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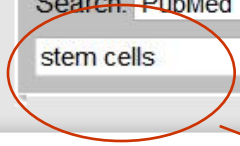
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[Caenorhabditis elegans as a model for stem cell biology.](#)

2. Joshi PM, Riddle MR, Djabrayan NJ, Rothman JH.  
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[Bifunctional Eu\(3+\)-doped Gd\(2\)O\(3\) nanoparticles as a luminescent and T\(1\) contrast agent for stem cell labeling.](#)

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Transgenic Res. 2010 Apr 24. [Epub ahead of print]  
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- [Interaction of retinoic acid and scl controls primitive blood development.](#)  
21. de Jong JL, Davidson AJ, Wang Y, Palis J, Opara P, Pugach E, Daley GQ, Zon LI.  
Blood. 2010 Apr 21. [Epub ahead of print]  
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John Hughes Bennett Laboratory, Edinburgh Cancer Centre, University of Edinburgh, Western General Hospital, Crewe Road, Edinburgh, EH4 2XU, UK.

**Abstract**  
The stem cell factor (SCF)-KIT signal transduction pathway plays a role in the proliferation, differentiation and survival of a range of stem and progenitor cell types but little is known about its function in embryonic stem (ES) cells. We generated ES cells carrying a null allele of Kit as well as a knock-in allele that encodes an SCF-independent hybrid KIT receptor that can be activated by the FKBP binding drug, AP20187. KIT null ES cells die when induced to differentiate upon withdrawal of leukaemia inhibitory factor in monolayer culture. This phenotype is recapitulated in wild-type ES cells treated with a KIT-neutralising antibody and reversed in mutant cells by activation of the hybrid KIT receptor. Differentiating KIT null ES cells exhibit elevated levels of DNA laddering and reduced BCL2 expression, indicative of apoptosis. We conclude that mouse ES cell differentiation in vitro is dependent on the SCF-KIT pathway contrasting with the apparently normal differentiation of KIT null inner cell mass or epiblast cells in vivo. This discrepancy could be explained by the presence of compensatory signals in the embryo or it could lend support to the idea of a phenotypic relationship between ES cells and early germ cells.

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**Abstract**

Human umbilical cord blood (UCB) contains cells able to differentiate into nonhaematopoietic cell lineages similar to primitive embryonic stem cells (ESCs) that can differentiate into pancreatic-like cells. However, little data has been reported regarding the possibility of expanding these cells or their differential gene expression occurring in vitro. We expanded previously frozen UCB cells by treatment with Stem Cell Factor (SCF) and Granulocyte-Macrophage Colony Stimulating Factor (GM-CSF) in the presence of valproic acid (VPA). Gene expression profiles for beta-cell differentiation and pluripotency (embryo stem cell phenotype) were analysed by RT-PCR and immunocytochemistry. There was a dramatic expansion (<150 fold) of hematopoietic progenitors (CD45+/CD133+) that also expressed embryo markers of pluripotency (nanog, klf-4,sox-2,oct3/4 and c-myc), nestin, pancreatic markers as pax-4, ngn-3, pdx-1 and syt-1. UCB cells can be expanded to produce large numbers of cells of hematopoietic lineage that naturally (without retroviral vectors or transposons) express a gene pattern compatible with endocrine pancreatic precursors and markers of pluripotency. Our results confirm that frozen UCB cells can be dramatically expanded along the hematopoietic cell lineage (CD45+/CD133+) which express both markers of pluripotency and a gene pattern compatible with endocrine pancreatic precursors.

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John Hughes Bennett Laboratory, Edinburgh Cancer Centre, University of Edinburgh, Western General Hospital, Crewe Road, Edinburgh

### Abstract

The stem cell factor (SCF)-KIT signal transduction pathway plays a role in the proliferation, differentiation and survival of a range of cell types but little is known about its function in embryonic stem (ES) cells. We generated ES cells carrying a null allele of Kit that encodes an SCF-independent hybrid KIT receptor that can be activated by the FKBP binding drug, AP20187. KIT null ES cells differentiate upon withdrawal of leukaemia inhibitory factor in monolayer culture. This phenotype is recapitulated in wild-type ES cells by neutralising antibody and reversed in mutant cells by activation of the hybrid KIT receptor. Differentiating KIT null ES cells exhibit cell laddering and reduced BCL2 expression, indicative of apoptosis. We conclude that mouse ES cell differentiation in vitro is dependent on the SCF-KIT pathway contrasting with the apparently normal differentiation of KIT null inner cell mass or epiblast cells in vivo. This discrepancy could be due to the presence of compensatory signals in the embryo or it could lend support to the idea of a phenotypic relationship between ES cells and the inner cell mass.

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