model for certain applications like the study of oncogenesis or drug discovery.



ATCC® No. PCS-600-010 Normal, Human Primary Mammary Epithelial Cells

Primary cells can be isolated in the laboratory setting, but the isolation process makes primary cell cultures vulnerable to contamination by bacteria or non-epithelial cells. Plus, access to an ethically-derived source of normal human mammary tissue may be difficult to obtain. These issues may be avoided entirely by using Primary Human Mammary Epithelial Cells from ATCC.

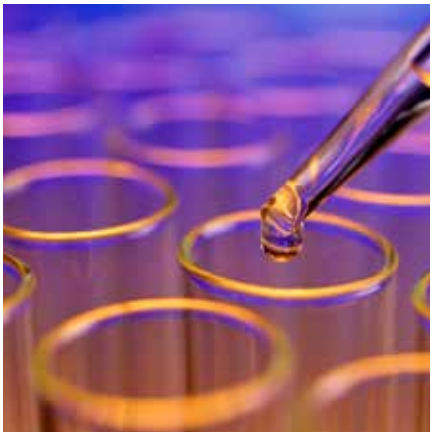
ATCC **Primary Mammary Epithelial Cells ; Normal, Human (ATCC® No. PCS-600-010)** are a mixed population of myoepithelial and luminal epithelial cells. They are cryopreserved at low passage (P2) to ensure high post-thaw viability and plating efficiency. These cells are thoroughly tested to confirm their proliferative capacity and that they are free of microbial contamination. Together with the ATCC Mammary Epithelial Cell Basal Medium (ATCC® No. PCS-600-030) and ATCC Mammary Epithelial Cell Growth kit (ATCC® No. PCS-600-040) they form a complete culture system that you can trust to help you achieve your research objectives.



Primary Mammary Epithelial Cells are a mixed population of CK14 positive myoepithelial cells, and CK14 and CK18 positive luminal epithelial cells. A. CK14-stained Primary Mammary Epithelial Cells, B. CK18-stained Primary Mammary Epithelial Cells, and C. Overlay overlay of A. and B.

CULTURE AND ASSAY REAGENTS

ATCC has the media you need to keep your cells healthy and behaving as expected. All ATCC media are filtered, ready-to-use, and shipped in 500 mL plastic bottles. Our collection includes all the "classic" media formulations, plus media and reagents specially designed to support the culture of mammary cells *in vitro*.



Phenol Red-free Media

Phenol red, which is commonly used as a pH indicator in culture media, bears a structural resemblance to non-steroidal estrogens. Additionally, it has been found to stimulate cell proliferation in a dose- and estrogen receptor-dependent manner[20]. Consequently, phenol red may mask or exaggerate the impact of experimentally applied estrogen, effectively altering experimental results in unforeseeable ways. To help researchers establish more controllable experimental conditions, ATCC now offers phenol red-free mammary epithelial cell basal medium and growth kit.

Every ATCC media product is manufactured to the exact specifications recommended by ATCC cell culture scientists, and rigorously tested to meet the quality and performance standards you've come to expect from ATCC.

Primary Mammary Epithelial Cell Complete Media

The Mammary Epithelial Cell Basal Medium and Growth Kit work together to provide a complete, serum-free growth medium designed to support the proliferation and plating efficiency of epithelial cells derived from normal human breast.

Primary Mammary Epithelial Cell Basal Medium (ATCC® No. PCS-600-030) is a sterile, phenol red-free, liquid tissue culture medium that contains essential and non-essential amino acids, vitamins, other organic compounds, trace minerals, and inorganic salts.

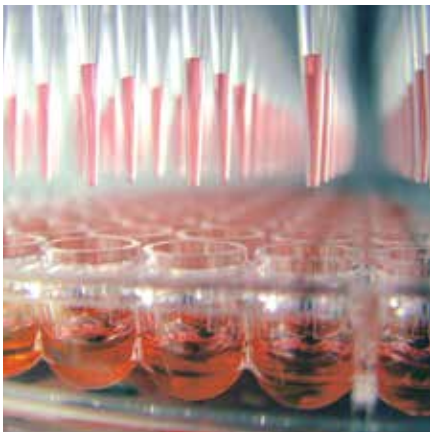
Primary Mammary Epithelial Cell Growth Kit (ATCC® No. PCS-600-040) includes rH-Insulin, L-Glutamine, Epinephrine, Apo-Transferrin, rH-TGFα, Extract P, and Hydrocortisone Hemisuccinate.

CellMatrix Basement Membrane Gel

The extracellular matrix is important for normal maintenance of the breast epithelium and may be involved in oncogenic transformation[2]. In the *in vitro* setting, CellMatrix Gel is used to support a differentiated phenotype in primary epithelial cultures, and it may be used to promote spheroid formation in cancer cell lines. This later feature of CellMatrix may be particularly useful for the screening of anti-cancer drugs. Drugs that are highly effective on cells grown in monolayer cultures are often ineffective when tested in pre-clinical animal models. This is likely due to the fact that cells grown *in vitro* in a monolayer are more accessible to a chemical compound than cells growing *in vivo*, in three-dimensions, within an extracellular matrix. Therefore, CellMatrix Basement Membrane Gel is essential for helping investigators recapitulate the *in vivo* conditions of normal and cancerous cells in an *in vitro* culture model.

CellMatrix Basement Membrane Gel (ATCC® No. ACS-3035) is a soluble, growth factor-reduced basement membrane extract purified from the Engelbreth Holm Swarm tumor. It comprises a mixture of laminin, collagen IV, entactin, and heparin sulfate proteoglycan, and is commonly used to generate physiologically relevant two- and three-dimensional culture conditions. It is supplied as a 12 to 18 mg/mL solution (refer to the product label for the actual concentration) stored in Dulbecco's Modified Eagle's Medium (DMEM) (ATCC® 30-2002) with 10 µg/mL gentamycin, and it has been tested to ensure that it is free from microbial contamination (i.e. bacteria (including mycoplasma), fungi, and 31 other pathogens, including viruses, as demonstrated by PCR. Endotoxin concentrations are less than 8 EU/mL by LAL assay).

MTT and XTT Proliferation Assay Kits



The reduction of tetrazolium salts to an easy-to-measure, colored precipitate is widely accepted as a reliable way to examine cell viability and proliferation. ATCC offers proliferation kits that rely on the reduction of the popular tetrazolium dyes MTT or XTT.

- **MTT Proliferation Kit (ATCC® No. 30-1010K)**

  The yellow tetrazolium MTT (3-(4, 5-dimethylthiazolyl-2)-2, 5-diphenyltetrazolium bromide) is reduced by metabolically active cells, in part by the action of dehydrogenase enzymes, to generate reducing equivalents such as NADH and NADPH. The resulting intracellular formazan derivative can be solubilized and quantified using a spectrophotometer.

  The MTT Cell Proliferation Assay measures the cell proliferation rate and conversely, when metabolic events lead to apoptosis or necrosis, the reduction in cell viability. The number of assay steps has been minimized as much as possible to expedite sample processing. The MTT Reagent yields low background absorbance values in the absence of cells. For each cell type, the linear relationship between cell number and signal produced is established, thus allowing an accurate quantification of changes in the rate of cell proliferation.

- **XTT Proliferation Kit (ATCC® No. 30-1011K)**

  The second generation tetrazolium dye, XTT, can be effectively used in cell proliferation, cytotoxicity, and apoptosis assays[21, 22]. XTT is reduced to a soluble brightly-colored orange derivative by a mix of cellular effectors. The sensitivity of an XTT assay is greatly improved by using the included intermediate electron carrier, PMS (N-methyl dibenzopyrazine methyl sulfate), to help drive XTT reduction and the formation of the formazan derivative.

GROWTH CURVES

Cells grow at different rates in each of the different phases of the growth cycle and the calculated doubling time may be a composite of growth during more than one of these phases. Growth during exponential growth or log phase is fairly constant and reproducible for a given set of growth conditions.

TRANSFECTION REAGENTS

Transfection reagents introduce nucleic acid constructs into eukaryotic cells. Protein synthesis, overexpression mutant DNA constructs, and RNA-targeted knockdown of protein expression are some of the laboratory applications of these reagents. Transfection systems may be used for the transient transfer of nucleic acids, or stable cell lines may be created. The most common transfection system is based on the cationic lipid-based transfection reagent. Due to their positive charge, these transfection reagents interact with the negatively charged backbone of nucleic acids and cell membranes. This effect, coupled with the hydrophobic nature of the associated lipids, allows the cationic lipid-DNA complex to be transported into eukaryotic cells. ATCC offers DNA transfection reagents that are specific for adherent cells and cells cultured in suspension. High transfection efficiencies may be obtained using protocols designed specifically for ATCC cells.

ATCC® TransfeX™ Transfection Reagent

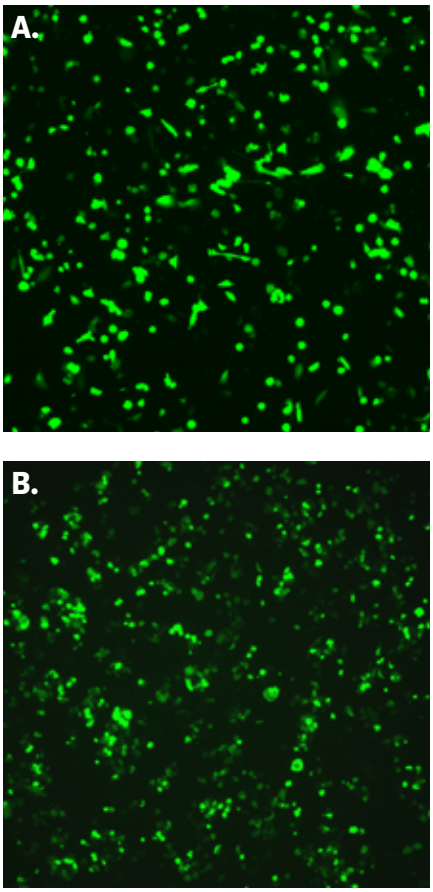
The **ATCC® TransfeX™ Transfection Reagent (ATCC® No. ACS-4005**) has been optimized for use on a wide range of cell types, including cells that are generally difficult to transfect, such as hTERT immortalized cell lines, primary cells, and stem cells. High transfection efficiencies were observed in breast cancer cell lines using TransfeX. MCF-7 cells, MDA-MB-231 cells, and mammary epithelial cells exhibited 50, 70, and 45 percent transfection efficiency, respectively.

ATCC® Gene*XPlus* Transfection Reagent for Suspension Cells

The **ATCC® Gene*XPlus* Transfection Reagent (ATCC® No. ACS-4004)** may be used to efficiently transfect a broad spectrum of cell types. For example, it has been successfully used to deliver high transfection efficiency with low cytotoxicity for primary human mesenchymal stem cells and various primary human endothelial cells, and to transfect notoriously hard-to-transfect cell lines such as THP-1 and Raw 264.7.

ATCC® siFEX™ Transfection Reagent for Gene Knockdown

siFEX™ RNAi Transfection Reagent (ATCC® No. ACS-4006) has been added to the ATCC arsenal of transfection reagents to mediate the transfer of siRNA or miRNA oligonucleotides into cells for efficient gene silencing. **si**FEX™ has been tested in more than 17 cell lines including primary cells and hard-to-transfect cell lines with protocols available online. **si**FEX™ is a cost effective solution amenable to scaling up with low cytotoxicity, as well as high transfection efficiency.



Breast cancer cell lines A. MDA-MB-231 or B. MCF-7 48 hours post-transfection with GFP using TransfeX.

APPENDIX

This Appendix contains a complete listing of ATCC Breast Cancer Tumor Cell Lines and Tumor Cell Panels as of September 2014. Within each of the subheadings of the appendix, the control cells are listed first. The cell lines are next arranged alphabetically: first by pathology, then by tumor source. Finally, the cell lines are organized by organism, and are placed in the following order based on experimental relevance: Human, monkey, mouse, rat, and dog. Where pertinent, information regarding mutations in the gene and the protein sequences is included.

Tumor Cell Lines

Complete List of ATCC Breast Cancer Cell Lines

Tumor Source	Pathology	Organism	Name	ATCC® No.
None	Normal; spontaneously immortalized	Human	MCF-12A	CRL-10782™
None	Normal; fibrocystic disease	Human	MCF-12F	CRL-10783™
None	Normal; chemically immortalized	Human	184A1	CRL-8798™
None	Normal; chemically immortalized	Human	184B5	CRL-8799™
None	Normal	Human	Hs 617.Mg	CRL-7379™*
None	Abnormal	Human	Hs 875.T	CRL7612™*
Primary	Acantholytic squamous cell carcinoma	Human	HCC1806	CRL-2335™
Metastasis; brain	Adenocarcinoma	Human	MDA-MB-361	HTB-27™
Metastasis; cutaneous effusion	Adenocarcinoma	Human	DU4475	HTB-123™
Metastasis; malignant pleural effusion	Adenocarcinoma	Human	AU565 [AU-565]	CRL-2351™
Metastasis; pleural effusion	Adenocarcinoma	Human	MCF7	HTB-22™
Metastasis; pleural effusion	Adenocarcinoma	Human	MDA-MB-231	HTB-26™
Metastasis; pleural effusion	Adenocarcinoma	Human	MDA-MB-415	HTB-128™
Metastasis; pleural effusion	Adenocarcinoma	Human	MDA-MB-436	HTB-130™
Metastasis; pleural effusion	Adenocarcinoma	Human	CAMA-1	HTB-21™
Metastasis; pleural effusion	Adenocarcinoma	Human	SK-BR-3	HTB-30™
Metastasis; pleural effusion	Adenocarcinoma	Human	MDA-MB-231	CRM-HTB-26™
Metastasis; pleural effusion	Adenocarcinoma	Human	UACC-2087	CRL-3180™
Metastasis; pleural effusion	Adenocarcinoma	Human	HCC1428	CRL-2327™
Primary	Adenocarcinoma	Human	Hs 281.T	CRL-7227™
Primary	Adenocarcinoma	Human	Hs 343.T	CRL-7245™
Primary	Adenocarcinoma	Human	Hs 362.T	CRL-7253™
Primary	Adenocarcinoma	Human	MDA-MB-468	HTB-132™
Primary	Adenocarcinoma	Human	UACC-1179	CRL-3127™
Primary	Adenocarcinoma	Human	Hs 274.T	CRL-7222™
Primary	Adenocarcinoma	Human	Hs 739.T	CRL-7477™
Primary	Adenocarcinoma	Human	SMT/2A LNM	CRL-6602™
Primary	Cancer	Human	MDA-kb2	CRL-2713™*
Primary	Cancer	Human	Hs 605.T	CRL-7365™*
Primary	Cancer	Human	Hs 748.T	CRL-7486™
Primary	Cancer	Human	Hs 566(B).T	CRL-7336™*
Primary	Cancer	Human	Hs 606.T	CRL-7368™*
Primary	Cancer	Human	Hs 329.T	CRL-7242™*
Primary	Cancer	Human	Hs 371.T	CRL-7256™*

Please see www.atcc.org for more information about each of the cell lines listed here

Complete List of ATCC Breast Cancer Cell Lines (continued)

Tumor Source	Pathology	Organism	Name	ATCC® No.
Primary	Cancer	Human	Hs 190.T	CRL-7145™*
Primary	Cancer	Human	Hs 344.T	CRL-7246™*
Primary	Cancer	Human	Hs 350.T	CRL-7248™*
Primary	Cancer	Human	Hs 841.T	CRL-7574™*
Primary	Cancer	Human	Hs 849.T	CRL-7583™*
Primary	Cancer	Human	Hs 851.T	CRL-7584™*
Primary	Cancer	Human	Hs 861.T	CRL-7596™*
Metastasis; pericardial effusion	Carcinoma	Human	MDA-MB-453	HTB-131™
Metastasis; pericardial effusion	Carcinoma	Human	MDA-kb2	CRL-2713™
Metastasis; pleural effusion	Carcinoma	Human	MB157	CRL-7721™
Primary	Carcinoma	Human	BT-20	HTB-19™
Primary	Carcinoma	Human	Hs 578Bst	HTB-125™
Primary	Carcinoma	Human	Hs 578T	HTB-126™
Primary	Carcinoma	Human	Hs 579.Mg	CRL-7347™
Metastasis; ascites	Ductal carcinoma	Human	ZR-75-1	CRL-1500™
Metastasis; ascites	Ductal carcinoma	Human	ZR-75-30	CRL-1504™
Metastasis; pleural effusion	Ductal carcinoma	Human	T47D-KBluc	CRL-2865™
Metastasis; pleural effusion	Ductal carcinoma	Human	T-47D	HTB-133™
Metastasis; pleural effusion	Ductal carcinoma	Human	MDA-MB-134-VI	HTB-23™
Metastasis; pleural effusion	Ductal carcinoma	Human	MDA-MB-175-VII	HTB-25™
Primary	Ductal carcinoma	Human	BT-474	HTB-20™
Primary	Ductal carcinoma	Human	UACC-812	CRL-1897™
Primary	Ductal carcinoma	Human	BT-483	HTB-121™
Primary	Ductal carcinoma	Human	BT-549	HTB-122™
Primary	Ductal carcinoma	Human	Hs 574.T	CRL-7345™
Primary	Ductal carcinoma	Human	UACC-893	CRL-1902™
Primary	Ductal carcinoma	Human	HCC38	CRL-2314™
Primary	Ductal carcinoma	Human	HCC70	CRL-2315™
Primary	Ductal carcinoma	Human	HCC202	CRL-2316™
Primary	Ductal carcinoma	Human	HCC1008	CRL-2320™
Primary	Ductal carcinoma	Human	HCC1143	CRL-2321™
Primary	Ductal carcinoma	Human	HCC1187	CRL-2322™
Primary	Ductal carcinoma	Human	HCC1395	CRL-2324™
Primary	Ductal carcinoma	Human	HCC1419	CRL-2326™
Primary	Ductal carcinoma	Human	HCC1500	CRL-2329™
Primary	Ductal carcinoma	Human	HCC1599	CRL-2331™
Primary	Ductal carcinoma	Human	HCC1937	CRL-2336™
Primary	Ductal carcinoma	Human	HCC1954	CRL-2338™
Primary	Ductal carcinoma	Human	HCC2218	CRL-2343™
Primary	Ductal carcinoma	Human	UACC-2648	CRL-3121™
Primary	Fibrocystic disease	Human	MCF 10A	CRL-10317™
Primary	Fibrocystic disease	Human	MCF 10F	CRL-10318™
Primary	Fibrocystic disease	Human	MCF 10-2A	CRL-10781™
Metastasis; axillary lymph node	Infiltrating ductal carcinoma	Human	UACC-3199	CRL-2983™
Primary	Infiltrating ductal carcinoma	Human	Hs 564(E).Mg	CRL-7329™
Primary	Infiltrating ductal carcinoma	Human	Hs309.T	CRL-7236™
Metastasis; pleural effusion	Infiltrating lobular carcinoma	Human	UACC-3133	CRL-2988™
Metastasis; pleural fluid	Inflammatory carcinoma	Human	UAC-732	CRL-3166™

Complete List of ATCC Breast Cancer Cell Lines (continued)

Tumor Source	Pathology	Organism	Name	ATCC® No.
Primary	Medullary carcinoma	Human	MDA-MB-157	HTB-24™
Primary	Metaplastic carcinoma	Human	HCC1569	CRL-2330™
Primary	Paget's disease	Human	SW527 [SW 527, SW-527]	CRL-7940™
Primary	Scirrhous adenocarcinoma	Human	Hs 742.T	CRL-7482™
Primary	Cancer	Monkey	CMMT	CRL-6299™
None	Normal	Mouse	NMuMG	CRL-1636™
None	Normal	Mouse	MM3MG	CRL-6376™*
Primary	Adenocarcinoma	Mouse	JC	CRL-2116™
Primary	Cancer	Mouse	MMT 060562	CCL-51™
Primary	Cancer	Mouse	4T1	CRL-2539™
Primary	Cancer	Mouse	+/+ MGT	CRL-6468™*
Primary	Cancer	Mouse	MM2MT	CRL-6373™*
Primary	Cancer	Mouse	RIIIMT	CRL-6449™*
Primary	Carcinoma	Mouse	EMT6	CRL-2755™
Primary	Hyperplastic alveolar nodules	Mouse	CL-S1	CRL-1615™
Metastasis; pericardial effusion	Papilloma	Mouse	CSMalphabeta6C [CSMab6C]	CRL-8400™
Primary	Papilloma	Mouse	CMH1a	CRL-8399™
Primary	Papilloma	Mouse	CMalphabeta1h [CMab1h]	CRL-8401™
Primary	Tumor	Mouse	C127I	CRL-1616™
Primary	Tumor	Mouse	MM5MTC	CRL-6378™*
Primary	Tumor	Mouse	Rn2T	CRL-6599™*
Primary	Adenocarcinoma	Rat	13762 MAT B III	CRL-1666™
Primary	Adenocarcinoma	Rat	NMU	CRL-1743™
Primary	Adenocarcinoma	Rat	RBA	CRL-1747™
Primary	Adenocarcinoma	Rat	Hs 741.T	CRL-7480™
Primary	Cancer	Rat	Rn1T	CRL-6598™*
Primary	Tumor	Rat	LA7	CRL-2283™
None	Normal	Dog	CF37.Mg	CRL-6230™*
None	Normal	Dog	CF38.Mg	CRL-6231™*
Primary	Cancer	Dog	CF41.Mg	CRL-6232™*
Primary	Tumor	Dog	CF34.Mg	CRL-6228™*

*Part of the NBL Cell Line Collection. This cell line is neither produced nor fully characterized by ATCC. We do not guarantee that it will maintain a specific morphology, purity, or any other property upon passage.

Breast Cancer Cell Lines by Gene

CDKN2A

Tumor Source	Pathology	Zygosity	Gene Sequence†	Protein Sequence†	Name	ATCC® No.
Primary	Acantholytic squamous cell carcinoma	Homozygous	c.1471del471	p.0?	HCC1806	CRL-2335™
Metastasis; brain	Adenocarcinoma	Homozygous	c.156G>C	p.M521	MDA-MB-361	HTB-27™
Metastasis; pleural effusion	Adenocarcinoma	Homozygous	c.1471del471	p.0?	MCF7	HTB-22™
Metastasis; pleural effusion	Adenocarcinoma	Homozygous	c.1471del471	p.0?	MDA-MB-231	HTB-26™

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

CDKN2A

Tumor Source	Pathology	Zygosity	Gene Sequence†	Protein Sequence†	Name	ATCC® No.
Primary	Carcinoma	Homozygous	c.1471del471	p.0?	BT-20	HTB-19™
Primary	Ductal carcinoma	Homozygous	c.1471del471	p.0?	HCC38	CRL-2314™
Primary	Ductal carcinoma	Homozygous	c.1471del471	p.0?	HCC1395	CRL-2324™

PIK3CA

Tumor Source	Pathology	Zygosity	Gene Sequence†	Protein Sequence†	Name	ATCC® No.
Metastasis; brain	Adenocarcinoma	Heterozygous	c.1633G>A	p.E545K	T-47D	HTB-27™
Metastasis; pleural effusion	Adenocarcinoma	Heterozygous	c.1633G>A	p.E545K	MDA-MB-361	HTB-27™
Metastasis; pleural effusion	Carcinoma	Heterozygous	c.3140A>G	p.H1047R	MCF7	HTB-131™
Primary	Carcinoma	Heterozygous	c.1616C>G	p.P539R	BT-20	HTB-19™
Primary	Carcinoma	Heterozygous	c.3140A>G	p.H1047R	BT-20	HTB-19™
Primary	Ductal carcinoma	Heterozygous	c.3140A>G	p.H1047R	HCC1954	CRL-2338™
Primary	Ductal carcinoma	Heterozygous	c.3140A>G	p.H1047R	UACC-893	CRL-1902™
Primary	Ductal carcinoma	Heterozygous	c.333G>C	p.K111N	BT-474	HTB-20™
Primary	Ductal carcinoma	Heterozygous	c.3140A>G	p.H1047R	MDA-MB-453	HTB-133™

PTEN

Tumor Source	Pathology	Zygosity	Gene Sequence†	Protein Sequence†	Name	ATCC® No.
Metastasis; pleural effusion	Adenocarcinoma	Homozygous	c.253+1G>T	p.?	MDA-MB-468	HTB-132™
Metastasis; pleural effusion	Adenocarcinoma	Heterozygous	c.274G>C	p.D92H	CAMA-1	HTB-21™
Metastasis; pleural effusion	Adenocarcinoma	Homozygous	c.407G>A	p.C136Y	MDA-MB-415	HTB-128™
Metastasis; pleural effusion	Adenocarcinoma	Heterozygous	c.831_834delCTTC	p.T277fs*13	CAMA-1	HTB-21™
Primary	Ductal carcinoma	Homozygous	c.270delT	p.F90fs*9	HCC70	CRL-2315™
Primary	Ductal carcinoma	Homozygous	c.635_1212del578	p.N212fs*1	HCC1395	CRL-2324™
Primary	Papillary ductal carcinoma	Homozygous	c.823delG	p.V275fs*1	BT-549	HTB-122™

TP53

Tumor Source	Pathology	Zygosity	Gene Sequence†	Protein Sequence†	Name	ATCC® No.
Primary	Acantholytic squamous cell carcinoma	Homozygous	c.766_767insAA	p.T256fs*90	HCC1806	CRL-2335™
Metastasis; pleural effusion	Adenocarcinoma	Homozygous	c.524G>A	p.R175H	AU565	CRL-2351™
Metastasis; pleural effusion	Adenocarcinoma	Homozygous	c.580C>T	p.L194F	T-47D	HTB-133™
Metastasis; pleural effusion	Adenocarcinoma	Homozygous	c.707A>G	p.Y236C	MDA-MB-415	HTB-128™
Metastasis; pleural effusion	Adenocarcinoma	Homozygous	c.818G>A	p.R273H	MDA-MB-468	HTB-132™
Metastasis; pleural effusion	Adenocarcinoma	Homozygous	c.839G>A	p.R280K	MDA-MB-231	HTB-26™
Metastasis; pleural effusion	Adenocarcinoma	Homozygous	c.839G>C	p.R280T	CAMA-1	HTB-21™
Primary	Carcinoma	Homozygous	c.394A>C	p.K132Q	BT-20	HTB-19™
Primary	Ductal carcinoma	Homozygous	c.1024C>T	p.R342*	UACC-893	CRL-1902™
Primary	Ductal carcinoma	Homozygous	c.220_226delGCCCCTG	p.A74fs*47	HCC1419	CRL-2326™

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

TP53 (continued)

Tumor Source	Pathology	Zygosity	Gene Sequence†	Protein Sequence†	Name	ATCC® No.
Primary	Ductal carcinoma	Homozygous	c.322_324delGGT	p.G108del	HCC1187	CRL-2322™
Primary	Ductal carcinoma	Homozygous	c.488A>G	p.Y163C	HCC1954	CRL-2338™
Primary	Ductal carcinoma	Homozygous	c.524G>A	p.R175H	HCC1395	CRL-2324™
Primary	Ductal carcinoma	Homozygous	c.659A>G	p.Y220C	HCC1419	CRL-2326™
Primary	Ductal carcinoma	Homozygous	c.673-2A>T	p.?	HCC1599	CRL-2331™
Primary	Ductal carcinoma	Homozygous	c.743G>A	p.R248Q	HCC70	CRL-2115™
Primary	Ductal carcinoma	Homozygous	c.743G>A	p.R248Q	HCC1143	CRL-2321™
Primary	Ductal carcinoma	Homozygous	c.818G>T	p.R273L	HCC38	CRL-2314™
Primary	Ductal carcinoma	Homozygous	c.847C>T	p.R283C	HCC2218	CRL-2343™
Primary	Ductal carcinoma	Homozygous	c.853G>A	p.E285K	BT-474	HTB-20™
Primary	Ductal carcinoma	Homozygous	c.916C>T	p.R306*	HCC1937	CRL-2336™
Metastasis; pleural effusion	Medullary carcinoma	Homozygous	c.261_286delAGCCCCCTCCTGGCCCCTGTCATCTT	p.A88fs*52	MDA-MB-157	HTB-24™
Primary	Metaplastic carcinoma	Homozygous	c.880G>T	p.E294*	HCC1569	CRL-2330™
Primary	Papillary ductal carcinoma	Homozygous	c.747G>C	p.R249S	BT-549	HTB-122™

Other genes

Tumor Source	Pathology	Gene	Zygosity	Gene Sequence†	Protein Sequence†	Name	ATCC® No.
Metastasis; pleural effusion	Adenocarcinoma	BRAF	Heterozygous	c.1391G>T	p.G464V	MDA-MB-231	HTB-26™
Metastasis; pleural effusion	Adenocarcinoma	RAS	Heterozygous	c.38G>A	p.G13D	MDA-MB-231	HTB-26™
Metastasis; pleural effusion	Adenocarcinoma	RB1	Homozygous	c.265_2787del2523	p.?	MDA-MB-468	HTB-132™
Metastasis; pleural effusion	Adenocarcinoma	SMAD4	Homozygous	c.1_1659del1659	p.0?	MDA-MB-468	HTB-132™
Metastasis; ascites	Ductal carcinoma	PIK3R1	Homozygous	c.335_427del93	p.?	ZR-75-30	CRL-1504™
Metastasis; skin	Ductal carcinoma	APC	Homozygous	c.4729G>T	p.E1577*	DU4475	HTB-123™
Metastasis; skin	Ductal carcinoma	BRAF	Heterozygous	c.1799T>A	p.V600E	DU4475	HTB-123™
Metastasis; skin	Ductal carcinoma	RB1	Homozygous	c.265_2787del2787	p.0?	CAMA-1	HTB-21™
Primary	Papillary ductal carcinoma	RB1	Homozygous	c.265_607del343	p.?	BT-549	HTB-122™

Paired Tumor/Normal Cell Lines

Tumor Cell Lines Tumor Source	Pathology	Name	ATCC® No.	Normal Pairing Tissue Source	Pathology	Name	ATCC® No.
Mammary gland	Ductal carcinoma	Hs574.T	CRL-7345™	Skin	Normal	Hs574.Sk	CRL-7346™
Mammary gland	Ductal carcinoma	Hs578T	HTB-126™	Mammary gland	Normal	Hs578Bst	HTB-125™
Mammary gland	Ductal carcinoma	HCC1954	CRL-2338™	B lymphoblast	Normal	HCC1954 BL	CRL-2339™
Mammary gland	Ductal carcinoma	HCC38	CRL-2314™	B lymphoblast	Normal	HCC38 BL	CRL-2346™
Mammary gland	Ductal carcinoma	HCC1143	CRL-2321™	B lymphoblast	Normal	HCC1143 BL	CRL-2362™
Mammary gland	Ductal carcinoma	HCC1187	CRL-2322™	B lymphoblast	Normal	HCC1187 BL	CRL-2323™
Mammary gland	Ductal carcinoma	HCC1395	CRL-2324™	B lymphoblast	Normal	HCC1395 BL	CRL-2325™
Mammary gland	Ductal carcinoma	HCC1599	CRL-2331™	B lymphoblast	Normal	HCC1599 BL	CRL-2332™
Mammary gland	Ductal carcinoma	HCC1937	CRL-2336™	B lymphoblast	Normal	HCC1937 BL	CRL-2337™
Mammary gland	Ductal carcinoma	HCC2218	CRL-2343™	B lymphoblast	Normal	HCC2218 BL	CRL-2363™
Metastasis; lymph node	Ductal carcinoma	HCC10008	CRL-2320™	B lymphoblast	Normal	HCC1007 BL	CRL-2319™

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

Tumor Cell Panels

Comprehensive Breast Cancer Cell Panel (ATCC® No. 30-4500K™)

Tumor Source	Pathology	Organism	Name	ATCC® No.
None	Normal; spontaneously immortalized	Human	MCF-12F	CRL-10782™
None	Normal; chemically-transformed	Human	184B5	CRL-8799™
Primary	Acantholytic squamous cell carcinoma	Human	HCC1806	CRL-2335™
Metastasis; brain	Adenocarcinoma	Human	MDA-MB-361	HTB-27™
Metastasis; skin	Adenocarcinoma	Human	DU4475	HTB-123™
Metastasis; malignant pleural effusion	Adenocarcinoma	Human	AU565 [AU-565]	CRL-2351™
Metastasis; pleural effusion	Adenocarcinoma	Human	CAMA-1	HTB-21™
Metastasis; pleural effusion	Adenocarcinoma	Human	HCC1428	CRL-2327™
Metastasis; pleural effusion	Adenocarcinoma	Human	MCF7	HTB-22™
Metastasis; pleural effusion	Adenocarcinoma	Human	MDA-MB-231	HTB-26™
Metastasis; pleural effusion	Adenocarcinoma	Human	MDA-MB-415	HTB-128™
Metastasis; pleural effusion	Adenocarcinoma	Human	MDA-MB-436	HTB-130™
Metastasis; pleural effusion	Adenocarcinoma	Human	SK-BR-3	HTB-30™
Primary	Adenocarcinoma	Human	MDA-MB-468	HTB-132™
Primary	Cancer	Human	MDA-kb2	CRL-2713™
Metastasis; pericardial effusion	Carcinoma	Human	MDA-MB-453	HTB-131™
Primary	Carcinoma	Human	BT-20	HTB-19™
Primary	Carcinoma	Human	Hs 578Bst	HTB-125™
Primary	Carcinoma	Human	Hs 578T	HTB-126™
Metastasis; ascites	Ductal carcinoma	Human	ZR-75-1	CRL-1500™
Metastasis; ascites	Ductal carcinoma	Human	ZR-75-30	CRL-1504™
Metastasis; pleural effusion	Ductal carcinoma	Human	MDA-MB-134-VI	HTB-23™
Metastasis; pleural effusion	Ductal carcinoma	Human	MDA-MB-175-VII	HTB-25™
Metastasis; pleural effusion	Ductal carcinoma	Human	T-47D	HTB-133™
Primary	Ductal carcinoma	Human	BT-474	HTB-20™
Primary	Ductal carcinoma	Human	BT-483	HTB-121™
Primary	Ductal carcinoma	Human	BT-549	HTB-122™
Primary	Ductal carcinoma	Human	HCC38	CRL-2314™
Primary	Ductal carcinoma	Human	HCC70	CRL-2315™
Primary	Ductal carcinoma	Human	HCC202	CRL-2316™
Primary	Ductal carcinoma	Human	HCC1187	CRL-2322™
Primary	Ductal carcinoma	Human	HCC1395	CRL-2324™
Primary	Ductal carcinoma	Human	HCC1419	CRL-2326™
Primary	Ductal carcinoma	Human	HCC1599	CRL-2331™
Primary	Ductal carcinoma	Human	HCC1937	CRL-2336™
Primary	Ductal carcinoma	Human	HCC1954	CRL-2338™
Primary	Ductal carcinoma	Human	HCC2157	CRL-2340™
Primary	Ductal carcinoma	Human	HCC2218	CRL-2343™
Primary	Ductal carcinoma	Human	UACC-812	CRL-1897™
Primary	Ductal carcinoma	Human	UACC-893	CRL-1902™
Primary	Fibrocystic disease	Human	MCF 10A	CRL-10317™
Primary	Fibrocystic disease	Human	MCF 10F	CRL-10318™
Metastasis; pericardial effusion	Medullary carcinoma	Human	HCC1569	CRL-2330™
Primary	Medullary carcinoma	Human	MDA-MB-157	HTB-24™
Primary	Carcinoma	Mouse	HCC1500	CRL-2329™

Triple-Negative Breast Cancer Panel 1; Basal-Like Morphology (ATCC® No. TCP-1001™)

Tumor Source	Pathology	Subtype*	Mutant Gene	Zygosity	Gene Sequence†	Protein Sequence†	Name	ATCC® No.
Metastasis; pleural effusion	Adenocarcinoma	BL1	PTEN RB1 SMAD4 TP53	Homozygous Homozygous Homozygous Homozygous	c.253+1G>T c.265_2787del2523 c.1_1659del1659 c.818G>A	p.? p.? p.0? p.R273H	MDA-MB-468	HTB-132™
Metastasis; skin	Carcinoma	IM	APC BRAF MAP2K4 RB1	Homozygous Heterozygous Homozygous Homozygous	c.4729G>T c.1799T>A c.1_1200del1200 c.1_2787del2787	p.E1577* p.V600E p.0? p.0?	DU4475	HTB-123™
Primary	Primary acantholytic squamous cell carcinoma	IM	CDKN2A KDM6A STK11 TP53	Homozygous Homozygous Homozygous Homozygous	c.1_471del471 c.444_564del121 c.1109_1302del194 c.766_767insAA	p.0? p.0 p.? p.T256fs*90	HCC1806	CRL-2335™
Primary	Ductal carcinoma	BL1	BRCA2 TP53	Homozygous Homozygous	c.4550_4559del10 c.673-2A>T	p.K1517fs*23 p.?	HCC1599	CRL-2331™
Primary	Ductal carcinoma	BL1	BRCA TP53	Homozygous Homozygous	c.5266_5267insC c.916C>T	p.Q1756fs*74 p.R306	HCC1937	CRL-2336™
Primary	Ductal carcinoma	BL1	TP53	Homozygous	c.743G>A	p.R248Q	HCC1143	CRL-2321™
Primary	Ductal carcinoma	BL1	CDKN2A TP53	Homozygous Homozygous	c.1_471del471 c.818G>T	p.0? p.R273L	HCC38	CRL-2314™
Primary	Ductal carcinoma	BL2	PTEN TP53	Homozygous Homozygous	c.270delT c.743G>A	p.F90fs*9 p.R248Q	HCC70	CRL-2315™
Primary	Ductal carcinoma	IM	TP53	Homozygous	c.322_324delGGT	p.G108del	HCC1187	CRL-2322™

Triple-Negative Breast Cancer Panel 2; Mesenchymal & Luminal Morphology (ATCC® No. TCP-1002™)

Tumor Source	Pathology	Subtype*	Mutant Gene	Zygosity	Gene Sequence†	Protein Sequence†	Name	ATCC® No.
Metastasis; pleural effusion	Adenocarcinoma	MSL	BRAF CDKN2A KRAS NF2 TP53	Heterozygous Homozygous Heterozygous Homozygous Homozygous	c.1391G>T c.1_471del471 c.38G>A c.691G>T c.839G>A	p.G464V p.0? p.G13D p.E231* p.R280K	MDA-MB-231	HTB-26™
Metastasis; pleural effusion	Adenocarcinoma	MSL	BRCA1 RB1	Homozygous Homozygous	c.5277+1G>A c.607_608ins227	p.? p.G203fs*9	MDA-MB-436	HTB-130™
Metastasis; pleural effusion	Carcinoma	LAR	"CDH1 PIK3CA"	Homozygous Heterozygous	c.1913G>A c.3140A>G	p.W638* p.H1047R	MDA-MB-453	HTB-131™
Primary	Carcinoma	MSL	CDKN2A HRAS PIK3R1 TP53	Homozygous Heterozygous Homozygous Homozygous	c.1_471del471 c.35G>A c.1358_1359insTAA c.469G>T	p.0? p.G12D p.N453_ T454insN p.V157F	Hs578T	HTB-126™
Primary	Ductal Carcinoma	M	PTEN RB1 TP53	Homozygous Homozygous Homozygous	c.823delG c.265_607del343 c.747G>C	p.V275fs*1 p.? p.R249S	BT-549	HTB-122™
Metastasis; pleural effusion	Medullary carcinoma	MSL	NF1 TP53	Homozygous Homozygous	c.8253_8268del16 c.261_286delAGCC CCCTCCTGGCCCCT GTCATCTT	p.S2751fs*27 p.A88fs*52	MDA-MB-157	HTB-24™

Triple Negative Breast Cancer Cell Panel 3 (ATCC® No. TCP-1003™): Cell lines in Panels 1 and 2 above plus the two unclassified cell lines listed below

Tumor Source	Pathology	Subtype*	Mutant Gene	Zygosity	Gene Sequence†	Protein Sequence†	Name	ATCC® No.
Primary	Carcinoma	-	CDKN2A PIK3CA PIK3CA TP53	Homozygous Heterozygous Heterozygous Homozygous	c.1_471del471 c.1616C>G c.3140A>G c.394A>C	p.0? p.P539R p.H1047R p.K132Q	BT-20	HTB-19™
Primary	Primary ductal carcinoma	-	BRCA1 CDKN2A PTEN TP53	Homozygous Homozygous Homozygous Homozygous	c.5251C>T c.1_471del471 c.635_1212del578 c.524G>A	p.R1751* p.0? p.N212fs*1 p.R175H	HCC1395	CRL-2324™

*These subtypes are classified as: (1) Basal-like, including subtypes BL1 (basal-like 1), BL2 (basal-like 2), and IM (immunomodulatory); (2) Mesenchymal-like, including subtypes M (mesenchymal) and MSL (mesenchymal stem-like); and, (3) LAR (luminal androgen receptor).

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

Breast Cancer Biomarkers Cell Line Panel 1 (ATCC® No. TCP-1004™)

Tumor Source	Pathology	Age	Positive Markers	Negative Markers	Other Significant Features	Patient Treatment	Name	ATCC® No.
Metastasis; pleural effusion	Adenocarcinoma	62	HER-2/neu	ER, PR, EGFR, MASPIN, DSC3	P53 R213X mutation and MASPIN promoter methylation have been reported for this line.	Adriamycin, Cytoxan, Methotrexate, Tamoxifen	UACC-1179	CRL-3127™
Metastasis; pleural effusion	Adenocarcinoma	35	HER-2/neu, PR	ER, EGFR	Drug resistant cell line to cyclin D kinase 4/6 inhibitor and HER-2	Vinblastine, Adriamycin, Cytoxan	UACC-732	CRL-3166™
Metastasis; pleural effusion	Adenocarcinoma	53	EGFR	ER, PR, HER-2/ Neu, vimentin MASPIN, DSC3	P53 V216M mutation has been reported in this cell line. It has also been reported that the MASPIN promoter is not methylated.	Cyclophosphamide, Methotrexate, 5-fluorouracil, Thymidine phophorylase, Tamoxifen	UACC-2087	CRL-3180™
Metastasis; pleural effusion	Ductal carcinoma	63	HER-2/neu, BMP-3	ER (very low), PR, EGFR, MASPIN, DSC3, BMP-2	MASPIN promoter methylation has been reported for this line	Surgery only	UACC-3133	CRL-2988™
Metastasis; axillary nodes	Infiltrating ductal carcinoma	58	EGFR	ER, PR, HER-2/ Neu	Methylated BRCA-1 promoter	Cytoxan, Adriamycin, 5-fluorouracil, Tamoxifen, Mitoxantrone, Vinblastine	UAAC-3199	CRL-2983™
Primary	Infiltrating ductal carcinoma	43	HER-2/neu	ER, PR, EGFR, P-glycoprotein	N/A	Vinblastine, Adriamycin, Cytoxan, Cyclophosphamide, Methotrexate, 5-fluorouracil	UACC-812	CRL-1897™
Primary	Infiltrating ductal carcinoma	57	HER-2/neu	ER, PR, EGFR, P-glycoprotein, MASPIN	MASPIN promoter methylation has been reported for this line	None	UACC-893	CRL-1902™

Breast Cancer Mouse Model Cell Line Panel (ATCC® No. TCP-1005™)

Tumor Source	Overexpression	Significant Features	Name	ATCC® No.
Mouse mammary gland	Empty vector	The cells were immortalized from mouse mammary epithelial cell line with an empty vector. They are useful as a control.	Eph4Ev	CRL-3063™
Mouse mammary gland	EGFR/Neu	The cells are a mouse mammary tumor cell line derived from Neu-initiated transgenic mice.	NF639	CRL-3090™
Mouse mammary gland	Ha-Ras	The cells are a mouse mammary tumor cell line derived from Ha-ras-initiated transgenic mice.	Ac 711	CRL-3092™
Mouse mammary gland	Mutant MEK1	The cells stable overexpression of glu-glu epitope-tagged MEK constant activated mutant: Asp218/Asp222 MEK1 phosphorylation site mutant (MEKDD).	B-MEKDD 116	CRL-3069™
Mouse mammary gland	Mutant MEK1	The cells were derived from a mouse primary breast tumor. The cells stably overexpress glu-glu epitope-tagged MEK1 constitutively activated mutant (MEKDD).	Eph4 1424	CRL-3071™
Mouse mammary gland	Mutant MEK1	The cells were derived from a mouse breast tumor metastasis to lung. The cells stably overexpress glu-glu epitope-tagged MEK1 constitutively activated mutant (MEKDD).	Eph4 1424.1	CRL-3209™
Mouse mammary gland	Mutant MEK1	The cells were derived from a mouse breast tumor metastasis to kidney. The cells stably overexpress glu-glu epitope tagged MEK1 constitutively activated mutant (MEKDD).	Eph4 1424.2	CRL-3210™
Mouse mammary gland	Myc	The cells are a mouse mammary tumor cell line derived from c-myc-initiated transgenic mice.	M158	CRL-3086™

Breast Cancer p53 Hotspot Mutation Cell Panel (ATCC® No. TCP-2010™)

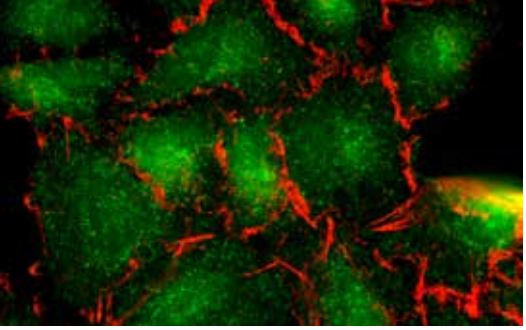
Tumor Source	Pathology	p53 Status	Zygosity	Gene Sequence†	Protein Sequence†	Name	ATCC® No.
Metastasis; brain	Adenocarcinoma	WT	-	-	-	MDA-MB-361	HTB-27™
Metastasis; pleural effusion	Adenocarcinoma	MUT	Homozygous	c.524G>A	p.R175H	AU565	CRL-2351™
Metastasis; pleural effusion	Adenocarcinoma	MUT	Homozygous	c.524G>A	p.R175H	SK-BR-3	HTB-30™
Metastasis; pleural effusion	Adenocarcinoma	MUT	Homozygous	c.818G>A	p.R273H	MDA-MB-468	HTB-132™
Metastasis; pleural effusion	Ductal carcinoma	WT	-	-	-	MDA-MB-175-VII	HTB-25™
Primary	Ductal carcinoma	MUT	Homozygous	c.743G>A	p.R248Q	HCC70	CRL-2315™
Primary	Ductal carcinoma	MUT	Homozygous	c.747G>C	p.R249S	BT-549	HTB-122™
Primary	Ductal carcinoma	MUT	Homozygous	c.818G>T	p.R273L	HCC38	CRL-2314™

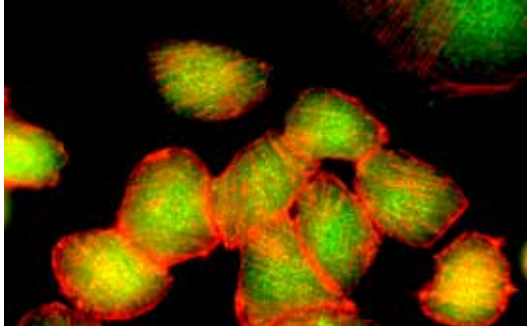
The mutation data was obtained from the Sanger Institute Catalogue Of Somatic Mutations In Cancer web site, http://www.sanger.ac.uk/cosmic Bamford et al (2004) The COSMIC (Catalogue of Somatic Mutations in Cancer) database and website. Br J Cancer, 91,355-358. ATCC and The Sanger Institute provide these data in good faith, but make no warranty, express or implied, nor assumes any legal liability or responsibility for any purpose for which the data are used.

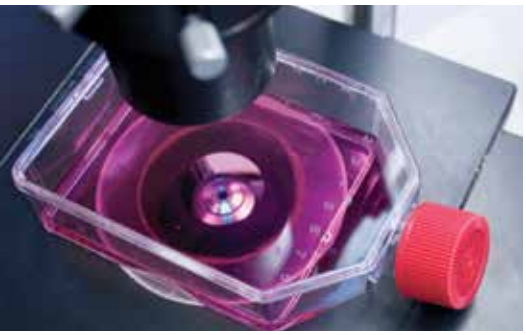
†For a description of the sequence variation nomenclature please refer to: *den Dunnen JT and Antonarakes SE (2000), Hum. Mutat. 15:7-12.*

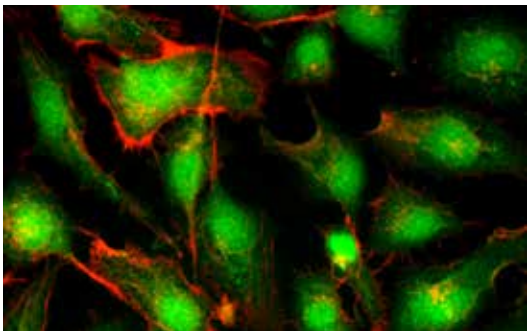
REFERENCES

1. Jemal A, *et al.* Global cancer statistics. CA Cancer J Clin 61:69-90, 2011.
2. Holliday DL, Speirs V. Choosing the right cell line for breast cancer research. Breast Cancer Res 13(4):215, 2011.
3. Grigoriadis A, *et al.* Molecular characterisation ofcell line models for triple-negative breast cancers. BMC Genomics 13:619, 2012.
4. Barretina J, *et al.* The Cancer Cell Line Encyclopedia enables predictive modelling of anticancer drug sensitivity. Nature 483(7391):603-7, 2013.
5. Borg A, *et al.* High frequency of multiple melanomas and breast and pancreas carcinomas in CDKN2A mutation-positive melanoma families. J Natl Cancer Inst 92(15):1260-6, 2000.
6. Janku F, *et al.* PI3K/AKT/mTOR inhibitors in patients with breast and gynecologic malignancies harboring PIK3CA mutations. J Clin Oncol 30(8):777-82, 2012.
7. Cizkova M, *et al.* PIK3CA mutation impact on survival in breast cancer patients and in ERalpha, PR and ERBB2-based subgroups. Breast Cancer Res 14(1):R28, 2012.
8. Li J, *et al.* PTEN, a putative protein tyrosine phosphatase gene mutated in human brain, breast, and prostate cancer. Science 275(5308):1943-7, 1997.
9. Carracedo A, Alimonti A, Pandolfi PP. PTEN level in tumor suppression: how much is too little? Cancer Res 71(3):629-33, 2011.
10. May P, May E. Twenty years of p53 research: structural and functional aspects of the p53 protein. Oncogene 18(53):7621-36, 1999.
11. Amundadottir LT, Leder P. Signal transduction pathways activated and required for mammary carcinogenesis in response to specific oncogenes. Oncogene 16(6):737-46, 1998.
12. Bais MV, *et al.* Role of Nanog in the maintenance of marrow stromal stem cells during post natal bone regeneration. Biochem Biophys Res Commun 417:211-6, 2012.
13. Pinkas J, Leder P. MEK1 signaling mediates transformation and metastasis of EpH4 mammary epithelial cells independent of an epithelial to mesenchymal transition. Cancer Res 62:4781-90, 2002.
14. Pinkas J, Martin SS, Leder P. Bcl-2-mediated cell survival promotes metastasis of EpH4 betaMEKDD mammary epithelial cells. Mol Cancer Res 2(10):551-6, 2004.
15. Dalerba P, *et al.* Reconstitution of human telomerase reverse transcriptase expression rescues colorectal carcinoma cells from *in vitro* senescence: evidence against immortality as a constitutive trait of tumor cells. Cancer Res 65:2321-9, 2005.
16. Herbert BS, Wright WE, Shay JW. p16(INK4a) inactivation is not required to immortalize human mammary epithelial cells. Oncogene 21:7897-900, 2002.
17. Zhang Y, *et al.* BRMS1 sensitizes breast cancer cells to ATP-induced growth suppression. Biores Open Access 2:77-83 2013.
18. Lee H, *et al.* Caveolin-1 mutations (P132L and null) and the pathogenesis of breast cancer: caveolin-1 (P132L) behaves in a dominant-negative manner and caveolin-1 (-/-) null mice show mammary epithelial cell hyperplasia. Am J Pathol 161:1357-69 2002.
19. Keller PJ, *et al.* Mapping the cellular and molecular heterogeneity of normal and malignant breast tissues and cultured cell lines. Breast Cancer Res 12:R87, 2010.
20. Berthois Y, Katzenellenbogen JA, Katzenellenbogen BS. Phenol red in tissue culture media is a weak estrogen: implications concerning the study of estrogen-responsive cells in culture. Proc Natl Acad Sci U S A 83:2496-500, 1986.
21. Scudiero DA, *et al.* Evaluation of a soluble tetrazolium/formazan assay for cell growth and drug sensitivity in culture using human and other tumor cell lines. Cancer Res 48:4827-33, 1988.
22. Berridge MV, Herst P, Tan AS. Tetrazolium dyes as tools in cell biology: new insights into their cellular reduction. Biotechnol Annu Rev 11:127-52, 2005.









CB-0415-06

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