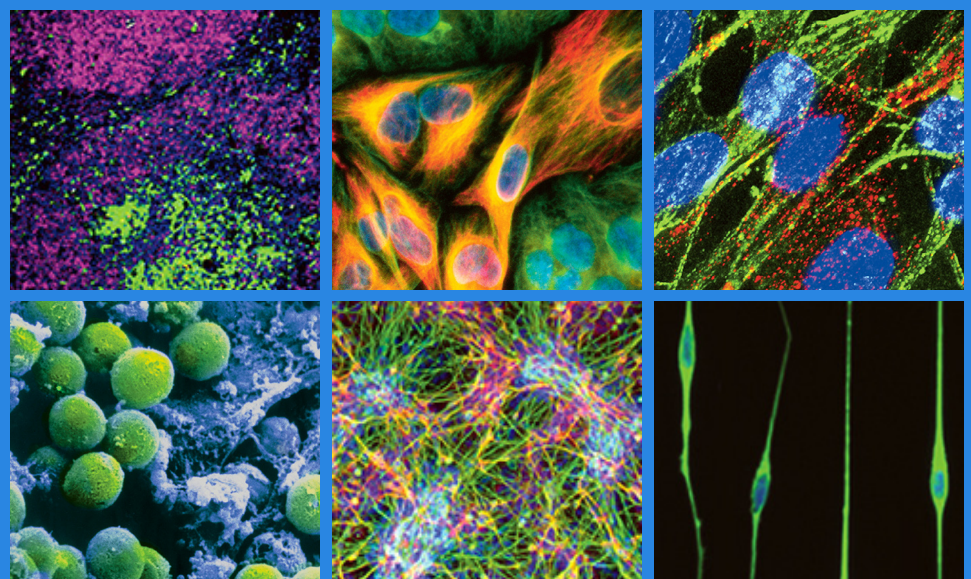
Tangible Materials Licensing Catalogue



Edition 3

Memorial Sloan Kettering Cancer Center

What's New

Memorial Sloan Kettering possesses an extensive collection of tangible research materials, which are available for licensing for research or commercial purposes. These materials are managed by MSK's Office of Technology Development.

With this 3rd Edition of its *Tangible Materials Licensing Catalogue*, MSK offers a comprehensive and expanded selection of more than a dozen categories of cell lines derived from cancer patients, including melanoma, renal cancer, neuroblastoma, and lung cancer.

Marking another important development, MSK's *Tangible Materials Licensing Catalogue* also now includes a broad range of antibodies, mouse models, organoids, and PDX models. A number of these materials have not been previously publicized for licensing purposes. Together with the rest of the portfolio described in this catalogue, they offer promising potential for commercial entities and academic-research institutions alike.

This 3rd Edition of MSK's *Tangible Materials Licensing Catalogue* is new and different in another important respect. The catalogue now includes a copy of MSK's Express License for selected cell lines included in this catalogue; the license is linked directly to each of these marketing sheets.

MSK's non-exclusive, non-negotiable Express License saves time and effort for our tangible materials licensing partners. It's worth noting that MSK now offers multiple payment options for tangible materials licenses: online credit card transactions (recommended method) or payment by invoice.

This is all part of our effort to make the process of tangible material licensing as quick and user-friendly as possible. We will periodically publish new and expanded editions of MSK's *Tangible Materials Licensing Catalogue*. Meanwhile, to view a list of other MSK technologies available for licensing, including therapeutics, diagnostics, vaccines, and medical devices, please see here.

Table of Contents

Anti-CD45.1 Mouse Monoclonal Antibody (Clone A-20)

Antigen: Mouse CD45.1 (Ly5.1)

Clone Name: A-20

Isotype: Mouse IgG2a (Kappa Light Chain)

Application(s)*: Flow Cytometry, Immunofluorescence Microscopy, Immunoprecipitation

Reactivity*: Mouse
*As reported in the literature and other commercial supplier websites

Description

Clone A-20 reacts with CD45 (Leukocyte Common Antigen) on leukocytes of mouse strains that express the CD45.1 alloantigen (e.g., RIII, SJL/J, STS/A, DA). It has been reported not to react with leukocytes from mouse strains expressing the CD45.2 alloantigen.

Source

This antibody was derived in 1981 by injection of thymocytes and splenocytes from SJL mice into A.SW mice. Splenocytes from these A.SW mice were fused with NS-1 cells to generate hybridomas.

Inventors

- Edward Boyse, MD, formerly of Memorial Sloan Kettering
- Fung-Win Shen, PhD

Key References

- Shen FW (1981) Monoclonal antibodies to mouse lymphocyte differentiation alloantigens. *Monoclonal Antibodies and T-Cell Hybridomas: Perspectives and Technical Advances*. Hämmerling GJ, Hämmerling U and Kearney JF, editors. Elsevier/North-Holland Biomedical Press, Amsterdam. 25-31 (ISBN: 9780444803511)
- Yakura H et al. (1983) On the function of Ly-5 in the regulation of antigen-driven B cell differentiation. Comparison and contrast with Lyb-2. *Journal of Experimental Medicine* 157: 1077-1088 (PubMed ID: 6220106)

MSK Tracking Code: SK2003-077

Anti-CD45.2 Mouse Monoclonal Antibody (Clone 104-2)

Antigen: Mouse CD45.2 (Ly5.2)

Clone Name: 104-2

Isotype: Mouse IgG2a (Kappa Light Chain)

Application(s)*: Flow Cytometry, Immunofluorescence Microscopy, Immunoprecipitation

Reactivity*: Mouse
*As reported in the literature and other commercial supplier websites

Description

Clone 104-2 reacts with CD45 (Leukocyte Common Antigen) on leukocytes of mouse strains that express the CD45.2 alloantigen, including A, AKR, BALB/c, CBA/Ca, CBA/J, C3H/He, C57BL, C57BR, C57L, C58, DBA/1, DBA/2, NZB, SWR, and 129. It has been reported not to react with leukocytes from mouse strains expressing the CD45.1 alloantigen.

Source

This antibody was derived in 1981 by injection of thymocytes and splenocytes from B10.S mice into SJL mice. Splenocytes from these SJL mice were fused with NS-1 cells to generate hybridomas.

Inventors

- Edward Boyse, MD, formerly of Memorial Sloan Kettering
- Fung-Win Shen, PhD

Key References

- Shen FW (1981) Monoclonal antibodies to mouse lymphocyte differentiation alloantigens. Monoclonal Antibodies and T-Cell Hybridomas: Perspectives and Technical Advances. Hämmerling GJ, Hämmerling U and Kearney JF, editors. Elsevier/North-Holland Biomedical Press, Amsterdam. 25-31 (ISBN: 9780444803511)
- Yakura H et al. (1983) On the function of Ly-5 in the regulation of antigen-driven B cell differentiation. Comparison and contrast with Lyb-2. Journal of Experimental Medicine 157: 1077-88 (PubMed ID: 6220106)

MSK Track Code: SK2003-077

Anti-NK1.1 Mouse Monoclonal Antibody (Clone PK136)

Antigen: Mouse NK1.1 (CD161, NKR-P1C, Ly-55)

Clone Name: PK136

Isotype: Mouse IgG2a (Kappa Light Chain)

Application(s)*: Flow Cytometry, Immunoprecipitation, Immunohistochemistry, Immunofluorescence

Reactivity*: Mouse
*As reported in the literature and other commercial supplier websites

Description

Clone PK136 recognizes mouse NK1.1, a cell surface antigen expressed by natural killer cells and a subset of T cells in the NK1.1 mouse strains including CE, C57BL/6, FVB/N, and NZB. NK1.1 is not expressed by NK cells from the following mouse strains: 129, A, AKR, BALB/c, C3H, CBA, and SJL.

Source

This antibody was derived in 1984 by injection of splenocytes (enriched for NK-1-positive cells) and bone marrow cells from CE mice into (C3H x BALB/c) F1 mice. Splenocytes from these mice were then fused with Sp2/0-Ag14 cells to generate hybridomas.

Inventors

- Gloria C. Koo, PhD, formerly at Memorial Sloan Kettering
- JoAnne R. Peppard, formerly at Memorial Sloan Kettering

Key References

- Koo GC and Peppard JR (1984) Establishment of monoclonal anti-Nk-1.1 antibody. *Hybridoma* 3: 301-303 (PubMed ID: 6500587)
- Koo GC et al. (1986) The NK-1.1(-) mouse: a model to study differentiation of murine NK cells. *Journal of Immunology*. 137: 3742-3747 (PubMed ID: 3782794)
- Reichlin A and Yokoyama WM (1998) Natural killer cell proliferation induced by anti-NK1.1 and IL-2. *Immunology and Cell Biology* 76: 143-152 (PubMed ID: 9619484)
- Kung SK et al. (1999) The NKR-P1B gene product is an inhibitory receptor on SJL/J NK cells. *Journal of Immunology* 162: 5876-5887 (PubMed ID: 10229823)

MSK Track Code: SK 787

Anti-TRP1 Mouse Monoclonal Antibody (Clone TA99)

Antigen: Human TRP1 (TYRP1, PAA, gp75)

Clone Name: TA99

Isotype: Mouse IgG2a

Application(s): Immunocytochemistry, Immunohistochemistry, Immunoprecipitation, Western Blot

Reactivity*: Mouse, Human
*As reported in the literature and other commercial supplier websites

Description

Clone TA99 is a mouse monoclonal antibody that reacts with tyrosinase-related protein 1 (TRP1), a 75kDa differentiation-related human glycoprotein (gp75), formerly referred to as pigmentation-associated antigen (PAA). It is expressed by pigmented melanoma cells and cultured melanocytes. TRP1 is involved in the pigmentation machinery of melanocytes and can be used as a differentiation marker.

Source

This antibody was derived in 1985 by injection of whole human melanoma cells (SK-MEL-23) into mice. Splenocytes from these immunized mice were fused with NS-1 cells to generate hybridomas producing anti-TRP1 antibodies.

Inventors

- Francisco X. Real, MD, PhD, formerly at Memorial Sloan Kettering
- Lloyd J. Old, MD, former William E. Snee Chair in Cancer Immunology, Memorial Sloan Kettering; former Director, New York Branch, Ludwig Institute for Cancer Research
- Timothy M. Thomson, MD, PhD

Key References

- Thomson TM et al. (1985) Pigmentation-Associated Glycoprotein of Human Melanomas and Melanocytes: Definition with a Mouse Monoclonal Antibody. *Journal of Investigative Dermatology* 85: 169-174 (PubMed ID: 3926906)
- Bevaart L et al. (2006) The high-affinity IgG receptor, FcgammaRI, plays a central role in antibody therapy of experimental melanoma. *Cancer Research* 66: 1261-1264 (PubMed ID: 16452176)

MSK Track Code: SK 413

Mre11-Petrini Antibody

Description

Armenian hamster hybridoma clone 15B8.1E7.6 produces antibodies directed against mMre11. Mre11 is a component of the Mre11 complex, which plays a central role in double-strand break (DSB) repair, DNA recombination, DNA damage signaling through ATM, maintenance of telomere integrity, and meiosis.

Source

Raised against a GST-tagged mMre11 peptide (aa68-608) in Armenian hamster.

Inventors

- MSK Antibody and Bioresource Core Facility, MSK
- John Petrini, PhD, Laboratory Head, Molecular Biology Program, MSK

Key References

Kim JH, Grosbart M, Anand R, Wyman C, Cejka P, Petrini JH. The Mre11-Nbs1 Interface Is Essential for Viability and Tumor Suppression.Cell Rep. 2017 Jan 10;18(2):496-507. doi: 10.1016/j.celrep.2016.12.035. PMID: 28076792

CAMA-1: Human Breast Cancer Cell Line

Description

CAMA-1 is a luminal-type human breast cancer cell line that displays rounded morphology in adherent tissue culture. These cells are considered Her2-negative and estrogen-receptor/progesterone-receptor (ER/PR)-positive. They are responsive to estrogen and sensitive to growth inhibition by tamoxifen. The CAMA-1 cells have an in-frame mutation in the E-cadherin gene, resulting in a truncated, non-functional protein. In addition, they have oncogenic mutations in PTEN and p53 and amplification of the cyclin D1 gene.

Source

This cell line was established in 1975 from the pleural effusion of a 51-year-old Caucasian female with malignant adenocarcinoma of the breast.

Lead Inventor

Jorgen Fogh, PhD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Fogh J et al. (1977) Absence of HeLa cell contamination in 169 cell lines derived from human tumors. *Journal of the National Cancer Institute* 58: 209-214 (PubMed ID: 833871)
- Ji H et al. (1994) Absence of transforming growth factor-beta responsiveness in the tamoxifen growth-inhibited human breast cancer cell line CAMA-1. *Journal of Cellular Biochemistry* 54: 332-342 (PubMed ID: 8200913)
- van Horssen R et al. (2012) E-cadherin promotor methylation and mutation are inversely related to motility capacity of breast cancer cells. *Breast Cancer Research and Treatment* 136: 365-377 (PubMed ID: 23053649)

MSK Tracking Code: SK 926

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SK-BR-3: Human Breast Cancer Cell Line

Description

SK-BR-3 is a human breast cancer cell line that overexpresses the Her2 (Neu/ErbB-2) gene product. These cells display an epithelial morphology in tissue culture and are capable of forming poorly differentiated tumors in immunocompromised mice. The SK-BR-3 cells and products derived from it are used often as positive controls in assays for Her2. In addition, the cell line is also a useful preclinical model to screen for therapeutic agents targeting Her2 and to delineate mechanisms of resistance to Her2-targeted therapies.

Source

This cell line was established in 1970 from the pleural effusion of a 43-year-old Caucasian female with malignant adenocarcinoma of the breast.

Inventors

- Lloyd J. Old, MD, former William E. Snee Chair in Cancer Immunology, Memorial Sloan Kettering Cancer Center; former Director, New York Branch, Ludwig Institute for Cancer Research
- Germain Trempe, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

Fogh J et al. (1977) One hundred and twenty-seven cultured human tumor cell lines producing tumors in nude mice. *Journal of the National Cancer Institute* 59: 221-226 (PubMed ID: 327080)

MSK Tracking Code: SK 808

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SK1041: Organ-Tropic Metastatic Breast Cancer Cell Lines

Description

This invention is a panel of human breast cancer cell lines selected to metastasize to specific organs. The cell lines are useful both for studying the biological mechanisms of breast cancer metastasis and for screening compounds for anti-metastatic activity.

Source

The SK1041 cell lines were derived from the human breast cancer cell line MDA-MB-231 following multiple rounds of *in vivo* selection in immunodeficient mice. They exhibit unique metastatic capacities compared to the parental MDA-MB-231 line, including organ-selective homing, distinct transcriptional profiles, and more aggressive phenotypes.

The panel includes lung-, bone-, brain-, and adrenal-selective metastatic derivatives in addition to cell lines with increased capacity for tumor self-seeding, a process in which circulating tumor cells return to and grow in the primary tumor, promoting tumor progression and further metastasis. Subsets of these populations have been engineered to express reporter plasmids, including a novel triple-modality reporter that permits nuclear, fluorescent, and bioluminescence imaging in a single experimental model.

Advantages

- Pure clonal populations of organ-tropic metastatic cells permit the selective study and comparison of biological mechanisms mediating metastasis to specific organs.
- *In vivo* metastatic lesions develop twice as fast and with a three-fold increase in penetrance compared to parental cell line (~6 weeks with ~90% penetrance vs. ~11 weeks with ~30% penetrance), reducing time and cost for each experiment.
- Aggressive phenotype allows easy detection of metastatic lesions by imaging and histochemical methods.
- These cell lines can be used for both *in vivo* and *in vitro* modeling (i.e. trans-well, Matrigel migration, etc.) of metastasis.

Lead Inventor

Joan Massagué, PhD, Director, Sloan Kettering Institute, and Laboratory Head, Cancer Biology & Genetics Program, MSK

Key References

- Cailleau R, et al. (1974) J Natl Cancer Inst. Sep;53(3):661-74, PMID: 4412247.
- Kang Y, et al. (2003) Cancer Cell. June;3(6):537-49, PMID 12842083.
- Minn AJ, et al. (2005) J Clin Invest. Jan;115(1):44-55, PMID: 15630443.
- Bos PD, et al. (2009) Nature. Jun 18;459(7249):1005-9. Epub 2009 May 6, PMID 19421193.
- Kim MY, et al. (2009) Cell. Dec 24;139(7):1315-26, PMID: 20064377.

HT-3: Human Cervical Cancer Cell Line

Description

HT-3 is a human cervical carcinoma cell line that grows in adherent culture. Although this cell line was initially classified as human papillomavirus (HPV) DNA negative, subsequent studies revealed that the cells harbor HPV30 DNA in their genome. The HT-3 cells have a homozygous mutation in the *TP53* gene, resulting in the expression of the transactivation-defective, dominant negative form of the protein. These cells form tumors when injected subcutaneously into immunocompromised mice.

Source

This cell line was established in 1963 from a metastatic site (lymph node) in a 53-year-old Caucasian female.

Lead Inventor

- Jorgen Fogh, PhD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Fogh J et al. (1977) One hundred and twenty-seven cultured human tumor cell lines producing tumors in nude mice. *Journal of the National Cancer Institute* 59: 221-226 (PubMed ID: 327080)
- Naeger LK et al. (1999) Bovine papillomavirus E2 protein activates a complex growth-inhibitory program in p53-negative HT-3 cervical carcinoma cells that includes repression of cyclin A and cdc25A phosphatase genes and accumulation of hypophosphorylated retinoblastoma protein. *Cell Growth & Differentiation* 10: 413-422 (PubMed ID: 10392903)
- Xiao X et al (2012) Metformin impairs the growth of liver kinase B1-intact cervical cancer cells. *Gynecologic Oncology* 127: 249-255 (PubMed ID: 22735790)

MSK Tracking Code: SK 784-01

HT-29: Human Colorectal Adenocarcinoma Cell Line

Description

HT-29 is a human colorectal adenocarcinoma cell line with epithelial morphology. These cells are sensitive to the chemotherapeutic drugs 5-fluorouracil and oxaliplatin, which are standard treatment options for colorectal cancer. In addition to being a xenograft tumor model for colorectal cancer, the HT-29 cell line is also used as an *in-vitro* model to study absorption, transport, and secretion by intestinal cells. Under standard culture conditions, these cells grow as a nonpolarized, undifferentiated multilayer. Altering culture conditions or treating the cells with various inducers, however, results in a differentiated and polarized morphology, characterized by the redistribution of membrane antigens and development of an apical brush-border membrane.

Source

This cell line was established in 1964 from the primary tumor of a 44-year-old Caucasian female with colorectal adenocarcinoma.

Lead Inventor

Jorgen Fogh, PhD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Fogh J et al. (1977) One hundred and twenty-seven cultured human tumor cell lines producing tumors in nude mice. *Journal of the National Cancer Institute* 59: 221-226 (PubMed ID: 327080)
- Cohen E at al. (1999) Induced differentiation in HT29, a human colon adenocarcinoma cell line. *Journal of Cell Science* 112: 2657-2666 (PubMed ID: 10413674)
- Nautiyal J et al. (2011) Combination of dasatinib and curcumin eliminates chemo-resistant colon cancer cells. *Journal of Molecular Signaling* 6: 7 (PubMed ID: 21774804)

MSK Tracking Code: SK 809

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SK-CO-1: Human Colorectal Adenocarcinoma Cell Line

Description

SK-CO-1 is a human colorectal adenocarcinoma cell line that displays epithelial morphology and grows in adherent tissue culture. In culture, these cells are capable of invading through an extracellular matrix, such as Matrigel. SK-CO-1 cells do not form tumors when injected into immunocompromised mice, and rarely form colonies in soft agar. These cells have oncogenic mutations in K-Ras (G12V) and adenomatous polyposis coli (APC) proteins.

Source

This cell line was established in 1972 from a metastatic site (ascites) in a 65-year-old Caucasian male with colorectal adenocarcinoma.

Inventors

- Lloyd J. Old, MD, former William E. Snee Chair in Cancer Immunology, Memorial Sloan Kettering Cancer Center; former Director, New York Branch, Ludwig Institute for Cancer Research
- Germain Trempe, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Fogh J et al. (1977) Absence of HeLa cell contamination in 169 cell lines derived from human tumors. *Journal of the National Cancer Institute* 58: 209-214 (PubMed ID: 833871)
- Trainer DL et al. (1988) Biological characterization and oncogene expression in human colorectal carcinoma cell lines. *International Journal of Cancer* 41: 287-296 (PubMed ID: 3338874)

MSK Tracking Code: SK2010-072

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SK-MM-1: Human Multiple Myeloma Cell Line

Description

SK-MM-1 is a multiple myeloma cell line that grows in suspension culture. These cells display plasmacytoid morphology and have a doubling time of approximately 32 hours. SK-MM-1 cells do not express the Epstein-Barr virus nuclear antigen. These cells express the pan-B-cell marker B1 and the late B-cell/plasma cell marker BL3, but do not express any T-lymphocyte, myeloid, or early B-lymphocyte markers. SK-MM-1 cells secrete kappa light chains, but do not secrete any heavy chains.

Source

This cell line was established in 1981 from immature plasma cells in the bone marrow of a 51-year-old male, of unknown ethnicity, with plasma cell leukemia.

Lead Inventor

Alan N. Houghton, MD, Attending Physician, Department of Medicine, Memorial Hospital; and Member, Immunology Program, Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Eton O et al. (1989) Establishment and characterization of two human myeloma cell lines secreting kappa light chains. *Leukemia* 3: 729-735 (PubMed ID: 2506399)

MSK Tracking Code: SK 440

SK-MM-2: Human Multiple Myeloma Cell Line

Description

SK-MM-2 is a multiple myeloma cell line that grows in suspension culture. These cells display plasmacytoid morphology and have a doubling time of approximately 60 hours. SK-MM-2 cells do not express the Epstein-Barr virus nuclear antigen. These cells express the pan-B-cell marker B1 and the late B-cell/plasma cell markers BL3, OKT10, and PCA-1, but do not express any T-lymphocyte, myeloid, or early B-lymphocyte markers. These cells secrete kappa light chains, but do not secrete any heavy chains. In addition, SK-MM-2 cells also express elevated levels of cyclin D1 mRNA.

Source

This cell line was established in 1982, from a leukapheresis sample of peripheral blood, from a 54-year-old male, of unknown ethnicity, with plasma cell leukemia.

Lead Inventor

Alan N. Houghton, MD, Attending Physician, Department of Medicine, Memorial Hospital; and Member, Immunology Program, Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Eton O et al. (1989) Establishment and characterization of two human myeloma cell lines secreting kappa light chains. *Leukemia* 3: 729-735 (PubMed ID: 2506399)
- Sáez B et al. (2007) Simultaneous translocations of FGFR3/MMSET and CCND1 into two different IGH alleles in multiple myeloma: lack of concurrent activation of both proto-oncogenes. *Cancer Genetics and Cytogenetics* 175: 65-68 (PubMed ID: 17498561)

MSK Tracking Code: SK1989-002

SK-HEP-1: Human Hepatic Adenocarcinoma Cell Line

Description

SK-HEP-1 is an immortal, human hepatic adenocarcinoma cell line that grows in adherent culture. This cell line is capable of forming tumors in immunocompromised mice. SK-HEP-1 cells in culture have been shown to produce fibronectin and functionally active alpha-1 protease inhibitor. In addition, they constitutively produce Interleukin-1.

Source

This cell line was established in 1971 from the ascites fluids of a 52-year-old Caucasian male with adenocarcinoma of the liver.

Inventors

- Germain Trempe, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center
- Jorgen Fogh, PhD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Fogh J et al. (1977) One hundred and twenty-seven cultured human tumor cell lines producing tumors in nude mice. *Journal of the National Cancer Institute* 59: 221-226 (PubMed ID: 327080)
- Glasgow JE et al. (1984) Fibronectin synthesized by a human hepatoma cell line. *Cancer Research* 44: 3022-3028 (PubMed ID: 6327032)
- Wang L et al. (2012) A novel monoclonal antibody to fibroblast growth factor 2 effectively inhibits growth of hepatocellular carcinoma xenografts. *Molecular Cancer Therapeutics* 11: 864-872 (PubMed ID: 22351746)

Comments

An alternative hypothesis regarding the origin of the SK-HEP-1 cells was presented by Heffelfinger and colleagues, who claim that these cells do not display properties of hepatocytes and are of endothelial origin (PubMed ID: 1371504).

MSK Tracking Code: SK1980-535

Calu-1: Human Lung Cancer Cell Line

Description

Calu-1 is a non-small-cell lung cancer (NSCLC) cell line that grows in adherent culture and displays epithelial morphology. These cells express wildtype LKB1, wildtype EGFR, and mutant K-Ras (G12C). In addition, they lack expression of both p53 (homozygous deletion) and FHIT (Fragile Histidine Triad) tumor-suppressor proteins. The Calu-1 cells are intrinsically resistant to erlotinib, an EGFR tyrosine kinase inhibitor used in the treatment of NSCLC patients. These cells are capable of forming tumors in immunocompromised mice.

Source

This cell line was established in 1971 from a metastatic site (pleura) in a 47-year-old Caucasian male with epidermoid carcinoma of the lung.

Inventors

- Germain Trempe, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center
- Jorgen Fogh, PhD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Fogh J et al. (1977) One hundred and twenty-seven cultured human tumor cell lines producing tumors in nude mice. *Journal of the National Cancer Institute* 59: 221-226 (PubMed ID: 327080)
- Cavazzoni A et al. (2007) Effect of inducible FHIT and p53 expression in the Calu-1 lung cancer cell line. *Cancer Letters* 246: 69-81 (PubMed ID: 16616810)

MSK Tracking Code: SK 784

Calu-3: Human Lung Cancer Cell Line

Description

Calu-3 is a non-small-cell lung cancer cell line that grows in adherent culture and displays epithelial morphology. These cells have constitutively active ErbB2/Her2 due to amplification of the *ERBB2* gene. They express wildtype EGFR and mutant K-Ras (G13D). In addition, they harbor mutations in *TP53* and *CDKN2A* genes. The Calu-3 cells are sensitive to erlotinib (EGFR tyrosine kinase inhibitor) and cetuximab (a monoclonal antibody that blocks ligand binding to EGFR and prevents downstream signaling), two commonly used drugs targeting ErbB receptors. These cells are capable of forming tumors in immunocompromised mice.

Source

This cell line was established in 1975 from a metastatic site (pleural effusion) in a 25-year-old Caucasian male with adenocarcinoma of the lung.

Inventors

- Germain Trempe, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center
- Jorgen Fogh, PhD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Fogh J et al. (1977) One hundred and twenty-seven cultured human tumor cell lines producing tumors in nude mice. *Journal of the National Cancer Institute* 59: 221-226 (PubMed ID: 327080)
- Cavazzoni A et al. (2012) Combined use of anti-ErbB monoclonal antibodies and erlotinib enhances antibody-dependent cellular cytotoxicity of wild-type erlotinib-sensitive NSCLC cell lines. *Molecular Cancer* 11: 91 (PubMed ID: 23234355)
- Blanco R et al. (2009) A gene-alteration profile of human lung cancer cell lines. *Human Mutation* 30: 1199-1206 (PubMed ID: 19472407)

MSK Tracking Code: SK1980-533

SK-LU-1: Human Lung Cancer Cell Line

Description

SK-LU-1 is a lung adenocarcinoma cell line that displays epithelial morphology and grows in adherent culture. This cell line expresses mutant K-Ras (G12D) and has homozygous deletions in the *CDH6* and *CDKN2A* genes. These cells do not express the enzyme telomerase reverse transcriptase (hTERT) and consequently lack telomerase activity. This correlates with significantly reduced tumorigenicity *in vitro* and *in vivo.* These cells, however, display characteristics of alternative telomere lengthening (ALT) mechanisms (i.e., heterogeneity of lengthening of telomeres and the presence of distinct nuclear structures called ALT-associated promyelocytic leukemia bodies). The SK-LU-1 cells do not form tumors when injected into immunocompromised mice.

Source

This cell line was established in 1969 from a 60-year-old Caucasian female with adenocarcinoma of the lung.

Lead Inventor

Chester M. Southam, MD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Fogh J et al. (1977) Absence of HeLa cell contamination in 169 cell lines derived from human tumors. *Journal of the National Cancer Institute* 58: 209-214 (PubMed ID: 833871)
- Lehman TA et al. (1991) p53 mutations, ras mutations, and p53-heat shock 70 protein complexes in human lung carcinoma cell lines. *Cancer Research* 51: 4090-4096 (PubMed ID: 1855224)
- Brachner A et al. (2006) Telomerase- and alternative telomere lengthening-independent telomere stabilization in a metastasis-derived human non-small cell lung cancer cell line: effect of ectopic hTERT. *Cancer Research* 66: 3584-3592 (PubMed ID: 16585183)

MSK Tracking Code: SK2005-049

SK-MES-1: Lung Cancer Cell Line

Description

SK-MES-1 is a human lung cancer cell line that displays epithelial morphology and grows as monolayers in tissue culture. These cells exhibit a cytokeratin expression pattern typical of simple epithelia (i.e., CK7, CK8, CK18, and CK19), and similar to that found in adenocarcinomas. In addition, the expression of Lamins A, B, and C is readily detected in these cells. The SK-MES-1 cells are known to form tumors in immunocompromised mice.

Source

This cell line was established in 1970 from a metastatic site (pleural effusion) in a 65-year-old Caucasian male with squamous cell carcinoma of the lung.

Inventors

- Lloyd J. Old, MD, former William E. Snee Chair in Cancer Immunology, Memorial Sloan Kettering Cancer Center; former Director, New York Branch, Ludwig Institute for Cancer Research
- Germain Trempe, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Fogh J et al. (1977) Absence of HeLa cell contamination in 169 cell lines derived from human tumors. *Journal of the National Cancer Institute* 58: 209-214 (PubMed ID: 833871)
- Blobel GA et al. (1984) Cytokeratins in normal lung and lung carcinomas. I. Adenocarcinomas, squamous cell carcinomas and cultured cell lines. *Virchows Archiv, Cell Pathology Including Molecular Pathology* 45: 407-429 (PubMed ID: 6203212)

MSK Tracking Code: SK2009-091

HTB-106. Human Carcinoma Cell Line Derived from a Lung Metastatic Site

Description

This a human malignant embryonal carcinoma cell line derived from a lung metastatic site. Karyotype characterization reveals (P13) hypotriploid (+A2, +A3, +B, +C, +E, +F, -A1) with abnormalities including acrocentric fragmentation and secondary constrictions.
It is blood type A; Rh+.

Source

This cell line was established from a metastatic site (lung) in a 22-year-old Caucasian male.

Lead Inventor

- Jorgen Fogh, PhD, former at Sloan Kettering Institute, MSK

Key References

- Fogh J, et al. Absence of HeLa cell contamination in 169 cell lines derived from human tumors. J. Natl. Cancer Inst. 58: 209-214, 1977. PubMed: 833871
- Faust JB, Meeker TC. Amplification and expression of the bcl-1 gene in human solid tumor cell lines. Cancer Res. 52: 2460-2463, 1992. PubMed: 1568216
- Fogh J. Cultivation, characterization, and identification of human tumor cells with emphasis on kidney, testis, and bladder tumors. Natl. Cancer Inst. Monogr. 49: 5-9, 1978. PubMed: 571047
- Hay RJ, Caputo JL, Macy, ML, Eds. (1992) ATCC Quality Control Methods for Cell Lines. 2nd edition, Published by ATCC.
- Caputo, J. L., Biosafety procedures in cell culture. J. Tissue Culture Methods 11:223-227, 1988.
- Fleming, D.O., Richardson, J. H., Tulis, J.J. and Vesley, D., (1995) Laboratory Safety: Principles and Practice. Second edition, ASM press, Washington, DC.
- Biosafety in Microbiological and Biomedical Laboratories, 5th ed. HHS. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention. Washington DC: U.S. Government Printing Office; 2007. The entire text is available online.

PCNSL-MSK: Primary CNS Lymphoma (Brain) Cell Line

Description

This is a primary CNS Lymphoma (brain) cell line available for licensing. It is fully genomically annotated.

Source

This cell line was established in 2014 from a tumor/tissue derived from a 64-year-old man.

Inventors

- Christian Grommes, MD, Assistant Attending, Department of Neurology, MSK
- Ingo Mellinghoff, MD, Vice Chair for Research, Department of Neurology, and Laboratory Head, Human Oncology & Pathogenesis Program, MSK
- Vivian Tabar, MD, Associate Attending, Department of Neurosurgery, and Vice Chair, Research and Education, MSK

Restrictions

This cell line is available for licensing but it may not be distributed to third parties.

HT-144: Human Melanoma Cell Line

Description

HT-144 is a malignant human melanoma cell line that displays aneuploid fibroblastic morphology and grows in adherent tissue culture. This cell line has been reported to be nonpermissive for human cytomegalovirus (HCMV). HT-144 cells form xenograft tumors when injected into immunocompromised mice. These cells contain a mutation in the ATM gene, resulting in the expression of a truncated protein, which causes increased sensitivity to UVB and ionizing radiation compared to other melanoma cell lines. The HT-144 cells also express mutant B-Raf (V600E).

Source

This cell line was established in 1966 from a metastatic site (subcutaneous tissue) in a 29-year-old Caucasian male with malignant melanoma.

Inventors

- Jorgen Fogh, PhD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center
- Germaine Trempe, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Fogh J et al. (1977) One hundred and twenty-seven cultured human tumor cell lines producing tumors in nude mice. *Journal of the National Cancer Institute* 59: 221-226 (PubMed ID: 327080)
- Smith JD (1986) Human cytomegalovirus: demonstration of permissive epithelial cells and nonpermissive fibroblastic cells in a survey of human cell lines. *Journal of Virology* 60: 583-588 (PubMed ID: 3021992)
- Ramsay J et al. (1998) Radiosensitive melanoma cell line with mutation of the gene for ataxia telangiectasia. *British Journal of Cancer* 77: 11-14 (PubMed ID: 9459139)
- Chen B et al. (2012) BRAFV600E negatively regulates the AKT pathway in melanoma cell lines. *PLoS One* 7: e42598 (PubMed ID: 22880048)

MSK Track Code: SK1980-544

Malme-3M: Human Melanoma Cell Line

Description

Malme-3M is a malignant human melanoma cell line that displays fibroblast-like morphology and grows in mixed culture (adherent-suspension). This cell line has been shown to be dependent upon micropthalmia-associated transcription factor (MITF) activity for growth and survival. Malme-3M cells form tumors when injected into immunocompromised mice. These cells express mutant B-Raf (V600E) and wildtype N-Ras.

Source

This cell line was established in 1975 from a metastatic site (lung) in a 43-year-old Caucasian male with metastatic melanoma.

Inventors

- Jorgen Fogh, PhD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center
- Germaine Trempe, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Fogh J et al. (1977) One hundred and twenty-seven cultured human tumor cell lines producing tumors in nude mice. *Journal of the National Cancer Institute* 59: 221-226 (PubMed ID: 327080)
- Xing F et al. (2012) Concurrent loss of the PTEN and RB1 tumor suppressors attenuates RAF dependence in melanomas harboring (V600E)BRAF. *Oncogene* 31: 446-457 (PubMed ID: 21725359)
- Ma J et al. (2013) HER2 as a Promising Target for Cytotoxicity T Cells in Human Melanoma Therapy. *PLoS One* 8: e73261 (PubMed ID: 24015299)

Comments

Malme-3, a normal skin fibroblast cell line, isolated from the same patient as the Malme-3M melanoma cell line, is also available for licensing.

MSK Tracking Code: SK2009-092

SK-MEL-1: Human Melanoma Cell Line

Description

SK-MEL-1 is the first of a series of melanoma cell lines established from patient-derived tumor samples. This cell line is grown in suspension culture and expresses mutant B-Raf (V600E) and wildtype N-Ras. This cell line is known to form tumors in immunocompromised mice.

Source

This cell line was established in 1966 from a metastatic site (thoracic lymph duct) in a 29-year-old Caucasian male with malignant melanoma.

Lead Inventor

Herbert F. Oettgen, MD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Oettgen HF et al. (1968) Suspension culture of a pigment-producing cell line derived from a human malignant melanoma. *Journal of the National Cancer Institute* 41: 827-843 (PubMed ID: 4879578)
- Fogh J et al. (1977) One hundred and twenty-seven cultured human tumor cell lines producing tumors in nude mice. *Journal of the National Cancer Institute* 59: 221-226 (PubMed ID: 327080)
- Xing F et al. (2012) Concurrent loss of the PTEN and RB1 tumor suppressors attenuates RAF dependence in melanomas harboring (V600E) BRAF. *Oncogene* 31(4):446-457 (PubMed ID: 21725359)

MSK Tracking Code: SK2004-052

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SK-MEL-2: Human Melanoma Cell Line

Description

SK-MEL-2 is one of a series of melanoma cell lines established from patient-derived tumor samples. This cell line expresses wildtype B-Raf and mutant N-Ras (Q61R). This cell line is known to form tumors in immunocompromised mice.

Source

This cell line was established in 1972 from a metastatic site on the thigh of a 60-year-old Caucasian male with malignant melanoma.

Inventors

- Lloyd J. Old, MD, former William E. Snee Chair in Cancer Immunology, Memorial Sloan Kettering Cancer Center; former Director, New York Branch, Ludwig Institute for Cancer Research
- Germain Trempe, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Fogh J et al. (1977) One hundred and twenty-seven cultured human tumor cell lines producing tumors in nude mice. *Journal of the National Cancer Institute* 59: 221-226 (PubMed ID: 327080)
- Xing F et al. (2012) Concurrent loss of the PTEN and RB1 tumor suppressors attenuates RAF dependence in melanomas harboring (V600E) BRAF. *Oncogene* 31(4):446-457 (PubMed ID: 21725359)

MSK Tracking Code: SK 779

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SK-MEL-3: Human Melanoma Cell Line

Description

SK-MEL-3 is one of a series of melanoma cell lines established from patient-derived tumor samples. This cell line is known to form tumors in immunocompromised mice.

Source

This cell line was established in 1972 from a metastatic site (lymph node) in a 42-year-old Caucasian female with malignant melanoma.

Inventors

- Lloyd J. Old, MD, former William E. Snee Chair in Cancer Immunology, Memorial Sloan Kettering Cancer Center; former Director, New York Branch, Ludwig Institute for Cancer Research
- Germain Trempe, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

Fogh J et al. (1977) One hundred and twenty-seven cultured human tumor cell lines producing tumors in nude mice. *Journal of the National Cancer Institute* 59: 221-226 (PubMed ID: 327080)

MSK Tracking Code: SK1980-523

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SK-MEL-5: Human Melanoma Cell Line

Description

SK-MEL-5 is one of a series of melanoma cell lines established from patient-derived tumor samples. This cell line expresses mutant B-Raf (V600E) and wildtype N-Ras. This cell line is known to form tumors in immunocompromised mice.

Source

This cell line was established in 1974 from a metastatic site (axillary lymph node) in a 24-year-old Caucasian female with malignant melanoma.

Inventors

- Lloyd J. Old, MD, former William E. Snee Chair in Cancer Immunology,Memorial Sloan Kettering Cancer Center; former Director, New York Branch, Ludwig Institute for Cancer Research
- Toshitada Takahashi, MD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Carey TE et al. (1976) Cell surface antigens of human malignant melanoma: mixed hemadsorption assays for humoral immunity to cultured autologous melanoma cells. *Proceedings of the National Academy of Sciences* 73: 3278-3282 (PubMed ID: 1067619)
- Xing F et al. (2012) Concurrent loss of the PTEN and RB1 tumor suppressors attenuates RAF dependence in melanomas harboring (V600E) BRAF. *Oncogene* 31(4):446-457 (PubMed ID: 21725359)

MSK Tracking Code: SK1980-522

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SK-MEL-24: Human Melanoma Cell Line

Description

SK-MEL-24 is one of a series of melanoma cell lines established from patient-derived tumor samples. This cell line expresses wildtype B-Raf and wildtype N-Ras. This cell line is known to form tumors in immunocompromised mice.

Source

This cell line was established from a metastatic site (lymph node) in a 67-year-old Caucasian male with malignant melanoma.

Inventors

- Lloyd J. Old, MD, former William E. Snee Chair in Cancer Immunology, Memorial Sloan Kettering Cancer Center; former Director, New York Branch, Ludwig Institute for Cancer Research
- Toshitada Takahashi, MD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Carey TE et al. (1976) Cell surface antigens of human malignant melanoma: mixed hemadsorption assays for humoral immunity to cultured autologous melanoma cells. *Proceedings of the National Academy of Sciences* 73: 3278-3282 (PubMed ID: 1067619)
- Xing F et al. (2012) Concurrent loss of the PTEN and RB1 tumor suppressors attenuates RAF dependence in melanomas harboring (V600E) BRAF. *Oncogene* 31(4):446-457 (PubMed ID: 21725359)

MSK Tracking Code: SK2003-078

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SK-MEL-26 Human Melanoma Cell Line

Description

SK-MEL-26 is one of a series of melanoma cell lines established from patient-derived tumor samples. This cell line expresses mutant B-Raf (V600E) and wildtype N-Ras. This cell line is known to form tumors in immunocompromised mice.

Source

This cell line was established in 1975 from a subcutaneous malignant melanoma on the right leg of a 54-year-old Caucasian female.

Inventors

- Lloyd J. Old, MD, former William E. Snee Chair in Cancer Immunology,Memorial Sloan Kettering Cancer Center; former Director, New York Branch, Ludwig Institute for Cancer Research
- Toshitada Takahashi, MD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Fogh J et al. (1977) One hundred and twenty-seven cultured human tumor cell lines producing tumors in nude mice. *Journal of the National Cancer Institute* 59: 221-226 (PubMed ID: 327080)
- Gomi K et al. (1984) Antitumor effect of human recombinant interferon-beta against human melanomas transplanted into nude mice. *Journal of Pharmacobiodynamics* 7: 951-961 (PubMed ID: 6533284)
- Fujino M et al. (1999) Effects of protein kinase inhibitors on radiation-induced WAF1 accumulation in human cultured melanoma cells. *British Journal of Dermatology* 141: 652-657 (PubMed ID: 10583112)
- Xing F et al. (2012) Concurrent loss of the PTEN and RB1 tumor suppressors attenuates RAF dependence in melanomas harboring (V600E) BRAF. *Oncogene* 31(4): 446-457 (PubMed ID: 21725359)

MSK Tracking Code: SK1980-546

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SK-MEL-28: Human Melanoma Cell Line

Description

SK-MEL-28 is one of a series of melanoma cell lines established from patient-derived tumor samples. This cell line expresses mutant B-Raf (V600E) and wildtype N-Ras. This cell line is known to form tumors in immunocompromised mice.

Source

This cell line was established from the primary tumor on the skin of a 51-year-old male of unknown ethnicity.

Inventors

- Lloyd J. Old, MD, former William E. Snee Chair in Cancer Immunology, Memorial Sloan Kettering Cancer Center; former Director, New York Branch, Ludwig Institute for Cancer Research
- Toshitada Takahashi, MD, formerly at Sloan Kettering Institute,Memorial Sloan Kettering Cancer Center

Key References

- Carey TE et al. (1976) Cell surface antigens of human malignant melanoma: mixed hemadsorption assays for humoral immunity to cultured autologous melanoma cells. *Proceedings of the National Academy of Sciences* 73: 3278-3282 (PubMed ID: 1067619)
- Xing F et al. (2012) Concurrent loss of the PTEN and RB1 tumor suppressors attenuates RAF dependence in melanomas harboring (V600E) BRAF. *Oncogene* 31(4):446-457 (PubMed ID: 21725359)

MSK Tracking Code: SK1980-524

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SK-MEL-29: Human Melanoma Cell Line

Description

SK-MEL-29 is one of a series of melanoma cell lines established from patient-derived tumor samples. This cell line expresses mutant B-Raf (V600E) and wildtype N-Ras. This cell line is known to form tumors in immunocompromised mice.

Source

This cell line was established in 1975 from a recurrent melanoma at the apex of the left axilla of a 19-year-old Caucasian male.

Inventors

- Lloyd J. Old, MD, former William E. Snee Chair in Cancer Immunology, Memorial Sloan Kettering Cancer Center; former Director, New York Branch, Ludwig Institute for Cancer Research
- Toshitada Takahashi, MD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Carey TE et al. (1976) Cell surface antigens of human malignant melanoma: mixed hemadsorption assays for humoral immunity to cultured autologous melanoma cells. *Proceedings of the National Academy of Sciences* 73: 3278-3282 (PubMed ID: 1067619)
- Lau YS et al. (2006) Malignant melanoma and bone resorption. *British Journal of Cancer* 94(10): 1496-1503 (PubMed ID: 16641914)
- Xing F et al. (2012) Concurrent loss of the PTEN and RB1 tumor suppressors attenuates RAF dependence in melanomas harboring (V600E) BRAF. *Oncogene* 31(4): 446-457 (PubMed ID: 21725359)

MSK Tracking Code: SK1980-525

SK-MEL-30: Human Melanoma Cell Line

Description:

SK-MEL-30 is one of a series of melanoma cell lines established from patient-derived tumor samples. This cell line expresses wildtype B-Raf and mutant N-Ras (Q61K). This cell line is known to form tumors in immunocompromised mice.

Source

This cell line was established in 1975 from a soft-tissue metastatic site (dermis) in a 66-year-old Caucasian male.

Inventors

- Lloyd J. Old, MD, former William E. Snee Chair in Cancer Immunology, Memorial Sloan Kettering Cancer Center; former Director, New York Branch, Ludwig Institute for Cancer Research
- Toshitada Takahashi, MD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Carey TE et al. (1976) Cell surface antigens of human malignant melanoma: mixed hemadsorption assays for humoral immunity to cultured autologous melanoma cells. *Proceedings of the National Academy of Sciences* 73: 3278-3282 (PubMed ID: 1067619)
- Xing F et al. (2012) Concurrent loss of the PTEN and RB1 tumor suppressors attenuates RAF dependence in melanomas harboring (V600E) BRAF. *Oncogene* 31(4): 446-457 (PubMed ID: 21725359)

MSK Tracking Code: SK1980-526

SK-MEL-31: Human Melanoma Cell Line

Description

SK-MEL-31 is one of a series of melanoma cell lines established from patient-derived tumor samples. This cell line expresses wildtype B-Raf and wildtype N-Ras. This cell line is known to form tumors in immunocompromised mice.

Source

This cell line was established from the tumor cells of a female, of unknown age and ethnicity, with malignant melanoma.

Inventors

- Lloyd J. Old, MD, former William E. Snee Chair in Cancer Immunology, Memorial Sloan Kettering Cancer Center; former Director, New York Branch, Ludwig Institute for Cancer Research
- Toshitada Takahashi, MD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Carey TE et al. (1976) Cell surface antigens of human malignant melanoma: mixed hemadsorption assays for humoral immunity to cultured autologous melanoma cells. *Proceedings of the National Academy of Sciences* 73: 3278-3282 (PubMed ID: 1067619)
- Xing F et al. (2012) Concurrent loss of the PTEN and RB1 tumor suppressors attenuates RAF dependence in melanomas harboring (V600E) BRAF. *Oncogene* 31(4):446-457 (PubMed ID: 21725359)

MSK Tracking Code: SK1980-527

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SH-SY5Y: Human Neuroblastoma Cell Line

Description

SH-SY5Y is a twice-subcloned cell line derived from the SK-N-SH neuroblastoma cell line. It serves as a model for neurodegenerative disorders since the cells can be converted to various types of functional neurons by the addition of specific compounds. In addition, the SH-SY5Y cell line has been used widely in experimental neurological studies, including analysis of neuronal differentiation, metabolism, and function related to neurodegenerative processes, neurotoxicity, and neuroprotection.

Source

This cell line was derived from the SH-SY subclone of the parental SK-N-SH human neuroblastoma cell line. The parental SK-N-SH cell line was established in 1970 from metastatic cells found in the bone marrow aspirate of a four-year-old female of unknown ethnicity.

Inventors

- June L. Biedler, PhD, former Chairman, Cell Biology and Genetics Program, Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center
- Barbara A. Spengler, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

Ross RA et al. (1983) Coordinate morphological and biochemical interconversion of human neuroblastoma cells. *Journal of the National Cancer Institute* 71: 741-748 (PubMed ID: 6137586)

MSK Tracking Code: SK 810

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SK-N-BE(2): Human Neuroblastoma Cell Line

Description

SK-N-BE(2) is a neuroblastoma cell line that displays *MYCN* amplification. These cells have moderate dopamine-β-hydroxylase activity and low-choline acetyltransferase activity. The SK-N-BE(2) cells are known to form tumors in immunocompromised mice.

Source

This cell line was established in 1972 from a metastatic site (bone marrow) in a two-year-old Caucasian male with malignant neuroblastoma.

Inventors

- June L. Biedler, PhD, former Chairman, Cell Biology and Genetics Program, Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center
- Barbara A. Spengler, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Biedler JL et al. (1976) A novel chromosome abnormality in human neuroblastoma and antifolate-resistant Chinese hamster cell lines in culture. *Journal of the National Cancer Institute 57*: 683-695 (PubMed ID: 62055)
- Biedler JL et al. (1978) Multiple neurotransmitter synthesis by human neuroblastoma cell lines and clones. *Cancer Research* 38: 3751-3757 (PubMed ID: 29704)
- Veas-Perez De Tudela M et al. (2010) Human neuroblastoma cells with *MYCN* amplification are selectively resistant to oxidative stress by transcriptionally up-regulating glutamate cysteine ligase. *Journal of Neurochemistry* 113: 819-825 (PubMed ID: 20180881)

MSK Tracking Code: SK1980-530

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SK-N-BE(2)-C: Human Neuroblastoma Cell Line

Description

SK-N-BE(2)-C is a clonal subline of the SK-N-BE(2) neuroblastoma cell line. Like the parental cell line, these cells display *MYCN* amplification. Treatment with trans-retinoic acid differentiates these cells into a distinct neuronal phenotype. These cells display high levels of tyrosine hydroxylase activity and dopamine-β-hydroxylase activity.

Source

This cell line is a subclone of the SK-N-BE(2) neuroblastoma cell line. The parental cell line was established in 1972 from a metastatic site (bone marrow) in a two-year-old Caucasian male with malignant neuroblastoma.

Inventors

- June L. Biedler, PhD, former Chairman, Cell Biology and Genetics Program, Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center
- Barbara A. Spengler, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Ciccarone V et al. (1989) Phenotypic diversification in human neuroblastoma cells: expression of distinct neural crest lineages. *Cancer Research* 49: 219-225 (PubMed ID: 2535691)
- Qiao J et al. (2012) PI3K/AKT and ERK regulate retinoic acid-induced neuroblastoma cellular differentiation. *Biochemical and Biophysical Research Communications* 424: 421-426 (PubMed ID: 22766505)

MSK Tracking Code: SK1980-532

SK-N-BE(2)-M17: Human Neuroblastoma Cell Line

Description

SK-N-BE(2)-M17 is a twice-sub-cloned cell line derived from the SK-N-BE(2) neuroblastoma cell line. Like the parental cell line, these cells display *MYCN* amplification. Treatment with trans-retinoic acid differentiates these cells into a distinct neuronal phenotype. These cells display high levels of tyrosine hydroxylase activity and moderate dopamine-β-hydroxylase activity.

Source

This cell line is a subclone of the SK-N-BE(2) neuroblastoma cell line. The parental cell line was established in 1972 from a metastatic site (bone marrow) in a two-year-old Caucasian male with malignant neuroblastoma.

Inventors

- June L. Biedler, PhD, former Chairman, Cell Biology and Genetics Program, Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center
- Barbara A. Spengler, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Ciccarone V et al. (1989) Phenotypic diversification in human neuroblastoma cells: expression of distinct neural crest lineages. *Cancer Research* 49: 219-225 (PubMed ID: 2535691)
- Andres D et al. (2013) Morphological and functional differentiation in BE(2)-M17 human neuroblastoma cells by treatment with trans-retinoic acid. *BMC Neuroscience* 14: 49 (PubMed ID: 23597229)

MSK Tracking Code: SK1980-531

SK-N-MC: Human Neuroblastoma Cell Line

Description

SK-N-MC was originally described as a neuroblastoma cell line. It is now widely regarded as having originated from an Askin's tumor (Ewing family of tumors). These cells harbor the oncogenic *EWS-FLI1* chromosomal rearrangement. They were initially found to contain double-minute chromosomes, which were lost upon prolonged *in vitro* culture. The SK-N-MC cells have little or no dopamine-β-hydroxylase activity but show elevated choline acetyltransferase activity compared to other neuroblastoma cell lines such as the SK-N-SH and SH-SY5Y. These cells are known to form tumors in immunocompromised mice.

Source

This cell line was established in 1971 from a metastatic site (supra-orbital region) in a 14-year-old Caucasian female with an Askin's tumor.

Inventors

- June L. Biedler, PhD, former Chairman, Cell Biology and Genetics Program, Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center
- Lawrence Helson, MD, formerly at Memorial Sloan Kettering Cancer Center
- Barbara A. Spengler, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Biedler JL et al. (1973) Morphology and growth, tumorigenicity, and cytogenetics of human neuroblastoma cells in continuous culture. *Cancer Research* 33: 2643-2652 (PubMed ID: 4748425)
- Helson L et al. (1975) Human neuroblastoma in nude mice. *Cancer Research* 35: 2594-2599 (PubMed ID: 167965)
- Biedler JL et al. (1978) Multiple neurotransmitter synthesis by human neuroblastoma cell lines and clones. *Cancer Research* 38: 3751-3757 (PubMed ID: 29704)

MSK Tracking Code: SK 776

SK-N-MC-IXC: Human Neuroblastoma Cell Line

Description

SK-N-MC-IXC is a twice-sub-cloned cell line derived from the SK-N-MC cell line. These cells share many characteristics with the parental cell line. Unlike the SK-N-MC cell line, however, these cells contain a variable number of double-minute chromosomes. At the time of establishment, double-minute chromosomes were present in almost 90 percent of the cells examined and varied in number and size. Like the parental cell line, the SK-N-MC-IXC cells have little or no dopamine-β-hydroxylase activity. They do, however, have significantly increased choline acetyltransferase activity compared to the parental SK-N-MC cell line.

Source

This cell line was derived from the SK-N-MC-IX subclone of the parental SK-N-MC cell line. The parental cell line was established in 1971 from a metastatic site (supra-orbital region) in a 14-year-old Caucasian female with an Askin's tumor.

Inventors

- June L. Biedler, PhD, former Chairman, Cell Biology and Genetics Program, Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center
- Barbara A. Spengler, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Biedler JL et al. (1973) Morphology and growth, tumorigenicity, and cytogenetics of human neuroblastoma cells in continuous culture. *Cancer Research* 33: 2643-2652 (PubMed ID: 4748425)
- Biedler JL et al. (1978) Multiple neurotransmitter synthesis by human neuroblastoma cell lines and clones. *Cancer Research* 38: 3751-3757 (PubMed ID: 29704)

MSK Tracking Code: SK2010-067

SK-N-SH: Human Neuroblastoma Cell Line

Description

SK-N-SH is a neuroblastoma cell line that displays epithelial morphology and grows in adherent culture. Treatment with all-trans-retinoic acid causes these cells to differentiate and adopt a neuronal phenotype, characterized by extensive neurite outgrowth. This makes them particularly useful for delineating signaling pathways involved in neuronal differentiation. In addition, the SK-N-SH cells are known to form tumors in immunocompromised mice.

Source

This cell line was established in 1970 from metastatic cells found in the bone marrow aspirate of a four-year-old female of unknown ethnicity.

Inventors

- June L. Biedler, PhD, former Chairman, Cell Biology and Genetics Program, Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center
- Barbara A. Spengler, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Biedler JL et al. (1973) Morphology and growth, tumorigenicity, and cytogenetics of human neuroblastoma cells in continuous culture. *Cancer Research* 33: 2643-2652 (PubMed ID: 4748425)
- Helson L et al. (1975) Human neuroblastoma in nude mice. *Cancer Research* 35: 2594-2599 (PubMed ID: 167965)

MSK Tracking Code: SK1980-529

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Caov-3: Human Ovarian Cancer Cell Line

Description

The Caov-3 cell line is a primary ovarian cancer cell line with epithelial morphology. These cells form tightly packed colonies in adherent culture. All-trans retinoic acid has been shown to suppress the growth of Caov-3 ovarian carcinoma cells *in vitro*. These cells express the NB/70K, CA-125, Ba-2, and Ca-1 tumor-associated antigens. The Caov-3 cells harbor a nonsense mutation in the *p53* gene, and have multiple copies of the ovarian cancer oncogene *PIK3CA*. They are sensitive to vinblastine, cisplatin, and adriamycin. These cells fail to grow in soft agar but are tumorigenic when injected into immunocompromised mice.

Source

This cell line was established from the primary tumor of a 54-year-old Caucasian female with adenocarcinoma of the ovary.

Lead Inventor

Jorgen Fogh, PhD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Buick RN et al. (1985) Comparative properties of five human ovarian adenocarcinoma cell lines. *Cancer Research* 45: 3668-3676 (PubMed ID: 4016745)
- Yaginuma Y et al. (1992) Abnormal structure and expression of the p53 gene in human ovarian carcinoma cell lines. *Cancer Research* 52: 4196-4199 (PubMed ID: 1638534)
- Shayesteh L et al. (1999) PIK3CA is implicated as an oncogene in ovarian cancer. *Nature Genetics* 21: 99-102 (PubMed ID: 9916799)

MSK Tracking Code: SK2010-069

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Caov-4: Human Ovarian Cancer Cell Line

Description

The Caov-4 cell line is an ovarian cancer cell line with epithelial morphology that grows in adherent culture. These cells harbor a loss-of-function mutation in the *p53* gene and are sensitive to cisplatin.

Source

This cell line was established from a metastatic site (fallopian tube) in a 45-year-old Caucasian female with adenocarcinoma of the ovary.

Lead Inventor

Jorgen Fogh, PhD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

Yaginuma Y et al. (1992) Abnormal structure and expression of the p53 gene in human ovarian carcinoma cell lines. *Cancer Research* 52: 4196-4199 (PubMed ID: 1638534)

MSK Tracking Code: SK2010-070

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SK-OV-3: Human Ovarian Cancer Cell Line

Description

SK-OV-3 is a human ovarian cancer cell line with epithelial-like morphology. These cells are resistant to tumor necrosis factor and to other cytotoxic drugs such as diphtheria toxin, cisplatin, and adriamycin. The SK-OV-3 cell line forms colonies in soft agar, which serves as a surrogate assay for tumorigenicity. Intra-peritoneal injection of these cells into immunocompromised mice results in the growth of tumors resembling clear cell adenocarcinoma, within two to three months.

Source

This cell line was established in 1973 from the ascites of a 64-year-old Caucasian female with adenocarcinoma of the ovary.

Inventors

- Lloyd J. Old, MD, former William E. Snee Chair in Cancer Immunology, Memorial Sloan Kettering Cancer Center; former Director, New York Branch, Ludwig Institute for Cancer Research
- Germain Trempe, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Fogh J et al. (1977) One hundred and twenty-seven cultured human tumor cell lines producing tumors in nude mice. *Journal of the National Cancer Institute* 59: 221-226 (PubMed ID: 327080)
- Shaw TJ et al. (2004) Characterization of intraperitoneal, orthotopic, and metastatic xenograft models of human ovarian cancer. *Molecular Therapy* 10: 1032–1042 (PubMed ID: 15564135)

MSK Tracking Code: SK1980-528

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Capan-1: Human Pancreatic Adenocarcinoma Cell Line

Description

Capan-1 is a human pancreatic ductal adenocarcinoma cell line. These cells grow in adherent tissue culture and display epithelial morphology. In culture, these cells are capable of invading through an extracellular matrix, such as Matrigel. The Capan-1 cells are resistant to 5-fluorouracil, reminiscent of the original tumor from which they were derived. They form poorly-differentiated tumors when injected into immunocompromised mice. These cells harbor a single base-pair deletion in the *BRCA2* allele, which results in the expression of a truncated and dysfunctional protein. In addition, they have an oncogenic mutation in K-Ras (G12V) and an inactivating mutation in p53. These cells express elevated levels of the Epidermal Growth Factor Receptor (EGFR) and do not express SMAD4 protein (i.e., SMAD4-null). The Capan-1 cells are useful both as a xenograft model for pancreatic cancer and as a cell system to study the effects of BRCA2-deficiency.

Source

This cell line was established in 1974 from a metastatic site (liver) in a 40-year-old Caucasian male with pancreatic ductal adenocarcinoma.

Lead Inventor

Jorgen Fogh, PhD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Fogh J et al. (1977) One hundred and twenty-seven cultured human tumor cell lines producing tumors in nude mice. *Journal of the National Cancer Institute* 59: 221-226 (PubMed ID: 327080)
- Kyriazis AP et al. (1982) Human pancreatic adenocarcinoma line Capan-1 in tissue culture and the nude mouse: morphologic, biologic, and biochemical characteristics. *American Journal of Pathology* 106: 250-260 (PubMed ID: 6278935)
- Deer EL et al. (2010) Phenotype and genotype of pancreatic cancer cell lines. *Pancreas* 39: 425-435 (PubMed ID: 20418756)

MSK Tracking Code: SK 923

Capan-2: Human Pancreatic Adenocarcinoma Cell Line

Description

Capan-2 is a human pancreatic ductal adenocarcinoma cell line. These cells grow in adherent tissue culture and display epithelial morphology. They form well-differentiated tumors when injected into immunocompromised mice and are used as a xenograft model for pancreatic cancer. The Capan-2 cells express mutant K-Ras (G12V) and elevated levels of the Epidermal Growth Factor Receptor (EGFR). In addition, they express wildtype p53 and normal levels of SMAD4 protein.

Source

This cell line was established in 1975 from the primary tumor of a 56-year-old Caucasian male with pancreatic ductal adenocarcinoma.

Inventors

- Jorgen Fogh, PhD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center
- James D. Loveless, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Kyriazis AA et al. (1986) Morphological, biological, biochemical, and karyotypic characteristics of human pancreatic ductal adenocarcinoma Capan-2 in tissue culture and the nude mouse. *Cancer Research* 46: 5810-5815 (PubMed ID: 3019537)
- Deer EL et al. (2010) Phenotype and genotype of pancreatic cancer cell lines. *Pancreas* 39: 425-435 (PubMed ID: 20418756)

MSK Tracking Code: SK2000-049

Caki-1: Human Renal Cancer Cell Line

Description

Caki-1 is a human clear cell renal cell carcinoma (ccRCC) line that displays epithelial morphology and grows in adherent culture. When grown on transwell filters, these cells form a polarized monolayer with microvilli on the apical surface and display characteristic features of the proximal tubule epithelium. In addition, the Caki-1 cells are also a useful model to study renal cancer. They are more sensitive to 5-fluorouracil and sorafenib (multi-kinase inhibitor of VEGFRs 1-3, PDGFR-β and Raf-1) than the Caki-2 cells. The Caki-1 cells express wildtype von Hippel-Lindau (VHL) tumor-suppressor protein and are known to form tumors in immunocompromised mice.

Source

This cell line was established in 1971 from a metastatic site (skin) in a 49-year-old Caucasian male with clear cell carcinoma of the kidney.

Inventors

- Jorgen Fogh, PhD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center
- Germain Trempe, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Fogh J et al. (1977) One hundred and twenty-seven cultured human tumor cell lines producing tumors in nude mice. *Journal of the National Cancer Institute* 59: 221-226 (PubMed ID: 327080)
- Glube N et al. (2007) Caki-1 cells represent an in vitro model system for studying the human proximal tubule epithelium. *Experimental Nephrology* 107: e47–e56 (PubMed ID: 17804913)
- Miyake M et al. (2012) 5-fluorouracil enhances the antitumor effect of sorafenib and sunitinib in a xenograft model of human renal cell carcinoma. *Oncology Letters* 3: 1195–1202 (PubMed ID: 22783417)

MSK Tracking Code: SK1980-534

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Caki-2: Human Renal Cancer Cell Line

Description

Caki-2 is a human clear cell renal cell carcinoma (ccRCC) line that displays epithelial morphology and grows in adherent culture. These cells are a useful preclinical model to study renal cancer. They are relatively less sensitive to 5-fluorouracil and sorafenib (multi-kinase inhibitor of VEGFRs 1-3, PDGFR-β, and Raf-1) compared to Caki-1 cells. The Caki-2 cells have a loss-of-function mutation in the von Hippel-Lindau (VHL) tumor-suppressor protein and are known to form tumors i n immunocompromised mice.

Source

This cell line was established in 1971 from the primary tumor of a 69-year-old Caucasian male with clear cell carcinoma of the kidney.

Inventors

- Jorgen Fogh, PhD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center
- Germain Trempe, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Fogh J et al. (1977) One hundred and twenty-seven cultured human tumor cell lines producing tumors in nude mice. *Journal of the National Cancer Institute* 59: 221-226 (PubMed ID: 327080)
- Miyake M et al. (2012) 5-fluorouracil enhances the antitumor effect of sorafenib and sunitinib in a xenograft model of human renal cell carcinoma. *Oncology Letters* 3: 1195–1202 (PubMed ID: 22783417)

MSK Tracking Code: SK2010-068

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SK-JHRCC-3: Human Renal Cancer Cell Line

Description

SK-JHRCC-3 is a human clear cell renal cell carcinoma (ccRCC) cell line that displays epithelial morphology and grows in adherent culture. When grown in Matrigel 3D culture, this cell line forms round colonies. The SK-JHRCC-3 cells are a useful preclinical model to study renal cancer. They form tumors when inoculated subcutaneously in immunocompromised NSG mice. These xenograft tumors are sensitive to Sunitinib (oral, small molecule, multi-targeted receptor tyrosine kinase inhibitor, approved for the treatment of renal cell carcinoma) *in vivo*. Histological analysis shows that SK-JHRCC-3 tumor xenografts exhibit the same clear cell feature as the primary tumor from which they were derived. Immunohistochemistry analysis shows that SK-JHRCC-3 xenograft tumors are positive for PAX8 and CAIX expression. Exome sequencing revealed that these cells have mutations in *PBRM1* and *SETD2* and express wildtype *BAP1*, *KDM5C*, *PTEN*, *mTOR,* and *PIK3CA*.

Source

This cell line was established in 2011 from the primary tumor of a 71-year-old Caucasian male with clear cell carcinoma of the kidney and was responsive to treatment with Sunitinib.

Inventors

- James J. Hsieh, MD, PhD, Laboratory Head, Memorial Hospital Research Laboratories, Human Oncology & Pathogenesis Program, Memorial Sloan Kettering Cancer Center
- Emily H. Cheng, MD, PhD, Laboratory Head, Memorial Hospital Research Laboratories, Human Oncology & Pathogenesis Program,Memorial Sloan Kettering Cancer Center
- Yiyu Dong, PhD, Research Associate, Hsieh Laboratory, Memorial Hospital Research Laboratories, Human Oncology & Pathogenesis Program, Memorial Sloan Kettering Cancer Center

SK-JHRCC-12: Human Renal Cancer Cell Line

Description

SK-JHRCC-12 is a human clear cell renal cell carcinoma (ccRCC) cell line that displays epithelial morphology and grows in adherent culture. This cell line forms big colonies when plated in soft agar and forms colonies with extensive branching when grown in Matrigel 3D culture. The SK-JHRCC-12 cells are a useful preclinical model to study renal cancer. They form tumors when inoculated subcutaneously in immunocompromised NSG mice. SK-JHRCC-12 cells are insensitive to Sunitinib (oral, small molecule, multi-targeted receptor tyrosine kinase inhibitor, approved for the treatment of renal cell carcinoma) both *in vitro* (cell culture) and *in vivo* (xenograft mouse models). Histological analysis shows that SK-JHRCC-12 xenografts exhibit the same sarcomatoid feature as the primary tumor they were derived from. Immunohistochemistry analysis shows that JHRCC12 xenograft tumors are positive for PAX8 and CAIX expression. The SK-JHRCC-12 cells have mutations in von Hippel-Lindau (*VHL*), *PBRM1,* and *SETD2* and express wildtype *KDM5C*, *PTEN*, *mTOR,* and *PIK3CA*.

Source

This cell line was established in 2011 from a metastatic site (bone) in a 47-year-old Caucasian male with clear cell carcinoma of the kidney and was refractory to treatment with Sunitinib.

Inventors

- James J. Hsieh, MD, PhD, Laboratory Head, Memorial Hospital Research Laboratories, Human Oncology & Pathogenesis Program, Memorial Sloan Kettering Cancer Center
- Emily H. Cheng, MD, PhD, Laboratory Head, Memorial Hospital Research Laboratories, Human Oncology & Pathogenesis Program, Memorial Sloan Kettering Cancer Center
- Yiyu Dong, PhD, Research Associate, Hsieh Laboratory, Memorial Hospital Research Laboratories, Human Oncology & Pathogenesis Program, Memorial Sloan Kettering Cancer Center

SK-JHRCC-92: Human Renal Cancer Cell Line

Description

SK-JHRCC-92 is a human clear cell renal cell carcinoma (ccRCC) cell line that displays epithelial morphology and grows in adherent culture. This cell line forms big colonies when plated in soft agar. The SK-JHRCC-92 cells are a useful preclinical model to study renal cancer. They form tumors when inoculated subcutaneously in immunocompromised NSG mice. These xenografts are insensitive to Sunitinib (oral, small molecule, multi-targeted receptor tyrosine kinase inhibitor, approved for the treatment of renal cell carcinoma) *in vivo*.

Source

This cell line was established in 2012 from a metastatic site (bone) in a 45-year-old Caucasian male with clear cell carcinoma of the kidney.

Inventors

- James J. Hsieh, MD, PhD, Laboratory Head, Memorial Hospital Research Laboratories, Human Oncology & Pathogenesis Program, Memorial Sloan Kettering Cancer Center
- Emily H. Cheng, MD, PhD, Laboratory Head, Memorial Hospital Research Laboratories, Human Oncology & Pathogenesis Program, Memorial Sloan Kettering Cancer Center
- Yiyu Dong, PhD, Research Associate, Hsieh Laboratory, Memorial Hospital Research Laboratories, Human Oncology & Pathogenesis Program, Memorial Sloan Kettering Cancer Center

SK-JHRCC-120: Human Renal Cancer Cell Line

Description

SK-JHRCC-120 is a human clear cell renal cell carcinoma (ccRCC) cell line that displays epithelial morphology and grows in adherent culture. This cell line forms big colonies when plated in soft agar. The SK-JHRCC-120 cells are a useful preclinical model to study renal cancer. They form tumors when inoculated subcutaneously in immunocompromised NSG mice. These xenografts are insensitive to Sunitinib (oral, small molecule, multi-targeted receptor tyrosine kinase inhibitor, approved for the treatment of renal cell carcinoma) *in vivo*.

Source

This cell line was established in 2013 from the primary tumor of a 49-year-old Caucasian male with clear cell carcinoma of the kidney and was refractory to treatment with Sunitinib.

Inventors

- James J. Hsieh, MD, PhD, Laboratory Head, Memorial Hospital Research Laboratories, Human Oncology & Pathogenesis Program, Memorial Sloan Kettering Cancer Center
- Emily H. Cheng, MD, PhD, Laboratory Head, Memorial Hospital Research Laboratories, Human Oncology & Pathogenesis Program, Memorial Sloan Kettering Cancer Center
- Yiyu Dong, PhD, Research Associate, Hsieh Laboratory, Memorial Hospital Research Laboratories, Human Oncology & Pathogenesis Program, Memorial Sloan Kettering Cancer Center

SK-RC-(M): Metastatic Human Renal Cell Carcinoma (RCC) Cell Line Collection

Description

This is a collection of renal cell carcinoma (RCC) cell lines derived from metastatic sites of patients who underwent surgery.
See chart for additional information.

Source

All cell lines were established in Dr. Lloyd Old's laboratory, from patients undergoing nephrectomy at Memorial Hospital, Memorial Sloan Kettering Cancer Center, between the years 1972 and 1987. Fresh surgical specimens were obtained from Tumor Procurement Services, Department of Surgical Pathology, Memorial Hospital, Memorial Sloan Kettering Cancer Center.

Lead Inventor

Lloyd J. Old, MD, former William E. Snee Chair in Cancer Immunology, Memorial Sloan Kettering Cancer Center; former Director, New York Branch, Ludwig Institute for Cancer Research, and members of the Old Laboratory

Key References

- Ueda R et al. (1979) Cell surface antigens of human renal cancer defined by autologous typing. *Journal of Experimental Medicine* 150: 564-579 (PubMed ID: 479762)
- Ebert T et al. (1990) Establishment and characterization of human renal cancer and normal kidney cell lines. *Cancer Research* 50: 5531-5536 (PubMed ID: 2386958)
- Sjölund J et al. (2008) Suppression of renal cell carcinoma growth by inhibition of Notch signaling in vitro and in vivo. *Journal of Clinical Investigation* 118: 217-228 (PubMed ID: 18079963)

Comments

Cell lines may be licensed individually or in any preferred combination.

Metastatic Human Renal Cell Carcinoma Cell Line Collection

Cell Line	Metastatic Site	Growth in Nude Mice	Growth in Soft Agar
SK-RC-9	Brain	x	√
SK-RC-13*	Brain	√	x
SK-RC-17	Soft Tissue	n.d.	√
SK-RC-18	Lymph Node	√	√
SK-RC-26a*	Lung	n.d.	n.d.
SK-RC-26b*	Lymph Node	n.d.	n.d.
SK-RC-29	Ovary	√	√
SK-RC-31	Lung	√	√
SK-RC-38	Lung	√	√
SK-RC-39	Soft Tissue	√	√
SK-RC-42	Bone	√	√
SK-RC-45*	Adrenal Gland	√	√
SK-RC-46	Bone	n.d.	x
SK-RC-52	Mediastinum	√	√
SK-RC-54	Lung	n.d.	n.d.

*A cell line established from the primary tumor of the same patient is available. Please contact us for more details.

Key: x = No; √ = Yes; n.d. = Not Determined

Adapted from: Ebert T et al. *Cancer Research* 50: 5531-5536 (1990)

SK-RC-(P): Primary Human Renal Cell Carcinoma (RCC) Cell Line Collection

Description

This is a collection of renal cell carcinoma (RCC) cell lines derived from the primary tumors of patients who underwent surgery.

Source

All cell lines were established in Dr. Lloyd Old's laboratory, from patients undergoing nephrectomy at Memorial Hospital, Memorial Sloan Kettering Cancer Center, between the years 1972 and 1987. Fresh surgical specimens were obtained from Tumor Procurement Services, Department of Surgical Pathology, Memorial Hospital, Memorial Sloan Kettering Cancer Center

Lead Inventor

Lloyd J. Old, MD, former William E. Snee Chair in Cancer Immunology, Memorial Sloan Kettering Cancer Center; former Director, New York Branch, Ludwig Institute for Cancer Research, and members of the Old Laboratory

Key References

- Ueda R et al. (1979) Cell surface antigens of human renal cancer defined by autologous typing. *Journal of Experimental Medicine* 150: 564-579 (PubMed ID: 479762)
- Ebert T et al. (1990) Establishment and characterization of human renal cancer and normal kidney cell lines. *Cancer Research* 50: 5531-5536 (PubMed ID: 2386958)
- Sjölund J et al. (2008) Suppression of renal cell carcinoma growth by inhibition of Notch signaling in vitro and in vivo. *Journal of Clinical Investigation* 118: 217-228 (PubMed ID: 18079963)

Comments

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Primary Human Renal Cell Carcinoma (RCC) Cell Line Collection

Cell Line	Growth in Nude Mice	Growth in Soft Agar
SK-RC-1	√	x
SK-RC-2	√	x
SK-RC-4	x	n.d.
SK-RC-6	√	√
SK-RC-7	x	x
SK-RC-10*	n.d.	n.d.
SK-RC-8	n.d.	x
SK-RC-12	n.d.	x
SK-RC-15	√	n.d.
SK-RC-26*	n.d.	n.d.
SK-RC-21	x	√
SK-RC-28	x	√
SK-RC-35	√	x
SK-RC-37	√	n.d.
SK-RC-40	n.d.	n.d.
SK-RC-41	x	√
SK-RC-44*	√	n.d.
SK-RC-47	n.d.	√
SK-RC-48	√	n.d.
SK-RC-49	√	n.d.
SK-RC-51	√	n.d.
SK-RC-53	√	n.d.
SK-RC-55	n.d.	n.d.
SK-RC-56	√	√
SK-RC-57	√	√
SK-RC-58	√	√
SK-RC-59	√•	√
SK-RC-60	√	√
SK-RC-61	√	√
SK-RC-62	x	x

*A cell line established from a metastatic site in the same patient is available. Please contact us for more details.

Key: x = No; √ = Yes; n.d. = Not Determined

Adapted from: Ebert T et al. *Cancer Research* 50: 5531-5536 (1990)

Saos-2: Human Osteosarcoma Cell Line

Description

Saos-2 is a human osteosarcoma cell line, which displays several osteoblastic features. These cells express receptors for 1,25-dihydroxyvitamin D_3 and have high basal alkaline-phosphatase activity. They express the parathyroid hormone (PTH) receptor and produce cyclic AMP in response to treatment with PTH. These cells do not form tumors when injected subcutaneously into immunocompromised mice. When injected into diffusion chambers that are implanted intra-peritoneally into immunocompromised mice, however, Saos-2 cells produce a mineralized matrix, which is a defining characteristic of osteoblastic cells. All of these characteristics make this cell line an attractive source of bone-related molecules for research.

Source

This cell line was established in 1973 from an 11-year-old Caucasian female with osteogenic sarcoma.

Lead Inventor

Jorgen Fogh, PhD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Fogh J et al. (1977) Absence of HeLa cell contamination in 169 cell lines derived from human tumors. *Journal of the National Cancer Institute* 58: 209-214 (PubMed ID: 833871)
- Rodan SB et al. (1987) Characterization of a human osteosarcoma cell line (Saos-2) with osteoblastic properties. *Cancer Research* 47: 4961-4966 (PubMed ID: 3040234)

MSK Tracking Code: SK 771

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SK-ES-1: Human Ewing Sarcoma Cell Line

Description

SK-ES-1 is a human Ewing sarcoma (anaplastic osteosarcoma) cell line that displays epithelial morphology and grows in adherent tissue culture. These cells are a useful preclinical model to study Ewing sarcoma and have been used in the assessment of experimental therapeutic agents. SK-ES-1 cells form xenograft small-cell malignant tumors consistent with Ewing sarcoma when injected into immunocompromised mice. These cells have been reported to express mutant p53 (C176F) protein.

Source

This cell line was established in 1971 from a bone biopsy in an 18-year-old Caucasian male with Ewing's sarcoma.

Lead Inventor

Eda T. Bloom, PhD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Bloom ET (1972) Further definition by cytotoxicity tests of cell surface antigens of human sarcomas in culture. *Cancer Research* 32: 960-967 (PubMed ID: 4502173)
- Komuro H et al. (1993) Mutations of the p53 gene are involved in Ewing's sarcomas but not in neuroblastomas. *Cancer Research* 53: 5284-5288 (PubMed ID: 8221663)
- McCarty G, Awad O, and Loeb DM. (2011) WT1 protein directly regulates expression of vascular endothelial growth factor and is a mediator of tumor response to hypoxia. *Journal of Biological Chemistry* 286: 43634-43643 (PubMed ID: 22030397)
- Sémiond D et al. (2013) Can taxanes provide benefit in patients with CNS tumors and in pediatric patients with tumors? An update on the preclinical development of cabazitaxel. *Cancer Chemotherapy and Pharmacology* 72: 515-528 (PubMed ID: 23820961)

MSK Track Code: SK1980-542

SK-NEP-1: Human Ewing Sarcoma Cell Line

Description

SK-NEP-1 was originally described as an anaplastic Wilms-tumor/renal-cancer cell line. It has, however, been reclassified as a cell line belonging to the Ewing sarcoma family of tumors, since these cells harbor the oncogenic *EWS-FLI1* chromosomal rearrangement. These cells express mutant p53 and are known to form tumors in immunocompromised mice.

Source

This cell line was established in 1971 from a metastatic site (pleural effusion) in a 25-year-old Caucasian female.

Inventors

- Germain Trempe, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center
- Jorgen Fogh, PhD, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Fogh J et al. (1977) One hundred and twenty-seven cultured human tumor cell lines producing tumors in nude mice. *Journal of the National Cancer Institute* 59: 221-226 (PubMed ID: 327080)
- Smith MA et al. (2008) SK-NEP-1 and Rh1 are Ewing family tumor lines. *Pediatric Blood & Cancer* 50: 703-706 (PubMed ID: 17154184)

MSK Tracking Code: SK2008-052

SK-UT-1: Human Uterine Leiomyosarcoma Cell Line

Description

SK-UT-1 is a human uterine leiomyosarcoma cell line that grows in adherent culture. This cell line has little or no phosphorylated retinoblastoma protein compared to the SK-UT-1B cells. SK-UT-1 cells are capable of forming tumors when inoculated in immunocompromised mice.

Source

This cell line was established in 1972 from a 75-year-old Caucasian female with a uterine mixed mesodermal tumor consistent with leiomyosarcoma grade III.

Inventors

- Lloyd J. Old, MD, former William E. Snee Chair in Cancer Immunology, Memorial Sloan Kettering Cancer Center; former Director, New York Branch, Ludwig Institute for Cancer Research
- Germaine Trempe, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Fogh J et al. (1977) One hundred and twenty-seven cultured human tumor cell lines producing tumors in nude mice. *Journal of the National Cancer Institute* 59: 221-226 (Pubmed ID: 327080)
- Ganiatsas S et al. (2001) A splice variant of Skp2 is retained in the cytoplasm and fails to direct cyclin D1 ubiquitination in the uterine cancer cell line SK-UT. *Oncog*ene 20: 3641-50 (PubMed ID: 11439327)
- Li B et al. (2013) Curcumin induces cross-regulation between autophagy and apoptosis in uterine leiomyosarcoma cells. *International Journal of Gynecological Cancer* 23: 803-808 (PubMed ID: 23532091)

MSK Tracking Code: SK1980-537

SK-UT-1B: Human Uterine Leiomyosarcoma Cell Line

Description

SK-UT-1B is a subline of the SK-UT-1 human uterine leiomyosarcoma cell line and grows in adherent culture. Although the SK-UT-1B cell line forms tumors when inoculated in immunocompromised mice, the resulting tumors are different from tumors produced by the parental SK-UT-1 cell line. This cell line displays relatively low chromosome instability, compared to other established cancer cell lines. SK-UT-1B maintains a near-diploid karyotype and is characterized by a very low percentage of polyploid cells. The SK-UT-1B cells have high levels of phosphorylated retinoblastoma protein, compared to the parental SK-UT-1 cells.

Source

This is a subline of the SK-UT-1 cell line. The parental cell line was established in 1972 from a 75-year-old Caucasian female with a uterine mixed mesodermal tumor consistent with leiomyosarcoma grade III.

Inventors

- Lloyd J. Old, MD, former William E. Snee Chair in Cancer Immunology, Memorial Sloan Kettering Cancer Center; former Director, New York Branch, Ludwig Institute for Cancer Research
- Germaine Trempe, formerly at Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Fogh J et al. (1977) One hundred and twenty-seven cultured human tumor cell lines producing tumors in nude mice. *Journal of the National Cancer Institute* 59: 221-226 (PubMed ID: 327080)
- Chen TR (1988) SK-UT-1B, a human tumorigenic diploid cell line. *Cancer Genetics and Cytogenetics* 33: 77-81 (PubMed ID: 3383166)
- Mao X et al. (2008) Subtle genomic alterations and genomic instability revealed in diploid cancer cell lines. *Cancer Letters* 267: 49-54 (PubMed ID: 18407410)

MSK Tracking Code: SK2010-071

Human Astrocyte Cell Lines Expressing Wildtype IDH1 or Mutant IDH1 (R132H)

Description

This is a pair of isogenic human astrocyte cell lines, immortalized with hTERT and SV60 and expressing either wildtype (WT) or mutant IDH1 (R132H). The mutant cells produce 2-hydroxyglutarate (2HG), which is an oncometabolite. The cells have been passaged for up to 40 generations. The later generations demonstrate the glioma hypermethylator phenotype. The mutant cells (but not the WT) efficiently form neurospheres in suspension culture and express glioma stem cell markers. This cell set is a system for studying the biology of the IDH1 (R132H) mutation and for screening compounds to target this oncogene.

Source

WT and mutant IDH1 (R132H) were cloned into the pLNCX2 lentiviral vector. These constructs were used to produce lentiviruses, which were then used for infection of the immortalized human astrocyte cell line. Stable cells were selected using the antibiotic G418.

Inventors

- Timothy A. Chan, MD, PhD, Associate Attending, Department of Radiation Oncology, Memorial Hospital; and Laboratory Head, Human Oncology and Pathogenesis Program, Memorial Hospital Research Laboratories, Memorial Sloan Kettering Cancer Center
- Sevin Turcan, PhD, Research Fellow, Sloan Kettering Institute, Memorial Sloan Kettering Cancer Center

Key References

- Turcan S et al. (2012) IDH1 mutation is sufficient to establish the glioma hypermethylator phenotype. *Nature* 483: 479-483 (PubMed ID: 22343889)

Comments

Expression of the IDH1 (R132H) mutant is known to remodel the epigenome of cells over time. Both early and late passages of the human astrocyte cell lines expressing WT or mutant IDH1 (R132H) are available.

MSK Tracking Code: SK2012-11

SK1040: The Ef-Luc Mouse

Description

The Ef-Luc mouse is a transgenic mouse model expressing luciferase driven by an E2F1 responsive promoter used for the sensitive, non-invasive, in vivo detection of tumor growth. It is patent protected by U.S. Patent 7,041,869.

E2F1 is a transcription factor whose activity is repressed by the retinoblastoma protein (Rb), a master regulator of cell-cycle progression through the G1 to S transition. A common feature in many distinct types of human malignancies is the loss of Rb function, resulting in upregulation of E2F1 transcriptional activity and dysregulation of cell-cycle control. Therefore, the Ef-Luc mouse can be considered a general reporter animal useful for the detection and imaging of multiple different tumor types. Tumor formation as well as efficacy of anticancer treatment can be monitored over time using a single Ef-Luc Mouse.

The Ef-Luc mouse is an ideal tool for monitoring cell-cycle activity during tumor development in a living animal using bioluminescence imaging. Areas of abnormally high cell proliferation in the Ef-Luc mouse, namely cancerous cells, drive expression of luciferase.

The resulting luciferase can be detected by injection of the Ef-Luc mouse with the luciferase substrate luciferin; luciferase oxidization of luciferin produces light that is then detected through the body of the mouse and is proportional to tumor cell burden.

Patent

The Ef-Luc Mouse is patent protected: U.S. Patent 7,041,869.

Advantages

- High sensitivity allows detection of small subcutaneous tumors (<1,000 cancer cells) and deeper lesions (1-3 cm deep), which can be undetectable by standard measurement methods.
- Universal tumor detection increases the applicability of the Ef-Luc mouse model to multiple tumor types.
- Quantitative measurement of tumor burden reveals subtle changes in tumor growth.
- Rapid real-time imaging allows spatial and temporal resolution of tumor growth.
- This noninvasive method with minimal toxicity allows repeated imaging of a single animal. Fewer mice are needed per study, which reduces the cost of animal studies.

Key References

- Uhrbom L, et al. (2004) Nature Medicine. Nov; 10(11):1257-1260.

Lead Inventor

- Eric C. Holland, MD, PhD, formerly of MSK

SK2011-042. Conditional ASXL1 Knock-out Mouse Model

Description

Mutations in Additional Sex Combs-Like 1 (ASXL1) were described in bcr-abl1 negative myeloproliferative neoplasms (MPN). These mutations are common in myelomonocytic leukemias, secondary acute myeloid leukemias, including blast-phase MPN, and in myelodysplastic syndromes, and they are associated with worsened overall survival.

As a research tool, this mouse model offers promising potential to investigators seeking insights into epigenetic modifiers of signal transduction in myeloproliferative disorders. It may therefore help facilitate the development of biomarkers, new drugs, and/or novel treatment regimens.

Source

This knock-out mouse was made at InGenious, and was developed by MSK investigators in collaboration with investigators at NYU.

Lead Inventor

- Ross Levine, MD, Director, MSK Center for Hematologic Malignancies, and Laboratory Head, Human Oncology & Pathogenesis Program, MSK

SK2011-043. Conditional BAP1 Knock-out Mouse Model

Description

The BAP1 nuclear deubiquitinase is known to target histones (together with ASXL1 as a Polycomb repressor subunit) and the HCF1 transcriptional co-factor. Mutations in BAP1 thus far have been most strongly associated with an increased risk of developing mesothelioma and uveal melanoma.

As a research tool, this mouse model offers promising potential to investigators seeking insights into epigenetic modifiers of signal transduction in myeloproliferative disorders. It may therefore help facilitate the development of biomarkers, new drugs, and/or novel treatment regimens.

Source

This mouse was generated entirely by MSK investigators using EUCOM ES cells.

Lead Inventor

- Ross Levine, MD, Director, MSK Center for Hematologic Malignancies, and Laboratory Head, Human Oncology & Pathogenesis Program, MSK

SK2011-047. MAD2 Overexpressing Mice

Description

MSK's MAD2 overexpressing mice are available for license as a research tool. The mitotic checkpoint protein hsMad2 is required to arrest cells in mitosis when chromosomes are unattached to the mitotic spindle. The presence of a single, lagging chromosome is sufficient to activate the checkpoint, producing a delay at the metaphase-anaphase transition until the last spindle attachment is made. Complete loss of the mitotic checkpoint results in embryonic lethality owing to chromosome mis-segregation in various organisms.

Investigators have also found that Mad2+/- mice develop lung tumors at high rates after long latencies, implicating defects in the mitotic checkpoint in tumorigenesis.

Lead Inventor

- Robert Benezra, PhD, Laboratory Head, Cancer Biology & Genetics Program, Sloan Kettering Institute, MSK

References

- Michel LS et al. (2001) MAD2 haplo-insuficiancy causes premature anaphase and chromosome instability in mammalian cells. Nature Jan. 18;409(6818): 355-9 (PMID: 11201745)

MSK-PCa1: Human Prostate Cancer Organoids

Description

MSK-PCa1 is a human prostate cancer organoid cell line. In culture, the tumor cells formed organoids that displayed an intra-ductal pattern reminiscent of the primary cancer. IHC showed that the organoids were positive for pan-cytokeratin and negative AR, recapitulating the biopsy specimen. It could be grafted in SCID mice and tumors formed that recapitulated both the histological and immunohistological pattern of the patient sample. The established organoid line exhibits a double time of 3 days.

Source

MSK-PCa1 is derived from a patient who underwent radical prostatectomy for a Gleason 4+5 adenocarcinoma with prominent intraductal features. He developed a metastatic lesion in the L2 vertebral body two months after surgery and was treated with androgen deprivation therapy (ADT) and radiotherapy. Six months later, the lesion progressed despite castrate level of testosterone and a nondetectable PSA. A biopsy was taken during a kyphoplasty procedure and was cultured.

Inventors

- Yu Chen, MD, PhD, Human Oncology and Pathogenesis Program, Department of Medicine, Memorial Sloan Kettering Cancer Center
- Dong Gao, PhD, formerly at Human Oncology and Pathogenesis Program, Department of Medicine, Memorial Sloan Kettering Cancer Center

Key References

- Gao D, et al. Organoid Cultures Derived from Patients with Advanced Prostate Cancer. Cell, 2014;159(1):176-87 (PubMed: 25201530)

MSK-PCa2: Human Prostate Cancer Organoids

Description

Organoid cultures recapitulated the adenocarcinoma features, and IHC showed the cells were diffusely positive for AR and pan-cytokeratin, matching the tumor sample. When grafted into the renal capsule of SCID mice, the tumors were histologically indistinguishable from the metastatic lesion and stained diffusely positive for pan-cytokeratin and AR. The established organoid line exhibits a double time of 3 days.

Source

MSK-PCa2 is derived from patient who presented with metastatic adenocarcinoma. He had a short-term response to ADT for 6-months and developed an expanding hip metastasis requiring arthroplasty.

Inventors

- Yu Chen, MD, PhD, Human Oncology and Pathogenesis Program, Department of Medicine, Memorial Sloan Kettering Cancer Center
- Dong Gao, PhD, formerly at Human Oncology and Pathogenesis Program, Department of Medicine, Memorial Sloan Kettering Cancer Center

Key References

- Gao D, et al. Organoid Cultures Derived from Patients with Advanced Prostate Cancer. Cell, 2014;159(1):176-87 (PubMed: 25201530).

MSK-PCa3: Human Prostate Cancer Organoids

Description

The organoids from the biopsy showed a similar mucinous histology that was diffusely positive for AR and pan-cytokeratin. When grafted into mice, the tumor exhibited the identical histology as well as positivity for AR and pan-cytokeratin. The established organoid line exhibits a double time of 1 week.

Source

MSK-PCa3 is derived from a patient who presented with metastatic prostate cancer to the pelvic lymph nodes with a diagnosis PSA of only 1.53 ng/dl. Biopsy showed adenocarcinoma with mucinous features that was positive for AR but negative for PSA. Despite a PSA response to <0.05 ng/dl, the patient progressed radiographically and a second biopsy was performed.

Inventors

- Yu Chen, MD, PhD, Human Oncology and Pathogenesis Program, Department of Medicine, Memorial Sloan Kettering Cancer Center
- Dong Gao, PhD, formerly at Human Oncology and Pathogenesis Program, Department of Medicine, Memorial Sloan Kettering Cancer Center

Key References

- Gao D, et al. Organoid Cultures Derived from Patients with Advanced Prostate Cancer. Cell, 2014;159(1):176-87 (PubMed: 25201530).

MSK-PCa4: Human Prostate Cancer Organoids

Description

Cultured organoids showed typical small cell morphology with high nucleus to cytoplasm ratio that stained positive for synaptophysin. Organoids with weaker synaptophysin staining showed perinuclear pancytokeratin staining typical of small cell prostate cancer, while those with stronger synaptophysin staining were pan-cytokeratin negative. When grafted into immuno-compromised mice, the tumors exhibited typical small cell histology with high nuclear to cytoplasmic ratio and frequent mitotic figures. The established organoid line exhibits a double time of 2 weeks.

Source

MSK-PCa4 is developed from a patient with Gleason 4+3 adenocarcinoma seven years after initial diagnosis and treatment with combined ADT and radiotherapy. The restarted ADT with progression of disease five years after diagnosis, subsequently progressed despite PSA of < 1 ng/dl. A malignant pleural effusion showed prostate cancer with neuroendocrine differentiation that was positive for synaptophysin.

Inventors

- Yu Chen, MD, PhD, Human Oncology and Pathogenesis Program, Department of Medicine, Memorial Sloan Kettering Cancer Center
- Dong Gao, PhD, formerly at Human Oncology and Pathogenesis Program, Department of Medicine, Memorial Sloan Kettering Cancer Center

Key References

- Gao D, et al. Organoid Cultures Derived from Patients with Advanced Prostate Cancer. Cell, 2014;159(1):176-87 (PubMed: 25201530).

MSK-PCa5: Human Prostate Cancer Organoids

Description

The organoids were from CD45 depleted buffy coat, and showed small and showed diffusely positive pan-cytokeratin and variable AR staining. The established organoid line exhibits a double time of 1 week.

Source

MSK-PCa5 is developed from a patient who developed widespread metastatic disease only five months after prostatectomy for radiographically localized Gleason 4+5 prostate cancer. He had a brief PSA response to ADT and a subsequent sustained PSA response to abiraterone acetate despite radiographic progression of disease.

Inventors

- Yu Chen, MD, PhD, Human Oncology and Pathogenesis Program, Department of Medicine, Memorial Sloan Kettering Cancer Center
- Dong Gao, PhD, formerly at Human Oncology and Pathogenesis Program, Department of Medicine, Memorial Sloan Kettering Cancer Center

Key References

- Gao D, et al. Organoid Cultures Derived from Patients with Advanced Prostate Cancer. Cell, 2014;159(1):176-87 (PubMed: 25201530).

MSK-PCa6: Human Prostate Cancer Organoids

Description

We cultured the extraprostatic tissue devoid of normal prostate glands, and the organoids had squamous features and displayed positive pan-cytokeratin and very low AR staining. The established organoid line exhibits a double time of 5 days.

Source

MSK-PCa6 is developed from a patient who underwent combined ADT and radiotherapy for Gleason 4+3 adenocarcinoma. Seven years after diagnosis, he developed symptomatic local recurrence with low PSA. Salvage prostatectomy showed residual adenocarcinoma with extensive squamous differentiation and invasion into the bladder and rectum.

Inventors

- Yu Chen, MD, PhD, Human Oncology and Pathogenesis Program, Department of Medicine, Memorial Sloan Kettering Cancer Center
- Dong Gao, PhD, formerly at Human Oncology and Pathogenesis Program, Department of Medicine, Memorial Sloan Kettering Cancer Center

Key References

- Gao D, et al. Organoid Cultures Derived from Patients with Advanced Prostate Cancer. Cell, 2014;159(1):176-87 (PubMed: 25201530).

MSK-PCa7: Human Prostate Cancer Organoids

Description

Organoids from the lymph node showed adenocarcinoma cribriform growth pattern (Gleason pattern 4) that was diffusely positive for AR and pan-cytokeratin, matching the tumor tissue. The established organoid line exhibits a double time of 3 weeks.

Source

MSK-PCa7, the only sample from clinically hormone-sensitive disease, is developed from a patient who underwent prostatectomy for Gleason 4+3 adenocarcinoma. He developed metastatically a single, radiographically enlarged, pelvic lymph node and underwent surgical resection of the lymph node.

Inventors

- Yu Chen, MD, PhD, Human Oncology and Pathogenesis Program, Department of Medicine, Memorial Sloan Kettering Cancer Center
- Dong Gao, PhD, formerly at Human Oncology and Pathogenesis Program, Department of Medicine, Memorial Sloan Kettering Cancer Center

Key References

- Gao D, et al. Organoid Cultures Derived from Patients with Advanced Prostate Cancer. Cell, 2014;159(1):176-87 (PubMed: 25201530).

MSK-LX29

Sex: Female

Histology: Adenocarcinoma

Key Mutations: EGFR L858R, TP53 R248Q, MET Amplified, ERBB2 Amplified

Molecular Characteristics: MSK-IMPACT

Matched Normal: Yes

Treatment: erlotinib

Site: Lung

Paired: No

Comments: erlotinib resistant

MSK-LX40

Sex: Male

Histology: Small cell lung cancer

Key Mutations: TP53 H179R, RB1 S567*, NOTCH1 P2Rfs*31, MYCL Amplified

Molecular Characteristics: MSK-IMPACT, whole exome sequencing

Matched Normal: Yes

Treatment: LDE225 + cisplatin + etoposide

Site: Lung

Paired: Yes

Comments: Chemosensitive relapse

MSK-LX40-R

Sex: Male

Histology: Small cell lung cancer

Key Mutations: TP53 H179R, RB1 S567*, NOTCH1 P2Rfs*31, MYCL Amplified

Molecular Characteristics: MSK-IMPACT, whole exome sequencing

Matched Normal: Yes

Treatment: LDE225 + cisplatin + etoposide, extensive cisplatin and etoposide treatment in PDX

Site: Lung

Paired: Yes

Comments: Chemoresistant

MSK-LX55

Sex: Male

Histology: Adenocarcinoma

Key Mutations: EML4-ALK Fusion

Molecular Characteristics: *Yes*

Matched Normal: Yes

Treatment: crizotinib

Site: Lung

Paired: No

Comments: crizotinib resistant

MSK-LX95

Sex: Male

Histology: Small cell lung cancer

Key Mutations: TP53 G154Afs*16, RB1 X203_splice, PTEN L181Wfs*13, MYCN Amplified

Molecular Characteristics: MSK-IMPACT, whole exome sequencing

Matched Normal: Yes

Treatment: cisplatin + etoposide

Site: Lung

Paired: Yes

Comments: Chemosensitive relapse

MSK-LX95-R

Sex: Male

Histology: Small cell lung cancer

Key Mutations: TP53 G154Afs*16, RB1 X203_splice, PTEN L181Wfs*13, MYCN Amplified

Molecular Characteristics: MSK-IMPACT, whole exome sequencing

Matched Normal: Yes

Treatment: cisplatin + etoposide, extensive cisplatin and etoposide treatment in PDX

Site: Lung

Paired: Yes

Comments: Chemoresistant

MSK-LX285

Sex: Male

Histology: Adenocarcinoma

Key Mutations: EGFR L858R, EGFR T790M, EGFR Amplified

Molecular Characteristics: MSK-IMPACT

Matched Normal: Yes

Treatment: erlotinib

Site: Lung

Paired: No

Comments: erlotinib resistant

NON-NEGOTIABLE NON-EXCLUSIVE INDUSTRY LICENSE AGREEMENT FOR MSK CELL LINES FOR RESEARCH USE ONLY

Table of Contents

PREAMBLE ARTICLES:

This non-exclusive license agreement (hereinafter referred to as "Agreement") is effective on the date of the last to subscribe below (hereinafter referred to as "Effective Date"), and is entered by and between **MEMORIAL SLOAN KETTERING CANCER CENTER, SLOAN KETTERING INSTITUTE FOR CANCER RESEARCH and MEMORIAL HOSPITAL FOR CANCER AND ALLIED DISEASES** (hereinafter referred to as "MSK"), a New York state not-for-profit corporation with principal offices at 1275 York Avenue, New York, New York 10065, and _____, a for-profit corporation with principal offices located at _____(hereinafter referred to as "LICENSEE").

WITNESSETH

WHEREAS, MSK is the owner of MATERIAL, as defined below; and

WHEREAS, LICENSEE desires to use MATERIAL, as defined below; and

WHEREAS, MSK desires to have the MATERIAL utilized in the public interest and is willing to grant a license to its interest thereunder and to provide such MATERIAL to LICENSEE; and

WHEREAS, LICENSEE desires to obtain said license.

NOW, THEREFORE, in consideration of the premises and the mutual covenants contained herein and for other good and valuable consideration, the receipt and sufficiency of which are hereby acknowledged, the parties intending to be legally bound hereto agree as follows:

ARTICLE I – PREAMBLE AND RECITALS

The foregoing preamble and recitals are hereby incorporated and made a part of this Agreement.

ARTICLE II – DEFINITIONS

For the purpose of this Agreement, the following words and phrases shall have the following meanings:

2.1 "Affiliate" shall mean any person, firm, corporation or other entity controlling, controlled by, or under common control with a party hereto. The term "control" wherever used throughout this Agreement shall mean ownership, directly or indirectly, of more than fifty percent (50%) of the ownership interest.

2.2 "MATERIAL" shall mean the cell line(s) LICENSEE selected in Section 3.1, developed by MSK, together with any unmodified derivatives, parts, products, progeny and purified or fractionated subsets.

2.3 "CRO" shall mean a contract research organization that provides research services to LICENSEE on a fee-for-service contract basis and has no interest in the results of these research services for its own purposes.

2.4 "Modifications" shall mean any substances created by LICENSEE which contain or incorporate the MATERIAL.

2.5 "Field of Use" shall mean the use of the MATERIAL or Modifications as a research reagent for internal research only. Specifically *excluded* from the Field of Use are i) any therapeutic, prophylactic or diagnostic uses either for human or veterinary applications; ii) any use which requires regulatory approval; or iii) any sale of or incorporation of the MATERIAL or Modifications into a commercial product or service for sale.

ARTICLE III – GRANT

3.1 Cell Lines:

- SK-BR-3 (Human Adenocarcinoma Cell Line: ATCC HTB-30)
- BE(2)-C (Human Neuroblastoma Cell Line: ATCC CRL-2268)
- CAMA-1 (Human Breast Cancer Cell Line: ATCC HTB-21)
- HT-29 (Human Colorectal Adenocarcinoma Cell Line: ATCC HTB-38)
- SK-CO-1 (Human Colorectal Adenocarcinoma Cell Line: ATCC HTB-39)
- SK-MEL-1 (Human Melanoma Cell Line: ATCC HTB-67)
- SK-MEL-2 (Human Melanoma Cell Line: ATCC HTB-68)
- SK-MEL-3 (Human Melanoma Cell Line: ATCC HTB-69)
- SK-MEL-5 (Human Melanoma Cell Line: ATCC HTB-70)
- SK-MEL-24 (Human Melanoma Cell Line: ATCC HTB-71)
- SK-MEL-28 (Human Melanoma Cell Line: ATCC HTB-72)
- SK-MEL-31 (Human Melanoma Cell Line: ATCC HTB-73)
- SH-SY5Y (Human Neuroblastoma Cell Line: ATCC CRL-2266)
- SK-N-MC (Human Neuroblastoma Cell Line: ATCC HTB-10)
- SK-N-SH (Human Neuroblastoma Cell Line: ATCC HTB-11)
- SK-N-BE(2) (Human Neuroblastoma Cell Line: ATCC CRL-2271)
- Saos-2 (Human Osteosarcoma Cell Line: ATCC HTB-85)
- Caov-3 (Human Ovarian Cancer Cell Line: ATCC HTB-75)
- Caov-4 (Human Ovarian Cancer Cell Line: ATCC HTB-76)
- Calu-1 (Human Lung Adenocarcinoma Cell Line: ATCC HTB-54)
- Calu-3 (Human Lung Adenocarcinoma Cell Line: ATCC HTB-55)
- Calu-6 (Human Lung Adenocarcinoma Cell Line: ATCC HTB-56)
- SK-OV-3 (Human Ovarian Cancer Cell Line: ATCC HTB-77)
- Capan-1 (Human Pancreatic Adenocarcinoma Cell Line: ATCC HTB-79)
- Capan-2 (Human Pancreatic Adenocarcinoma Cell Line: ATCC HTB-80)
- Caki-1 (Human Renal Cancer Cell Line: ATCC HTB-46)
- Caki-2 (Human Renal Cancer Cell Line: ATCC HTB-47)

MSK hereby grants to LICENSEE, and LICENSEE hereby accepts, a nonexclusive, non-transferable, worldwide right and license, without the right to sublicense, to use the MATERIAL and Modifications in the Field of Use, to _____ [*insert a brief description of the intended use of the cells*], for a period of [*choose one*]

- One (1) year
- Five (5) years
- Ten (10) years

from the Effective date, unless this Agreement is extended or terminated before that time according to the terms hereof, and subject to the rights reserved or observed in this Article III (hereinafter "Term").

Expressly *excluded* from the license is the right to:

i) sublicense;

ii) file patent applications claiming the MATERIAL or Modifications or uses of the MATERIAL or Modifications;

iii) use the MATERIAL or Modifications for sale, manufacture for sale, or use in the manufacture of another product or service for sale; and

iv) distribute MATERIAL or Modifications to a third party, except when said third party is an Affiliate of LICENSEE or CRO as specified in Section 3.3

3.2 Notwithstanding any other provisions of this Agreement, it is agreed that MSK and its Affiliates shall retain the right to transfer, use and distribute the MATERIAL for any purpose. All rights reserved to MSK and its Affiliates, third parties, and to the United States Government and others under 35 USC 200-212, as amended, shall remain and shall in no way be affected by this Agreement.

3.3 Name of CRO _____

Notwithstanding any other provisions of this Agreement, it is understood that LICENSEE may transfer the MATERIAL and Modifications to a CRO to conduct research on behalf of LICENSEE provided, however, said CRO shall use the MATERIAL and Modifications under terms and conditions at least as restrictive as those set forth in this Agreement. Furthermore, LICENSEE is obligated to verify that any MATERIAL and Modifications transferred to a CRO is destroyed upon completion of LICENSEE's research.

ARTICLE IV – PAYMENTS

4.1 For the rights, privileges and licenses granted hereunder, LICENSEE shall pay to MSK, in the manner hereinafter provided, until this Agreement shall terminate as hereinafter provided, a one-time, non-creditable, non-refundable, non-discountable license issue fee hereinafter provided, due upon the Effective Date and payable within thirty (30) days of receipt of an invoice from MSK for such sum.

A). If no high-throughput screening, a license fee of:

- Two Thousand Five Hundred dollars ($2,500) per cell line for a one (1) year term
- Five Thousand dollars ($5,000) per cell line for a five (5) year term
- Seven Thousand Five Hundred dollars ($7,500) per cell line for a ten (10) year term

B). If used for high-throughput screening only, excluding any other use:

A one-time license fee per cell line of:

- Ten Thousand dollars ($10,000)

C). If used for high-throughput screening as well as internal research:

License fees shall consist per cell line of A). and B).

4.2 All payments shall be made using MSK's credit card payment link.

Payment Link:
pay.usbank.com/Form/Payments/New?id=mskcc_rtmtechdev

Please reference the license fee $_____ and SK#_____(note: *SK # provided by MSK)*

If LICENSEE is unable to pay by credit card, a request can be made to Alexandra Buga bugaa@mskcc.org for an invoice.

4.3 All invoices shall reference PO# _____, LICENSEE and shall be sent to:

_____

_____

_____

All payments shall be made by remittance to Memorial Sloan Kettering Cancer Center (Tax Payer ID 13-1924236). Payment shall show, "**Payment, Contract SK**_____" (note: *SK # provided by MSK*) on the check stub, shall include the applicable invoice, and shall be sent to:

Memorial Sloan Kettering Cancer Center
PO Box 29035
New York, New York 10087

4.4 LICENSEE is responsible for obtaining the MATERIAL from the American Tissue Culture Collection (ATCC), at its own expense.

ARTICLE V – OPTION TO RENEW

LICENSEE may, at its option, extend the term of this Agreement by providing written notification to MSK, prior to the termination of this Agreement and by remittance of a renewal fee of One Thousand U.S. dollars ($1,000) per cell line, for each additional year that LICENSEE wishes to extend the term.

ARTICLE VI – INDEMNIFICATION AND PRODUCT LIABILITY

6.1 LICENSEE shall at all times during the term of this Agreement and thereafter, indemnify, defend and hold MSK, its Affiliates, trustees, Board of Managers, officers, employees, agents, and contractors, harmless against all claims and expenses, including legal expenses and reasonable attorneys' fees, arising out of the death of or injury to any person or persons or out of any damage to property and against any other claim, proceeding, demand, expense and liability of any kind whatsoever resulting from the development, production, manufacture or use of the MATERIAL or Modifications, or arising from any obligation of LICENSEE hereunder, except to the extent that such liability or claims arise from the gross negligence or willful misconduct of MSK.

6.2 For the term of this Agreement, upon the commencement of use of any MATERIAL or Modification, LICENSEE shall obtain and carry in full force and effect liability insurance. The nature and extent of the insurance coverage shall be commensurate with usual and customary industry practices, as determined by LICENSEE's good faith assessment.

6.3 It is understood that MSK is subject to United States laws and regulations controlling the export of technical data, computer software, laboratory prototypes and other commodities (including the Arms Export Control Act, as amended and the Export Administration Act of 1979), and that its obligations hereunder are contingent on compliance with applicable United States export laws and regulations. The transfer of certain technical data and commodities may require a license from the cognizant agency of the United States Government and/or written assurances by LICENSEE that LICENSEE shall not export data or commodities to certain foreign countries without prior approval of such agency. MSK neither represents that a license shall not be required nor that, if required, it shall be issued.

6.4 Except as otherwise expressly set forth in this Agreement, MSK MAKES NO REPRESENTATIONS AND EXTENDS NO WARRANTIES OF ANY KIND, EITHER EXPRESS OR IMPLIED, INCLUDING BUT NOT LIMITED TO WARRANTIES OF MERCHANTABILITY, FITNESS FOR A PARTICULAR PURPOSE, OR THAT ANY USE OF THE MATERIAL WILL NOT INFRINGE ON ANY PATENT, COPYRIGHT, TRADEMARK, OR OTHER PROPRIETARY RIGHTS.

ARTICLE VII – PUBLICITY AND NON-USE OF NAMES

7.1 Except as required by law, neither MSK nor LICENSEE shall release any information, publicity, news release or other public announcement or publication regarding the terms of this Agreement or performance hereunder without the prior written approval of the other party.

7.2 Neither party shall use the name of the other party, nor any of its employees, nor any adaptation thereof, in any advertising, marketing, promotional or commercial literature without prior written consent obtained from the other party in each case, except as may be required by law.

7.3 Notwithstanding the above, in the event LICENSEE publishes the results of its research with the MATERIALS or Modifications, relevant MSK employees and the source of the MATERIAL shall be acknowledged in accordance with academic standards.

ARTICLE VIII – TERMINATION

8.1 MSK may terminate this Agreement if LICENSEE becomes insolvent or, a petition in bankruptcy is filed against LICENSEE and is consented to, acquiesced in or remains undismissed for ninety (90) days; or makes a general assignment for the benefit of creditors, or a receiver is appointed for LICENSEE, and LICENSEE does not return to solvency before the expiration of a thirty (30) day period.

8.2 Should LICENSEE fail to pay MSK fees due and payable hereunder for more than thirty (30) days, MSK shall have the right to terminate this Agreement on thirty (30) days written notice, unless LICENSEE shall pay MSK within the thirty (30) day period all such fees due and payable. Upon the expiration of the thirty (30) day period, if LICENSEE shall not have paid all such license fees and interest due and payable, the rights, privileges and license granted hereunder shall terminate. The payment shall accrue interest beginning the tenth day following the due date, calculated at the annual rate of the sum of: a) two percent (2%) plus (b) the prime interest rate quoted by the Wall Street Journal on the date said payment is due.

8.3 Upon any breach of this Agreement by LICENSEE, other than those occurrences set out in Sections 8.1 and 8.2, which shall always take precedence in that order over any material breach or default referred to in this Section 8.3, MSK shall have the right to terminate this Agreement and the rights, privileges and license granted hereunder by thirty (30) days' notice to LICENSEE. Such termination shall become effective unless LICENSEE shall have cured any such breach prior to the expiration of the thirty (30) day period.

8.4 LICENSEE shall be entitled to terminate this Agreement upon thirty (30) days advance written notice to MSK, in the event of MSK's material breach of any of the provisions of this Agreement, which breach is not cured (if capable of being cured) within this thirty (30) day period.

8.5 Upon termination of this Agreement for any reason, all rights granted herein shall cease and revert to MSK for the sole benefit of MSK and nothing herein shall be construed to release either party from any obligation that matured prior to the effective date of such termination, including the obligation to remit any and all fees accrued prior to such termination. LICENSEE must return to MSK or, at MSK's written request, destroy all MATERIAL and Modifications.

8.6 Other than any claim arising from LICENSEE's failure to pay license fees due under this contract, any controversy or bonafide disputed claim arising between the parties to this Agreement, which dispute cannot be resolved by mutual agreement shall, by the election of either party, be resolved by submitting to dispute resolution before a fact-finding mediation body composed of one or more experts in the field, selected by mutual agreement within thirty (30) days of written request by either party. Said dispute resolution shall be held in New York at such place as shall be mutually agreed upon in writing by the parties. The fact-finding body shall determine who shall bear the cost of said resolution. In the event that the parties cannot mutually agree within said thirty (30) days on the dispute resolution body, the parties will apply the procedural rules of a mutually agreeable forum.

8.7 Articles VI, VII, X and this Section 8.8 of this Agreement shall survive termination.

ARTICLE IX – NOTICES AND OTHER COMMUNICATIONS

Any notice or other communication pursuant to this Agreement shall be sufficiently made or given on the date of mailing if sent to such party by certified first class mail, postage prepaid, addressed to it at its address below or as it shall designate by written notice given to the other party:

In the case of MSK:

Memorial Sloan Kettering Cancer Center

Attention: Yashodhara Dash, Ph.D.
Director, Technology Management and Commercialization

With a copy to:

Attention: Shilpi Banerjee, Ph.D., J.D.
Chief Intellectual Property Counsel

If by mail: 1275 York Avenue, Box 524
New York, NY 10065

If by courier: Office of Technology Development
600 3rd Avenue 16th Floor
New York, NY 10016

In the case of LICENSEE:

_____

_____

_____

Attention: _____

_____

ARTICLE X - MISCELLANEOUS PROVISIONS

10.1 This Agreement shall be construed, governed, interpreted and applied in accordance with the laws of the State of New York, U.S.A.

10.2 This Agreement may not be assigned by LICENSEE without prior written consent from MSK; provided, however, that LICENSEE may transfer or assign this Agreement without the prior written consent of MSK, to an Affiliate of LICENSEE or in connection with a merger, consolidation, or a sale or transfer of all or substantially all of the assets to which the purpose of this Agreement relates.

10.3 The parties hereto acknowledge that this Agreement sets forth the entire Agreement and understanding of the parties hereto as to the subject matter hereof, and shall not be subject to any change or modification except by the execution of a written instrument subscribed to by the parties hereto.

10.4 The provisions of this Agreement are severable, and in the event that any provisions of this Agreement shall be determined to be invalid or unenforceable under any controlling body of the law, such invalidity or unenforceability shall not in any way affect the validity or enforceability of the remaining provisions hereof.

10.5 The failure of either party to assert a right hereunder or to insist upon compliance with any term or condition of this Agreement shall not constitute a waiver of that right or excuse a similar subsequent failure to perform any such term or condition by the other party.

10.6 This Agreement may be executed in any number of counterparts and each of such counterparts shall for all purposes be an original and all such counterparts shall together constitute but one and the same agreement.

IN WITNESS WHEREOF, the parties have hereunto set their hands and seals and duly executed this Agreement the day and year set forth below.

_____

By: _____

Name: _____

Title: _____

Date: _____

MEMORIAL SLOAN KETTERING CANCER CENTER

By: _____

Name: Gregory S. Raskin, MD

Title: Vice President, Technology Development

Date: _____