Research Studies

One component of bone marrow stem cells is called CD34+. StemEnhance™ helps facilitate its release into the bloodstream. There are numerous research studies that support **increased CD34+ being very beneficial to health.**

Here are a few:

Neurological and functional recovery in human stroke is associated with peripheral blood CD34+ cell mobilization.

This study demonstrates that following a stroke the body releases CD34+ stem cells, and the more stem cells, the greater the recovery.

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Dunac A, Frelin C, Popolo-Blondeau M, Chatel M, Mahagne MH, Philip PJ.

BACKGROUND: A spontaneous mobilization of Peripheral Blood-Mononuclear CD34+ Cells (PB-MNC-CD34+) has recently been reported in human myocardial infarction and found to be related to improved heart function and survival. However, nothing is known regarding a possible relation between PB-MNC-CD34+ mobilization and neurological recovery in human acute cerebral ischemia.
METHODS AND RESULTS: PB-MNC-CD34+ were determined daily after an acute cerebral ischemic attack for 14 days in 25 patients with acute ischemic stroke and compared with controls. Results indicated that stroke was followed by large and bursting mobilizations of PB-MNC-CD34+. The amplitude of the mobilizations was similar to those observed in Granulocyte Colony Stimulating Factor (G-CSF) conditioned aplastic patients following myeloablative therapy before leukapheresis and autologous bone graft. The extent of PB-MNC-CD34+ mobilization in each patient was directly related to neurological and functional recoveries as assessed by NIH Stroke Scale, and modified Rankin Scale respectively.
CONCLUSIONS: The mobilization of PB-MNC-CD34+ cells might be predictive of neurological and functional recovery.

J Neurol. 2007 Mar;254(3):327-32. Epub 2007

Stem cell mobilisation for myocardial repair

Expert Opin Biol Ther. 2008 Nov;8(11):1675-90 Brunner S, Engelmann MG, Franz WM.
(Research articles available on request)

This study is AMAZING! It demonstrates that the more circulating bone marrow stem cells a person has, the greater the life expectancy and that increased bone marrow stem cells decrease the cardiovascular risk.

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BACKGROUND: The idea that autologous bone marrow derived stem cells (BMCs) can transdifferentiate into cardiomyocytes or vascular cells has been challenged in several scientific reports.
OBJECTIVE/METHODS: This review summarises conditions for stem cell mobilisation, their use for therapeutic approaches to prevent ischaemic cardiomyopathy after acute myocardial infarction and current clinical trials. Mechanisms for mobilisation and homing of BMCs are discussed.
RESULTS/CONCLUSIONS: The improvement in cardiac function after migration of autologous BMCs to the heart can be explained by their paracrine effects, inducing angiogenesis and preventing ischaemic myocardium from apoptosis. These effects may explain why the number of circulating BMCs is directly correlated with cardiovascular risk and life expectancy. Exercise and hormones are physiological stimuli for the mobilisation of BMCs, whereas cardiovascular risk factors severely reduce their number and functions. Current cardiovascular medications increase the amounts of autologous BMCs.

1: J Hum Hypertens. 2008 Mar;22(3):183-90. Epub 2007 Nov 8.

Administrations of peripheral blood CD34-positive cells contribute to medial collateral ligament healing via vasculogenesis.

This study demonstrates that CD34+ greatly improves the ligament healing process. This was a knee ligament and it's thought CD34+ will help in the healing of virtually all ligaments and soft tissues.

Tei K, Matsumoto T, Mifune Y, Ishida K, Sasaki K, Shoji T, Kubo S, Kawamoto A, Asahara T, Kurosaka M, Kuroda R. (Research articles available on request)
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Neoangiogenesis is a key process in the initial phase of ligament healing. Adult human circulating CD34+ cells, an endothelial/hematopoietic progenitor-enriched cell population, have been reported to contribute to neoangiogenesis; however, the therapeutic potential of CD34+ cells for ligament healing is still unclear. Therefore, we performed a series of experiments to test our hypothesis that ligament healing is supported by CD34+ cells via vasculogenesis. Granulocyte colony-stimulating factor-mobilized peripheral blood (GM-PB) CD34+ cells with atelocollagen (CD34+ group), GM-PB mononuclear cells (MNCs) with atelocollagen (MNC group), or atelocollagen alone (control group) was locally transplanted after the creation of medial collateral ligament injury in immunodeficient rats. Reverse transcriptase-polymerase chain reaction (RT-PCR) and immunohistochemical staining at the injury site demonstrated that molecular and histological expression of human-specific markers for endothelial cells was higher in the CD34+ group compared with the other groups at week 1. Endogenous effect, assessed by capillary density and mRNA expression of vascular endothelial growth factor, was significantly higher in CD34+ cell group than the other groups. In addition to the observation that, as assessed by real-time RT-PCR, gene expression of ligament-specific marker was significantly higher in the CD34+ group than in the other groups, ligament healing assessed by macroscopic, histological, and biomechanical examination was significantly enhanced by CD34+ cell transplantation compared with the other groups. Our data strongly suggest that local transplantation of circulating human CD34+ cells may augment the ligament healing process by promoting a favourable

environment through neo-vascularisation.

1: Am J Pathol. 2006 Oct;169(4):1440-57.

Dose-dependent contribution of CD34-positive cell transplantation to concurrent vasculogenesis and cardiomyogenesis for functional regenerative recovery after myocardial infarction.

This study demonstrates that CD34+ helps in the healing of the heart following a heart attack (myocardial infarction), and the more CD34+ the better.

Iwasaki H, Kawamoto A, Ishikawa M, Oyamada A, Nakamori S, Nishimura H, Sadamoto K, Horii M, Matsumoto T, Murasawa S, Shibata T, Suehiro S, Asahara T.
(Research articles available by request)
Stem Cell Translational Research, Kobe Institute of Biomedical Research and Innovation/RIKEN Center for Developmental Biology, Kobe, Japan.

BACKGROUND: Multilineage developmental capacity of the CD34+ cells, especially into cardiomyocytes and smooth muscle cells (SMCs), is still controversial. In the present study we performed a series of experiments to prove our hypothesis that vasculogenesis and cardiomyogenesis after myocardial infarction (MI) may be dose-dependently enhanced after CD34+ cell transplantation.
METHODS AND RESULTS: Peripheral blood CD34+ cells were isolated from total mononuclear cells of patients with limb ischemia by apheresis after 5-day administration of granulocyte colony-stimulating factor. PBS and 1x10(3) (low), 1x10(5) (mid), or 5x10(5) (high) CD34+ cells were intramyocardially transplanted after ligation of the left anterior descending coronary artery of nude rats. Functional assessments with the use of echocardiography and a microtip conductance catheter at day 28 revealed dose-dependent preservation of left ventricular function by CD34+ cell transplantation. Necropsy examination disclosed dose-dependent augmentation of capillary density and dose-dependent inhibition of left ventricular fibrosis. Immunohistochemistry for human-specific brain natriuretic peptide demonstrated that human cardiomyocytes were dose-dependently observed in ischemic myocardium at day 28 (high, 2480+/-149; mid, 1860+/-141; low, 423+/-9; PBS, 0+/-0/mm2; $P<0.05$ for high versus mid and mid versus low). Immunostaining for smooth muscle actin and human leukocyte antigen or Ulex europaeus lectin type 1 also revealed dose-dependent vasculogenesis by endothelial cell and SMC development after CD34+ cell transplantation. Reverse transcriptase-polymerase chain reaction indicated that human-specific gene expression of cardiomyocyte (brain natriuretic peptide, cardiac troponin-I, myosin heavy chain, and Nkx 2.5), SMC (smooth muscle actin and sm22alpha), and endothelial cell (CD31 and KDR) markers were dose-dependently augmented in MI tissue.
CONCLUSIONS: Human CD34+ cell transplantation may have significant and dose-dependent potential for vasculogenesis and cardiomyogenesis with functional recovery from MI.

1: Atherosclerosis. 2006 Aug;187(2):423-32. Epub 2005 Nov 9.

CD34+ blood cells accelerate vascularisation and healing of diabetic mouse skin wounds.

This study demonstrates that CD34+ helps in the healing of skin wounds in diabetic mice

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(Research articles available by request)
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Diabetes is characterized by poor circulation and impaired angiogenesis, which appear to contribute to the frequent skin lesions and poor wound healing common in diabetic patients. Therapies to improve circulation commonly improve wound healing in diabetic patients. Administration of circulating CD34+ cells, cells that can function as endothelial cell progenitors, accelerates blood flow restoration to ischemic limbs of diabetic mice. We have investigated the potential of these cells to accelerate revascularization and healing in full-thickness skin wounds of hypoinsulinemic (streptozotocin-treated) diabetic mice. Wounds were injected with human CD34+ or CD34- peripheral blood mononuclear cells or no cells, and analyzed for vascularity and healing at various times thereafter. Treatment with CD34+ enriched cells decreased wound size by 4 days after treatment, accelerated epidermal healing, and rapidly and dramatically accelerated revascularization of the wounds compared to controls. Initially increased vascularization was mediated principally by an increase in vessel diameter, but later, both an increase in vascular size and number were observed. These findings indicate that blood-derived progenitors may have therapeutic potential in the treatment of skin lesions in the setting of diabetes, and give insights into how bone marrow cells exert their effects on neovascularization.

 PMID: 12891006 [PubMed - indexed for MEDLINE]

Low circulating CD34(+) cell count is associated with poor prognosis in chronic hemodialysis patients.

This study demonstrates that a reduced number of circulating CD34(+) cells is significantly associated with vascular risks and all-cause mortality in patients on chronic hemodialysis.

Source: 1Department of Nephrology, Nagoya University Graduate School of Medicine, Nagoya, Japan.

Maruyama S, Taguchi A, Iwashima S, Ozaki T, Yasuda K, Kikuchi-Taura A, Soma T, Ishii H, Murohara T, Takahashi H, Kasuga H, Kumada Y, Toriyama T, Ito Y, Kawahara H, Yuzawa Y, Matsuo S. (Research articles available upon request)

Circulating CD34-positive (CD34(+)) cells, a population that includes endothelial progenitor cells, are believed to contribute to vascular homeostasis. Here we determine the prognostic value of CD34(+) cell measurements in 216 chronic hemodialysis patients. A total of 43 cardiovascular events and 13 deaths occurred over an average 23 months follow-up in this cohort. A cut-off number for circulating CD34(+) cells was determined by receiver operating characteristic curve analysis to maximize the power of the CD34(+) cell count in predicting future cardiovascular events. Based on this, 93 patients were categorized as having low and 123 patients as having high numbers of CD34(+) cells, determined by flow cytometry at the time of enrolment. Both cumulative cardiovascular event-free survival and all-cause survival were significantly less in the group of patients with low numbers of CD34(+) cells. By multivariate analyses, a low level of circulating CD34(+) cells was an independent and significant predictor for both cardiovascular events and all-cause mortality. Our study shows that a reduced number of circulating CD34(+) cells is significantly associated with vascular risks and all-cause mortality in patients on chronic hemodialysis. These cells may be a useful

biomarker. Kidney
International advance online publication, 8 October 2008; doi:10.1038/ki.2008.495.

Circulating endothelial progenitor cells and cardiovascular outcomes.

This study demonstrates that low CD34+ circulating in the blood can predict cardiovascular events and DEATH from cardiovascular disease

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(Research articles available upon request)

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BACKGROUND: Endothelial progenitor cells derived from bone marrow are believed to support the integrity of the vascular endothelium. The number and function of endothelial progenitor cells correlate inversely with cardiovascular risk factors, but the prognostic value associated with circulating endothelial progenitor cells has not been defined.
METHODS: The number of endothelial progenitor cells positive for CD34 and kinase insert domain receptor (KDR) was determined with the use of flow cytometry in 519 patients with coronary artery disease as confirmed on angiography. After 12 months, we evaluated the association between baseline levels of endothelial progenitor cells and death from cardiovascular causes, the occurrence of a first major cardiovascular event (myocardial infarction, hospitalization, revascularization, or death from cardiovascular causes), revascularization, hospitalization, and death from all causes.
RESULTS: A total of 43 participants died, 23 from cardiovascular causes. A first major cardiovascular event occurred in 214 patients. The cumulative event-free survival rate increased stepwise across three increasing baseline levels of endothelial progenitor cells in an analysis of death from cardiovascular causes, a first major cardiovascular event, revascularization, and hospitalization. After adjustment for age, sex, vascular risk factors, and other relevant variables, increased levels of endothelial progenitor cells were associated with a reduced risk of death from cardiovascular causes (hazard ratio, 0.31; 95 percent confidence interval, 0.16 to 0.63; P=0.001), a first major cardiovascular event (hazard ratio, 0.74; 95 percent confidence interval, 0.62 to 0.89; P=0.002), revascularization (hazard ratio, 0.77; 95 percent confidence interval, 0.62 to 0.95; P=0.02), and hospitalization (hazard ratio, 0.76; 95 percent confidence interval, 0.63 to 0.94; P=0.01). Endothelial progenitor-cell levels were not predictive of myocardial infarction or of death from all causes.
CONCLUSIONS: The level of circulating CD34+KDR+ endothelial progenitor cells predicts the occurrence of cardiovascular events and death from cardiovascular causes and may help to identify patients at increased cardiovascular risk.

PMID: 16148285 [PubMed - indexed for MEDLINE] Circulation. 2005 Jan 18;111(2):204-11. Epub 2005 Jan 10.

Depletion of endothelial progenitor cells in the peripheral blood of patients with rheumatoid arthritis.

This study demonstrates that low EPCs, including CD34+ is found in patients with rheumatoid arthritis.

Grisar J, Aletaha D, Steiner CW, Kapral T, Steiner S, Seidinger D, Weigel G, Schwarzinger I, Wolozcszuk W, Steiner G, Smolen JS.
(Research articles available on request)

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BACKGROUND: Rheumatoid arthritis (RA) is characterized by increased cardiovascular morbidity and mortality that cannot be explained solely by traditional cardiovascular risk factors. Cardiovascular morbidity is related to disease activity and can be normalized by effective therapy. Because the quantity of endothelial progenitor cells (EPCs) in the peripheral blood is correlated inversely with cardiovascular risk, we studied whether such abnormalities could also be observed in patients with RA.
METHODS AND RESULTS: EPCs were determined in 52 RA patients and in 16 healthy referents (HRs) by fluorescence-activated cell-sorting (FACS) analysis. Patients were divided into groups characterized by active disease (n=36) and low disease activity (n=16). Cells that were positive by flow cytometry for CD34/KDR/AC133 within the lymphocyte population were characterized as EPCs. Furthermore, in subgroups of patients, circulating EPCs were also quantified by a colony-forming unit (CFU) and a circulating angiogenic cell (CAC) assay. EPCs were significantly decreased in RA patients suffering from active disease compared with those from HRs, as measured by FACS analysis (0.026+/-0.002% versus 0.045+/-0.008%, respectively, P<0.05), CFU assay (mean of 5+/-2 versus 18+/-5 CFU/well in HRs, P<0.05), and CAC assay (mean of 7+/-2 versus 52+/-16 positive cells/high-power field, P<0.005). In contrast, the frequency of circulating EPCs from patients with low disease activity was comparable to that of healthy individuals (0.052+/-0.006% by FACS analysis), CFU assay (10+/-5 CFU/well), and CAC assay (mean of 25+/-5 positive cells). Moreover, EPC quantities in peripheral blood were correlated inversely with disease activity as assessed by the disease activity score (r=-0.38, P<0.01).
CONCLUSIONS: Our observations indicate that active RA is associated with a depletion of circulating EPCs. This might be one of several factors contributing to the increased cardiovascular risk in RA.

J Appl Physiol. 2008 Apr;104(4):1006-13. Epub 2008 Jan 24.

A maximal exercise bout increases the number of circulating CD34+/KDR+ endothelial progenitor cells in healthy subjects. Relation with lipid profile.

This study demonstrates that one benefit of exercise is an increase of CD34+, which may explain why exercise improves blood vessel function.

Van Craenenbroeck EM, Vrints CJ, Haine SE, Vermeulen K, Goovaerts I, Van Tendeloo VF, Hoymans VY, Conraads VM.
(Research articles available on request)

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Mobilization of bone marrow-derived endothelial progenitor cells (EPC) might explain exercise-induced improvement of endothelial function. We assessed whether a maximal exercise bout could alter the number of circulating EPC in healthy subjects and whether this effect is related to their cardiovascular risk profile. Additionally, we investigated possible mediators of this effect, namely nitric oxide (NO) bioavailability and vascular endothelial growth factor (VEGF) release. Healthy subjects (group 1, n = 11; group 2, n = 14) performed a symptom-limited cardiopulmonary exercise test on a bicycle ergometer. Numbers of CD34+/kinase insert domain receptor (KDR)+ cells were determined by flow-cytometric analysis, either after magnetic separation of CD34+ cells (group 1) or starting from whole blood (group 2). Serum concentrations of VEGF and NO metabolites were measured by using ELISA. Following exercise, EPC increased by 76% (15.4 +/- 10.7 cells/ml vs. 27.2 +/- 13.7 cells/ml; P = 0.01) in group 1 and by 69% in group 2 (30.9 +/- 14.6 cells/ml vs. 52.5 +/- 42.6 cells/ml; P = 0.03). The increase in EPC correlated positively with LDL and total cholesterol/HDL ratio and negatively with peak oxygen consumption and oxygen consumption at anaerobic threshold. VEGF levels increased with exercise, with a strong trend toward significance (P = 0.055). NO levels remained unchanged. The present study demonstrates that a maximal bout of exercise induces a significant shift in CD34+ cells toward CD34+/KDR+ cells. This response was larger in subjects with a less favorable lipid profile.

Endothelial progenitor cells are reduced in refractory hypertension.

This study demonstrates that people with high blood pressure have less circulating stem cells, including CD34+, and this decrease may explain their increase in atherosclerosis and cardiovascular events (heart attacks and strokes).

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(Research articles available on request)

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Circulating endothelial progenitor cells (EPCs) play a key role in the maintenance of endothelial homoeostasis and promote vascular repair. They may also be of predictive value for cardiovascular events. Reduced EPC number and functional activity have been associated with several cardiovascular risk factors, but their relationship with hypertension remains unclear. The objective of this study was to investigate if number and function of circulating EPCs are reduced in patients with refractory hypertension (RHT). Circulating EPCs (CD34+ CD133+/CD45+) were isolated from peripheral blood by flow cytometry in 39 RHT and 30 normotensive controls. EPC number was also determined in vitro after 7 days in culture. After age adjustment, EPC concentration was significantly reduced in RHT as compared with controls (mean (95% CI), 33.8 (18.1-49.6) vs 69.1 (50.7-87.5) EPCs per 10(5) peripheral mononuclear cells (MNCs), respectively; P=0.014). After in vitro culture, EPCs were also reduced in patients as compared with controls (mean (95% CI), 142.3 (49.5-235.0) vs 611.0 (480.2-741.8) EPCs per field, respectively, P<0.001). In multiple linear regression analysis, circulating EPCs were significantly reduced by 56.3% in RHT as compared with control (P=0.006), independently of all other known risk factors. Moreover, RHT had a high

independent predictive value for lower EPC proliferation. The number of EPCs per field was reduced by 76.7% in RHT with respect to controls (P<0.001). In summary, the number of circulating EPCs after culture is reduced in patients with RHT, which may be related to the increased rate of endothelial dysfunction, atherosclerotic disease and cardiovascular events observed in this population.

Therapeutic potential of vasculogenesis and osteogenesis promoted by peripheral blood CD34-positive cells for functional bone healing.

This study demonstrates that increased CD34+ helps in the healing of bone, even in non-union fractures.

Matsumoto T, Kawamoto A, Kuroda R, Ishikawa M, Mifune Y, Iwasaki H, Miwa M, Horii M, Hayashi S, Oyamada A, Nishimura H, Murasawa S, Doita M, Kurosaka M, Asahara T.
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Failures in fracture healing are mainly caused by a lack of vascularization. Adult human circulating CD34+ cells, an endothelial/hematopoietic progenitor-enriched cell population, have been reported to differentiate into osteoblasts in vitro; however, the therapeutic potential of CD34+ cells for fracture healing is still unclear. Therefore, we performed a series of experiments to test our hypothesis that functional fracture healing is supported by vasculogenesis and osteogenesis via regenerative plasticity of CD34+ cells. Peripheral blood CD34+ cells, isolated from total mononuclear cells of adult human volunteers, showed gene expression of osteocalcin in 4 of 20 freshly isolated cells by single cell reverse transcriptase-polymerase chain reaction analysis. Phosphate-buffered saline, mononuclear cells, or CD34+ cells were intravenously transplanted after producing nonhealing femoral fractures in nude rats. Reverse transcriptase-polymerase chain reaction and immunohistochemical staining at the peri-fracture site demonstrated molecular and histological expression of human-specific markers for endothelial cells and osteoblasts at week 2. Functional bone healing assessed by biomechanical as well as radiological and histological examinations was significantly enhanced by CD34+ cell transplantation compared with the other groups. Our data suggest circulating human CD34+ cells have therapeutic potential to promote an environment conducive to neovascularization and osteogenesis in damaged skeletal tissue, allowing the complete healing of fractures.
1: Circulation. 2006 Mar 14;113(10):1311-25. Comment in: Circulation. 2006 Mar 14;113(10):1275-7.

Circulating endothelial progenitor cells from healthy smokers exhibit impaired functional activities.

This study demonstrates that smoking DECREASES circulating EPCs (of which CD34+ is a part) which may explain why smokers have a higher risk of circulatory problems. Increased EPCs (like CD34+)may prevent that from occurring.

Michaud SE, Dussault S, Haddad P, Groleau J, Rivard A.
(Research articles available by request)
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OBJECTIVE: Endothelial dysfunction is one of the earliest pathological effects of cigarette smoking. It has recently been suggested that endothelial progenitor cells (EPCs) could contribute to ongoing endothelial maintenance and repair. Accordingly, we tested the hypothesis that cigarette smoking is associated with EPC dysfunction.
METHODS AND RESULTS: EPCs were isolated from the peripheral venous blood of 15 healthy smokers and 11 age-matched nonsmokers. The number of EPCs was significantly reduced in smokers versus control subjects (51.6+/-1.9 versus 120.3+/-10.0 per power field, p<0.001). Moreover, the functional activities of EPCs isolated from smokers were severely compromised. First, the proliferative and migratory response of EPCs isolated from smokers were reduced by 75% and 19%, respectively (p<0.05). Second, EPCs from smokers showed an important decreased adherence to HUVECs that had been previously activated with tumor necrosis factor-alpha (TNF-alpha) (p<0.01). Finally, the participation of EPCs to tube formation in a matrigel assay was reduced by 38% in smokers versus control subjects (p<0.001). We found that EPCs from smokers had a significant reduction in the expression of the endothelial cell-specific markers (VE-cadherin, KDR, and vWF). Moreover, ROS formation was significantly increased in EPCs from smokers, whereas the serum antioxidant and nitrite levels of smokers were reduced and correlated with impaired EPC number and functional activity.
CONCLUSIONS: Cigarette smoking is associated with a reduced number of EPCs together with an important impairment of EPC differentiation and functional activities. Our results suggest that EPC dysfunction could contribute to impair blood vessel healing and growth in smokers.
1: J Vasc Res. 2003 Jul-Aug;40(4):368-77. Epub 2003 Jul 29.

The Following Research Articles show the relationship of bone marrow stem cells to brain and nervous system repair.

Proc Natl Acad Sci U S A. 1997 Apr 15;94(8):4080-5.

Hematopoietic cells differentiate into both microglia and macroglia in the brains of adult mice.

Brain cells can come from bone marrow stem cells

Eglitis MA, Mezey E.
(Research articles available by request)
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Glial cells are thought to derive embryologically from either myeloid cells of the hematopoietic system (microglia) or neuroepithelial progenitor cells (astroglia and oligodendrocytes). However, it is unclear whether the glia in adult brains free of disease or injury originate solely from cells present in the brain since the fetal stage of development, or if there is further input into such adult brains from cells originating outside the central

nervous system. To test the ability of hematopoietic cells to contribute to the central nervous system, we have transplanted adult female mice with donor bone marrow cells genetically marked either with a retroviral tag or by using male donor cells. Using in situ hybridization histochemistry, a continuing influx of hematopoietic cells into the brain was detected. Marrow-derived cells were already detected in the brains of mice 3 days after transplant, and their numbers increased over the next several weeks, exceeding 14,000 cells per brain in several animals. Marrow-derived cells were widely distributed throughout the brain, including the cortex, hippocampus, thalamus, brain stem, and cerebellum. When in situ hybridization histochemistry was combined with immunohistochemical staining using lineage-specific markers, some bone marrow-derived cells were positive for the microglial antigenic marker F4/80. Other marrow-derived cells surprisingly expressed the astroglial marker glial fibrillary acidic protein. These results indicate that some microglia and astroglia arise from a precursor that is a normal constituent of adult bone marrow.

PMID: 9108108 [PubMed - indexed for MEDLINE]

Eur J Pharmacol. 2000 Sep 29;405(1-3):297-302.
Bone marrow: a possible alternative source of cells in the adult nervous system.

Bone marrow stem cells can become brain and liver and muscle.

Mezey E, Chandross KJ.(Research articles available by request)
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There is increasing evidence that stem cell populations can undergo a transition between mesodermal and neural ectodermal cell fates. Bone marrow-derived cells have been shown to be extremely versatile: they can become brain and liver cells and muscle, while other mesodermal derived cells have been shown to migrate into the brain and differentiate into neurons. Moreover, under the appropriate conditions, neural stem cells can differentiate into hematopoietic and muscle cell fates. It is now well established that newly differentiated cell types are continuously generated from immature stem cells throughout development and in adult mammals, including humans. This review addresses the contribution that bone marrow-derived stem cells may play during neurogenesis. We transplanted male bone marrow into female recipients to track and characterize the Y chromosome containing cells in the CNS (central nervous system) of mice.

Publication Types: Review PMID: 11033336 [PubMed - indexed for MEDLINE]

Comment in: Science. 2000 Dec 1;290(5497):1672-4.
Turning blood into brain: cells bearing neuronal antigens generated in vivo from bone marrow.

Bone marrow stem cells become brain cells

Mezey E, Chandross KJ, Harta G, Maki RA, McKercher SR.
(Research articles available by request)
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Bone marrow stem cells give rise to a variety of hematopoietic lineages and repopulate the

blood throughout adult life. We show that, in a strain of mice incapable of developing cells of the myeloid and lymphoid lineages, transplanted adult bone marrow cells migrated into the brain and differentiated into cells that expressed neuron-specific antigens. These findings raise the possibility that bone marrow-derived cells may provide an alternative source of neurons in patients with neurodegenerative diseases or central nervous system injury.

Publication Types: Research Support, U.S. Gov't, P.H.S. PMID: 11099419 [PubMed - indexed for MEDLINE]

J Neurol Sci. 2005 Jun 15;233(1-2):121-3. Epub 2005 Apr 21.
Transplanted human bone marrow cells generate new brain cells.

Bone marrow cells generate brain cells

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Multiple studies have reported that adult cells of bone marrow origin can differentiate into muscle, skin, liver, lung, epithelial cells, and neurons. To determine whether such cells might produce neurons and other cells in the human brain, we examined paraffin sections from female patients who had received bone marrow transplants from male donors. Y-chromosomes were labeled using autoradiography and fluorescent in situ hybridization. Neurons and astrocytes were identified histologically and immunohistochemically in neocortex, hippocampus, striatum, and cerebellum. However, most labeled cells in both gray and white matter appeared to be glia. Others have suggested that such Y-labeling represents fusion between host and donor cells, rather than true transdifferentiation. The possibilities of fusion and microchimerism were therefore examined using buccal epithelial cells as a model system. The female patients in this study had received either bone marrow or stem cell (CD34+ enriched) transplants from their brothers. Double labeling for X- and Y-chromosomes showed that Y-labeled buccal cells could not be explained by fusion. Genotyping studies of one patient, her brother, and her son ruled out the possibility of microchimerism. Whether, and under what circumstances, some form of bone marrow transplantation might provide adequate number of cells capable of replacing lost brain cells or enhancing their function will require additional studies.

Publication Types: Review PMID: 15949500 [PubMed - indexed for MEDLINE]

For more information on the natural release and stimulation of your own stem cells:

http://impact.stemtechhealth.co.za