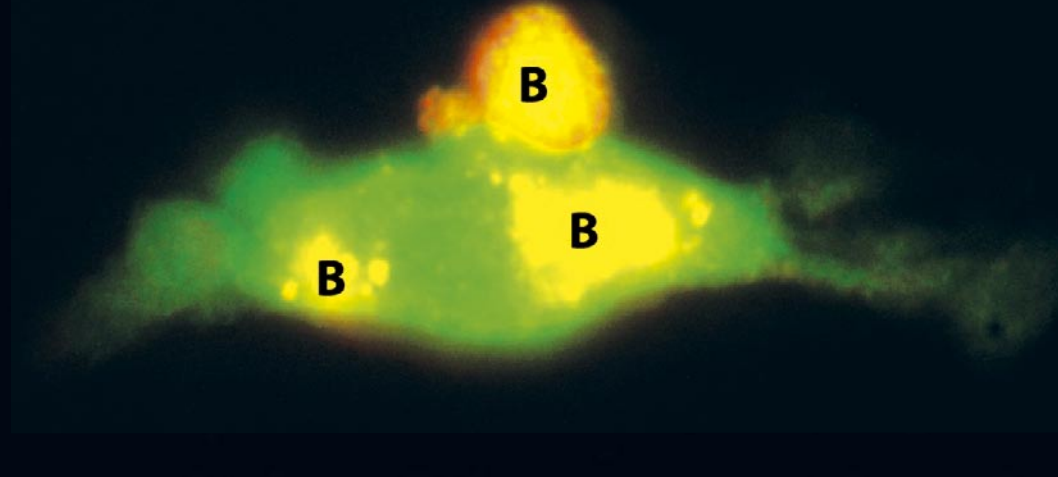




CD133 – the pluripotent stem cell marker

CD133 – the pluripotent stem cell marker



Characterization of 6-week-old adherent cells. Adherent cells that emerged from CD133⁺ cells initially transfected with enhanced green fluorescent protein (EGFP) were counterstained with CD133/1 (AC133)-PE. The yellow fluorescence indicates double staining of the buds as a result of an overlay of green (EGFP) and red (CD133⁺) fluorescence. **B** = bud on the cell surface. Courtesy of Selim Kuçi, Tübingen, Germany

CD133 – a marker for primitive hematopoietic stem and progenitor cells

CD133 (formerly AC133) is known to be a marker of primitive hematopoietic stem and progenitor cells (HSC and HPC, respectively) and was originally found on HSCs and HPCs deriving from human fetal liver, bone marrow, and peripheral blood.¹ Phenotypical analysis of CD133-expressing cells (CD133⁺ cells) revealed a high expression on primitive hematopoietic and myeloid progenitor cells², whereas CD133⁻CD34⁺ cells were shown to mostly consist of B cell and late erythroid progenitors³ as well as other more committed hematopoietic progenitors⁴. Moreover, functional studies showed that CD133 is dimly or not expressed on late progenitors, such as pre-B cells, CFU-E (colony forming unit–erythrocytes), and CFU-G (colony forming unit–granulocytes), and populations of CD133⁺ cells are highly enriched in long-term culture initiating cells (LTC-ICs), the most primitive human hematopoietic cells which can be assayed *in vitro*.^{4,5} Thus, CD133⁺ cells appear to be ancestral to CD34⁺ cells, especially as the latter can be generated *in vitro* from CD133⁺CD34⁻ cells⁶, and CD133⁺ cells display a higher proliferative activity^{6,7}. Populations of CD133⁺ cells are also able to repopulate severe combined immunodeficient (SCID) mice, and can serve as precursors to functionally active dendritic cells^{4,8} and neutrophils⁹. CD133-selected cells are currently under investigation for use in stem cell transplantation for the reconstitution of the hematopoietic system after irreversible damage, e.g. by chemotherapy.⁸

CD133 – a marker for stem cells with pluripotent plasticity

CD133, the structural homolog of murine prominin-1¹⁰, is regarded as an important marker for the identification and isolation of primitive stem and progenitor cells in both hematopoietic and nonhematopoietic tissues. CD133-expressing stem and progenitor cells can be found in liver, muscle, kidney, prostate, and neural tissues and have been shown *in vitro* to be capable of induced differentiation to endothelial cells, neural cells, hepatocytes, myocytes, and osteoblasts. CD133⁺ cells isolated from mobilized peripheral blood partly adhere to plastic and become negative for CD133 expression. After several weeks of cultivation, adherent cells are capable of giving rise to non-adherent CD133⁺ cells.^{11,12} Transplantation of these non-adherent CD133⁺ cells into NOD/SCID mice not only induced a multilineage human hematopoiesis, including T cells and NK cells, but CD133⁺ were also present in the liver, lung, brain, heart, gut, and striated muscle of the mice, indicating that CD133⁺ cells can repopulate these organs, sustain tissue-specific regenerating processes, show SCID-repopulating potential, and thus possess the pluripotent capacity to differentiate into tissues of mesodermal, ectodermal and endodermal origin.^{11,12} Furthermore, adherent cells show the potential to differentiate to muscle-, liver- and neural-like cells *in vitro*.¹³

Epithelial cells

Liver

The liver is well known for possessing a large capacity for regeneration after resection, therefore tissue regeneration and cell therapies are being discussed as alternative solutions to organ transplantation in severe liver disorders. The appearance of donor-derived hepatocytes after HSC transplantation¹⁴ has also given support to theories of regenerated tissue originating from circulating stem cells: intraportal administration of CD133⁺ bone marrow-derived stem cells successfully resulted in an increased regeneration of liver tissue after partial resection.¹⁵ Following liver injury, transplanted human HSC/HPC populations from bone marrow and cord blood also had the capacity to generate cells within the recipient liver that synthesized and secreted human albumin into the sera of mice.¹⁶ Furthermore, CD133⁺ CD117⁺ CD45⁻ Tryptase⁻ cells are discussed to be circulating liver progenitors which are also found in the cellular infiltrate during massive hepatic necrosis (MHN).¹⁷ *In vitro*, MAPCs (multipotent adult progenitor cells), described as being CD133⁺¹⁸, differentiated into hepatocytes expressing cytokeratin 18 and albumin and also acquired functional characteristics of hepatocytes¹⁹.

Prostate

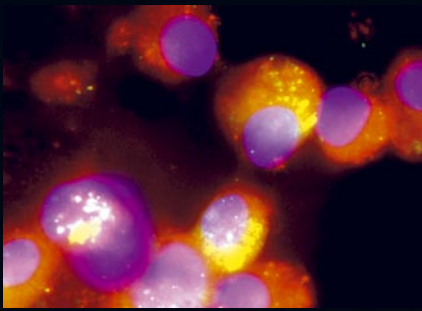
Genetic damage to stem cells in different tissues has long been linked to causing cancer, including prostate cancer and benign prostatic hyperplasia.²⁰ Further characterization of pathways governing proliferation and differentiation of epithelial stem cells is a major goal. In the prostate, CD133 expression was shown to be found on $\alpha_2\beta_1^{hi}$ basal cells²¹, which were defined as the prostate epithelial stem cell population.²² When transplanted into athymic nude mice, separated CD133⁺ cells from prostatic tissue regenerated a fully differentiated prostatic epithelium, including acini that secreted prostate-specific products.²¹

Kidney

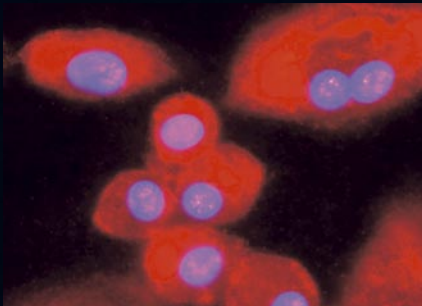
CD133⁺ cells were also isolated from the human kidney and could be differentiated *in vitro* into epithelial or endothelial cells.²³ Upon transplantation into tubulonecrotic SCID mice, CD133⁺ cells migrated to the damaged kidneys and integrated into tubules, forming tubular structures that expressed renal epithelial markers. Therefore, CD133⁺ cells deriving from the kidney may be applicable to renal tissue regeneration therapies.

Neural cells

In recent years, the long-standing principle that the central nervous system (CNS) is non-renewable has been challenged by studies showing the ability of specific cell types to perform neurogenesis. Neural stem and progenitor cells have even further come into focus for their potential to promote recovery from neurodegenerative diseases or injuries to the central nervous system. CD133⁺ cells isolated from fetal liver²⁴, umbilical cord blood²⁵, bone marrow²⁶, and mobilized blood²⁷, were capable of *in vitro* differentiation to neuronal cells as well as to astrocytes^{24,25,27}, oligodendrocytes^{25,27}, and glial cells²⁵. Similarly, CD133⁺ cells isolated from human fetal brain²⁸⁻³² or skin³³ tissues were able to form self-renewing neurospheres *in vitro*, and to differentiate into neurons^{28,33} and glia²⁸. Moreover, when injected into mice, human CD133⁺ cells differentiated into fully integrated neurons and glial cells^{29,31} as well as astrocytes and endothelial cells³². Animal models of stroke damaged brains in rats³⁰ and spinal cord injuries in mice³¹ have also demonstrated the ability of injected neural stem cells to migrate to sites of lesions as well as to differentiate into functional neuronal phenotypes. In the case of spinal cord injuries, re-myelination, locomotive recovery, and synapse formation were all observed.³¹

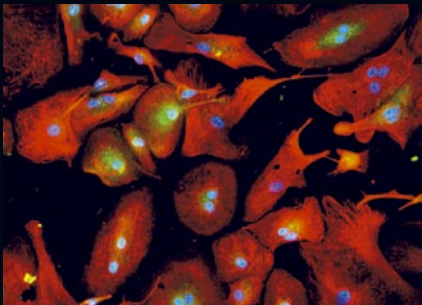


a

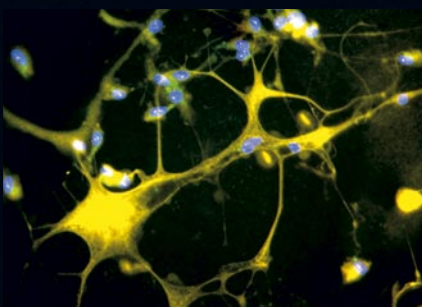


b

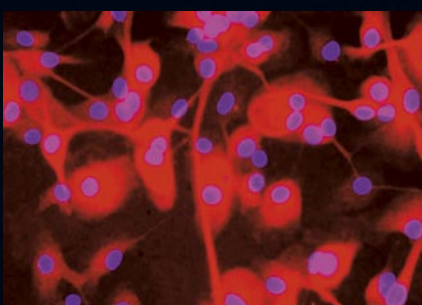
CD133⁺ cells, isolated from mobilized blood, gave rise to adherent cells after 3–5 weeks of cultivation. These cells were able to differentiate to hepatocyte-like cells. a) The cells are stained for hepatocyte nuclear factor-3 (FITC), albumin (Cy3) and nuclei (DAPI). b) The cells are stained for cytokeratin 19 (Cy3) and nuclei (DAPI) (200 \times). Courtesy of Selim Kuçi, Tübingen, Germany



a



b



c

CD133-selected cells from mobilized PBMCs were cultivated for 3–5 weeks. Adherent cells were able to differentiate to neural-like cells. a) astrocyte-like cells stained for GFAP (Cy3), EPO (FITC) and nuclei (DAPI). b) oligodendrocyte-like cells stained with GFAP (FITC), MBP (Cy3) and DAPI (nuclei) c) neuronal-like cells stained for beta-tubulin III (Cy3) and nuclei (DAPI)(200 \times). Courtesy of Selim Kuçi, Tübingen, Germany

Endothelial cells

Endothelial progenitor cells (EPCs) have been suggested to play an important role in postnatal neoangiogenesis and neovascularization, and have also come into focus for the potential treatment of ischemic or injured tissue^{34,35} and myocardial infarction³⁵⁻³⁷. Circulating EPC (cEPC) levels are also being considered as a clinically measurable parameter in the assessment of risk factors for various diseases.³⁸⁻⁴⁰ CD133⁺ cells isolated from bone marrow^{41,42}, cord blood³⁴, mobilized⁴⁰ and unmobilized^{38,43} peripheral blood are capable of giving rise to endothelial cells *in vitro* in addition to the reconstitution of the hematopoietic system *in vivo*⁹. CD133⁺ cell populations, therefore, potentially contain hemangioblasts, the common precursor of HSCs, HPCs, and EPCs.⁹ Furthermore, after transplantation into nude mice suffering from ischemic hind limb, separated CD133⁺ cord blood cells were able to incorporate into capillary networks, augment neovascularization, and improve ischemic limb salvage.³⁴

Circulating EPCs, defined by the expression of CD133, CD34 and VEGFR-2 (flk-1/KDR)⁴⁴⁻⁴⁷, were found to be mobilized in the peripheral blood after heart failure⁴⁸, during the early phase of myocardial infarction⁴⁹, vascular trauma⁵⁰, after the application of cytokines (e.g. G-CSF, GM-CSF)⁵¹, physical training^{43,52,53}, or after the cessation of smoking⁵⁴, indicating their potential to serve as a diagnostic parameter and individual risk factor. Moreover, cEPCs in chronic disease states have also been shown to be functionally impaired⁵⁵; EPCs of dialysis patients display an impaired adhesive ability and a reduced migratory activity⁵⁶, the latter also being described in sufferers of coronary artery disease⁵⁷, suggesting that the endothelial integrity itself may even be connected with the functional regenerative potential of cEPCs.

Myocytes

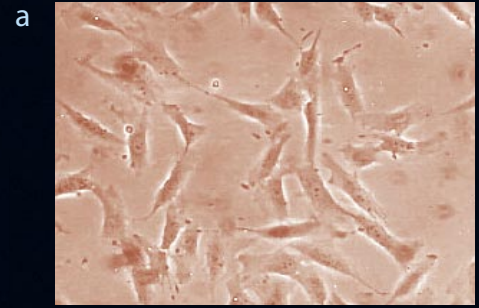
Myocytic progenitors are suggested to be a promising tool for the treatment of myopathies, e.g. to regenerate muscular dystrophies or to revitalize scar tissue generated by myocardial infarction. Circulating EPCs isolated from PBMCs were able to transdifferentiate into functionally active cardiomyocytes *in vitro* when co-cultivated with rat cardiomyocytes.⁵⁸ CD133⁺ cells isolated from PBMCs underwent myogenesis when co-cultivated with mouse myoblasts, forming myotubes, and were capable of migrating within muscle tissue and participated in dystrophin-positive muscle regeneration, myogenic satellite cell replenishment and endothelial cell differentiation when transplanted into dystrophic muscles of scid/mdx mice.⁵⁹ Furthermore, cEPCs from umbilical cord blood were also able to give rise to skeletal muscle cells when transplanted into mice.⁶⁰

The use of cord blood- or bone marrow-derived CD133⁺ cells to restore myocardial tissue viability after infarction has already been shown to be beneficial *in vivo*: CD133⁺ cells were able to migrate, colonize and survive in the infarcted myocardium and support functional recovery by preventing scar thinning and dsytolic dilatation⁶¹. CD133⁺ cells may also induce angiogenesis within the infarcted myocardium.^{36,37} The successful use of CD133⁺ fetal liver cells to generate functional cardiomyocytes *in vitro* also raises the possibility of alternative cell sources for therapeutic angiomyogenic cell transplantation.⁶² Moreover, stem cells positive for CD133 expression but negative for hematopoietic and endothelial markers such as CD34, CD45, CD31, and KDR, could be isolated from skeletal muscle by cultivation.⁶³

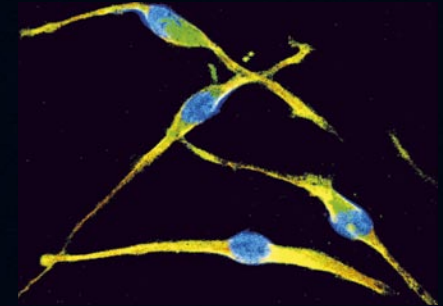
Other cell types

CD133 is found to be expressed on undifferentiated embryonic stem (ES) cell lines, e.g. H1, H7, and H9 cells^{64,65}, but becomes down-regulated when cells obtain functional and phenotypical properties upon differentiation toward hematopoietic lineages⁶⁶.

It is highly probable that all tissues contain a population of stem cells that are responsible for their continued growth and regeneration.⁶⁷ CD133⁺ cells from the stroma of human cornea have the capacity to proliferate *in vitro*; colonies derived from CD133⁺ cells could be differentiated into fibroblastic cells, indicating that CD133⁺ cells represent stem cell of the corneal stroma.⁶⁸ Stem cells with mesodermal differentiation capacity could also be highly enriched from cord blood using CD133.⁶⁹ With certainty, the list of tissues containing populations of CD133⁺ stem cells will broaden, possibly to include nearly all tissues within the human body.



Phase-contrast micrographs of EC cultures
a) CD133-selected cells after two weeks of culture. The cells grown on fibronectin form isolated colonies with cells showing a central body and short cytoplasmic dendrites (400 \times).
b) UEA-1-selected cells at confluence after three weeks of culture. The cells have a typical spindle-shaped morphology (100 \times). Courtesy of Nadia Quirici, Milan, Italy.



CD133-selected cells from mobilized PBMC were cultivated for 3-5 weeks. Adherent cells were able to differentiate to skeletal muscle-like cells. The cells are stained for Desmin (FITC), Actin (Cy3) and nuclei (DAPI) (200 \times). Courtesy of Selim Kuçi, Tübingen, Germany

References

- ¹Yin, A.H. *et al.* (1997) *Blood* 90: 5002–5012. [383]
- ²Freund, D. *et al.* (2006) *Cell Prolif.* 39: 325–332.
- ³Buhring, H.J. *et al.* (1999) *Ann. N. Y. Acad. Sci.* 872: 25–38.
- ⁴De Wynter, E.A. *et al.* (1998) *Stem Cells* 16: 387–396.
- ⁵Matsumoto, K. *et al.* (2000) *Stem Cells* 18: 196–203.
- ⁶Summers, Y.J. *et al.* (2004) *Stem Cells* 22: 704–715.
- ⁷Goussetis, E. *et al.* (2000) *J. of Hematother & Stem Cell Res.* 9: 827–840.
- ⁸Bornhauser, M. *et al.* (2005) *Leukemia* 19: 161–165.
- ⁹Loges, S. *et al.* (2004) *Stem Cells Dev.* 13: 229–42.[4218]
- ¹⁰Corbeil, D. *et al.* (1998) *Blood* 91: 2625–2626.
- ¹¹Kuçi, S. *et al.* (2002) *Stem Cell Research Customer Report* 9–11.
- ¹²Kuçi, S. *et al.* (2003) *Blood* 101: 869–876.[3966]
- ¹³Kuçi, S. *et al.* (2004) Abstract 2nd International Meeting, Stem Cell Network, North-Rhine Westphalia.
- ¹⁴Theise, N.D. *et al.* (2000) *Hepatology* 32: 11–16.
- ¹⁵Schulte am Esch II, J. *et al.* (2005) *Stem Cells* 23: 463–470.
- ¹⁶Wang, X. *et al.* (2003) *Blood* 101: 4201–4208.[4511]
- ¹⁷Craig, C. *et al.* (2004) *Semin. Liver. Dis.* 24: 49–64.[7447]
- ¹⁸Reyes, M. *et al.* (2002) *J. Clin. Invest.* 109: 337–346.[1270]
- ¹⁹Schwartz, R.E. *et al.* (2002) *J. Clin. Invest.* 109: 1291–1302.[3868]
- ²⁰Rizzo, S. *et al.* (2005) *Cell Prolif.* 38: 363–374.
- ²¹Richardson, G. *et al.* (2004) *J. Cell Sci.* 117: 3539–3545.[7531]
- ²²Collins, A. *et al.* (2001) *J. Cell Sci.* 114: 3865–3872.
- ²³Bussolati, B. *et al.* (2005) *Am. J. Pathol.* 166: 545–555.
- ²⁴Hao, H.N. *et al.* (2003) *J. Hematother. Stem Cell Res.* 12: 23–32.
- ²⁵Jang, Y.K. *et al.* (2004) *J. Neurosci. Res.* 75: 573–584.[7478]
- ²⁶Padovan, C.S. *et al.* (2003) *Cell Transp.* 12: 839–848.[4220]
- ²⁷Piechaczek, C. *et al.* (2002) *Stem Cell Research Customer Report* 2–3.
- ²⁸Uchida, N. *et al.* (2000) *PNAS* 97: 14720–14725.[933]
- ²⁹Tamaki, S. *et al.* (2002) *J. Neuro. Res.* 69: 976–986.
- ³⁰Kelly, S. *et al.* (2004) *PNAS* 101: 11839–11844.
- ³¹Cummings, B.J. *et al.* (2005) *PNAS* 102: 14069–14074.
- ³²Yu, S. *et al.* (2004) *Biotech. Let.* 26: 1131–1136.[6531]
- ³³Belicchi, M. *et al.* (2004) *J. Neurosci. Res.* 77: 475–486.[7436]
- ³⁴Yang, C. *et al.* (2004) *Thromb. Haemost.* 91: 1202–12.[7146]
- ³⁵Rafii, S. and Lyden, D. (2003) *Nat. Med.* 9: 702–712.
- ³⁶Stamm, C. *et al.* (2003) *Lancet* 361: 45–46.[4253]
- ³⁷Stamm, C. *et al.* (2004) *Thorac. Cardio. Surg.* 52: 152–158.
- ³⁸Hill, J. *et al.* (2003) *N. Engl. J. Med.* 348: 593–600.[4251]
- ³⁹Urbich, C. and Dimmeler, S. (2005) *Kidney Int.* 67: 1672–1676.
- ⁴⁰Adams, V. *et al.* (2004) *Arterioscler. Thromb. Vasc. Biol.* 24: 684–690.
- ⁴¹Quirici, N. *et al.* (2001) *Br. J. Haem.* 115: 186–194.[1949]
- ⁴²Quirici, N. *et al.* (2002) *Stem Cell Research Customer Report* 4–5.
- ⁴³Laufs, U. *et al.* (2004). (Abstract) 70. Jahrestagung der Deutschen Gesellschaft für Kardiologie- Herz- und Kreislaufforschung.
- ⁴⁴Gehling, U. *et al.* (2000) *Blood* 95: 3106–3112.[934]
- ⁴⁵Peichev, M. *et al.* (2000) *Blood* 95: 952–958.[931]
- ⁴⁶Hristov, J. *et al.* (2004) *J. Cell. Mol. Med.* 8: 498–508.
- ⁴⁷Rafii, S. (2000) *J. Clin. Invest.* 105: 71–79.
- ⁴⁸Valgimigli, M. *et al.* (2004) *Circulation* 110: 1209–1212.[7553]
- ⁴⁹Massa, M. *et al.* (2005) *Blood* 105: 199–206.
- ⁵⁰Gill, M. *et al.* (2001) *Circ. Res.* 88: 167–174.[962]
- ⁵¹Powell, T.M. *et al.* (2005) *Arterioscler. Thromb. Vasc. Biol.* 25: 296–301.
- ⁵²Rehman, J. *et al.* (2004) *J. Am. Coll. Cardiol.* 43: 2314–2318.
- ⁵³Steiner, S. *et al.* (2005) *Atherosclerosis* 181: 305–310.
- ⁵⁴Kondo, T. *et al.* (2004) *Arterioscler. Thromb. Vasc. Biol.* 24: 1442–1447.
- ⁵⁵Werner, N. and Nickenig, G. (2006) *Arterioscler. Thromb. Vasc. Biol.* 26: 112–116.
- ⁵⁶Herbrig, K. *et al.* (2004) *Am. J. Kidney Dis.* 44: 840–849.
- ⁵⁷Vasa, M. *et al.* (2001) *Circulation* 103: 2885–2890.
- ⁵⁸Rupp, S. *et al.* (2004) *Basic Res. Cardiol.* 99: 61–68.[7533]
- ⁵⁹Torrente, Y. *et al.* (2004) *J. Clin. Invest.* 114: 182–195.
- ⁶⁰Pesce, M. *et al.* (2003) *Circ. Res.* 93: e51–62.
- ⁶¹Leor, J. *et al.* (2005) *Stem Cells* 24: 772–780.
- ⁶²Schmelkov, S.V. *et al.* (2005) *Circulation* 111: 1175–1183.
- ⁶³Alessandri, G. *et al.* (2004) *Lancet* 364: 1872–1883.
- ⁶⁴Kaufman, D.S. *et al.* (2001) *PNAS* 98: 10716–10721.
- ⁶⁵Carpenter, M.K. *et al.* (2003) *Cloning Stem Cells* 5: 79–88.
- ⁶⁶Vodyanik, M.A. *et al.* (2005) *Blood* 105: 617–626.
- ⁶⁷Majka, M. *et al.* (2005) *Acta Biochem. Pol.* 52: 353–358.
- ⁶⁸Thill, M. (2004) *Invest. Ophthalmol. Vis. Sci.* 45: 3519.[7392]
- ⁶⁹Tondreau, T. *et al.* (2005) *Stem Cells* 23: 1105–1012.

Stem cell differentiation tracking by gene expression profiling



PIQOR™ Stem Cell Microarray (human: 942 genes/ mouse: 916 genes)

Relevant marker genes for stem cells

The PIQOR™ Stem Cell Microarray comprises relevant marker genes for human and murine stem cells and key genes involved in their differentiation. It is available as a convenient microarray kit* or within the scope of the Microarray Service.

Thorough selection of genes

Genes have been assembled in cooperation with experts in the field following extensive literature screening, gene expression profiling data analyses, and bioinformatics-based homology screening.

Quality control and evaluation of stem cell differentiation

Gene expression experiments allow for the quality control of different stem cell types, the comparison of differentiation stages, and the optimization of differentiation protocols.

Accurate quantification with high-quality microarrays.

Each PIQOR™ Microarray contains six housekeeping genes and six controls (herring sperm DNA, salt, four artificial control RNAs) for the accurate quantification of the differential expression patterns. Genes are spotted in quadruplicates. The a-Hyb™ Hybridization Station enables fully automated processing of microarrays.

*PIQOR™ Microarray Kits are available in Europe only.

Microarray and Bioinformatics Services

Save time and resources

No need to establish microarray technologies or optimize protocols: simply send cell, tissue, or blood samples and receive reliable results and detailed documentation in return.

Gene expression profiling from 1–100,000 cells

Cells sorted with MACS® Technology, cells sorted by flow cytometry, laser capture microdissected cells, primary cells, and tissue biopsies can be analyzed using the SuperAmp Service.

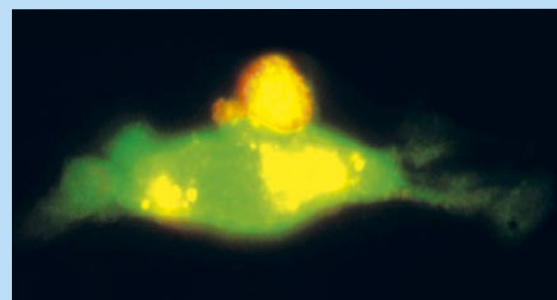
Ready-to-publish data

Bioinformatics Services allow the in-depth analysis of expression data. Experienced bioinformatics specialists perform the biological interpretation based on extensive cluster and pathway analyses.

mRNA isolation and cDNA synthesis

MACS® Technology enables the ultra-fast magnetic isolation of high-quality mRNA and in-column cDNA synthesis – from just a few cells up to 10⁷ cells. The one-step procedure gives pure, first-strand cDNA – for PCR or microarray analysis.

MACS® stem cell products	Size	Order no.
Cell separation products		
CD133 MicroBead Kit, human	2×10 ⁹ total cells	# 130-050-801
CD34 MicroBead Kit, human	2×10 ⁹ total cells	# 130-046-702
	1×10 ¹⁰ total cells	# 130-046-703
CD34 MultiSort Kit, human	2×10 ⁹ total cells	# 130-056-701
CD117 MicroBead Kit, human	2×10 ⁹ total cells	# 130-091-332
MSC Research Tool Box – CD71 (LNGFR), human	1×10 ⁹ total cells	# 130-092-291
CD271 (LNGFR) MicroBead Kit, human	1×10 ⁹ total cells	# 130-092-283
CD105 MicroBeads, human	1×10 ⁹ total cells	# 130-051-201
Anti-Fibroblast MicroBeads, human	1×10 ⁹ total cells	# 130-050-601
Lineage Cell Depletion Kit, human	1×10 ⁹ total cells	# 130-092-211
Fluorochrome-conjugated antibodies		
CD133/1 (AC133)-PE, human	100×10 ⁷ cells	# 130-080-801
CD133/1 (AC133)-APC, human	100×10 ⁷ cells	# 130-090-826
CD133/1 (AC133)-Biotin, human	100×10 ⁷ cells	# 130-090-664
CD133/1 (AC133) pure, human	50 µg in 1 mL	# 130-090-422
CD133/1 (W3B6C1) pure, human – for Western blot	100 µg in 1 mL	# 130-092-395
CD133/2 (293C3)-PE, human	100×10 ⁷ cells	# 130-090-853
CD133/2 (293C3)-APC, human	100×10 ⁷ cells	# 130-090-854
CD34-FITC, human	100×10 ⁷ cells	# 130-081-001
CD34-PE, human	100×10 ⁷ cells	# 130-081-002
CD34-APC, human	100×10 ⁷ cells	# 130-090-954
CD117 (A3C6E2)-PE, human	100×10 ⁷ cells	# 130-091-734
CD117 (A3C6E2)-APC, human	100×10 ⁷ cells	# 130-091-733
CD271 (LNGFR)-PE, human	100×10 ⁷ cells	# 130-091-885
CD271 (LNGFR)-APC, human	100×10 ⁷ cells	# 130-091-883
Stem cell media		
NH CFU-F Medium	24×5 mL	#130-091-676
NH Expansion Medium	500 mL	#130-091-680
NH AdipoDiff Medium	100 mL	#130-091-677
NH ChondroDiff Medium	100 mL	#130-091-679



Microarray products and services	No. of genes/ microarray	Order no.
Microarray Service		
SuperAmp™ Service includes SuperAmp Preparation Kit for sample lysis and storage. Minimum order quantity 4x SuperAmp Service		# 160-000-936
Service Stem Cell Microarray Plus Amplification, human	942	# 160-000-765
Service Stem Cell Microarray Plus Amplification, mouse	916	# 160-000-766
Microarray Service Plus Amplification requires at least 1 µg total RNA/sample (approx. 5×10 ⁴ cells or 1 mg tissue). Microarray Service includes RNA preparation, optional amplification, sample labeling, hybridization with PIQOR™ Microarray, Scanning, primary data analysis and optional Bioinformatics Services.		
PIQOR™ Microarray Kit		
PIQOR™ Stem Cell Microarray Kit, human, antisense, 4 microarrays	942	# 130-092-033
PIQOR™ Stem Cell Microarray Kit, human, antisense, 8 microarrays	942	# 130-092-034
PIQOR™ Stem Cell Microarray Kit, mouse, antisense, 4 microarrays	916	# 130-092-037
PIQOR™ Stem Cell Microarray Kit, mouse, antisense, 8 microarrays	916	# 130-092-036

PIQOR™ Microarray Kits are available in Europe only.

Unless otherwise specifically indicated, all Miltenyi Biotec products and services are for research use only and not for diagnostic or therapeutic use.

MACS is a registered trademark of Miltenyi Biotec GmbH. PIQOR and SuperAmp are trademarks of Miltenyi Biotec GmbH.

Miltenyi Biotec

Miltenyi Biotec GmbH
Friedrich-Ebert-Straße 68
51429 Bergisch Gladbach
Germany
Phone +49 2204 8306-0
Fax +49 2204 85197
macs@miltenyibiotec.de

www.miltenyibiotec.com

Miltenyi Biotec Inc.
12740 Earhart Avenue
Auburn CA 95602, USA
Phone 800 FOR MACS,
+1 530 888 8871
Fax +1 530 888 8925
macs@miltenyibiotec.com

**Miltenyi Biotec Pty. Ltd.
(Australia)**
Phone +61 2 8877 7400
macs@miltenyibiotec.com.au

Miltenyi Biotec B. V. (Benelux)
macs@miltenyibiotec.nl
Customer service, Netherlands
Phone 0800 4020120
Customer service, Belgium
Phone 0800 94016

Customer service, Luxembourg
Phone 800 24971

Miltenyi Biotec Shanghai Office
Phone +86 21 6235 1005
miltenyibiotec@china.com

Miltenyi Biotec (France)
Phone +33 1 56 98 16 16
macs@miltenyibiotec.fr

Miltenyi Biotec S.r.l. (Italy)
Phone +39 051 646 0411
macs@miltenyibiotec.it

Miltenyi Biotec K.K. (Japan)
Phone +81 3 56 46 8910
macs@miltenyibiotec.jp

**Miltenyi Biotec Asia Pacific
Pte. Ltd. (Singapore)**
Phone +65 6238 8183
macs@miltenyibiotec.com.sg

Miltenyi Biotec S.L. (Spain)
Phone +34 91 512 12 90
macs@miltenyibiotec.es

Miltenyi Biotec Ltd. (UK)
Phone +44 1483 799 800
macs@miltenyibiotec.co.uk

