FEATURES

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Fetal Cells and Non-Invasive Prenatal Diagnosis

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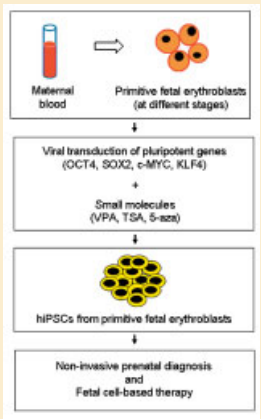
Prenatal diagnosis provides valuable information on the health of the unborn child. Currently, prenatal diagnosis is invasive, posing risks to the fetus and there is no reliable routine method for non-invasive prenatal diagnosis (NPID). The use of fetal cells and their genetic material in the maternal blood circulation is an attractive approach for future NIPD. However, the scarcity of these fetal cells in the maternal circulation and their difficulties in *ex vivo* expansion are major limitations to NIPD. Of the various fetal cell types, the primitive fetal erythroblast (pFE) appears to be a good candidate for NPID as its presence is indicative of the current ongoing pregnancy. In this review Huang et al highlight the current status of NIPD and suggest some novel insights in generating adequate numbers of pFEs for future NIPD. These studies include (1) understanding the pFE enucleation process, (2) developing a micromanipulation and microdroplet culture method to enrich pFEs from maternal blood, (3) evaluating the use of mitogens for *ex vivo* expansion of pFEs and (4) evaluating the reprogramming of pFEs using gene delivery protocols with/without epigenetic factors and small molecules to increase pFE proliferation rates for NIPD.



The Hematopoietic Stem Cell Niche

Sofie Singbrant, Maria Askmyr, Louise E. Purton, and Carl R. Walkley

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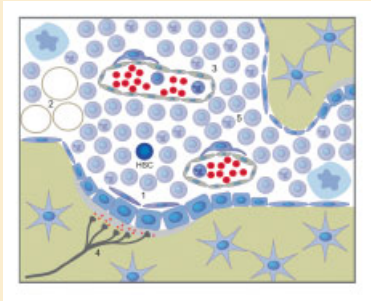
Understanding the *in vivo* regulation of hematopoietic stem cells (HSC) is critical to identifying factors involved in the control of HSC self-renewal and differentiation. The increasing sophistication of *in vivo* imaging techniques and genetic manipulation in the mouse has rapidly improved our knowledge of the HSC microenvironment and the contribution of several niche elements to the regulation of HSCs. But is it possible to manipulate a given cell lineage *in vivo* without impacting on those that remain? In this issue Singbrant et al. discuss the difficulty of defining the *in vivo* niche. HSCs are rare cells and to date it has not been possible to prove that an HSC in proximity of candidate niche elements is functionally regulated and contributing to hematopoiesis. The authors raise the possibility that when interpreting murine models investigating the niche there is a tight nexus coordinating homeostasis between the separate elements of the bone marrow microenvironment *in vivo*. It remains to be determined how modulating one niche component will in turn impact on the function and HSC supportive capacity of the other cell types within the niche.



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Directed Neural Differentiation

Lin-Feng Li, Chun-Yu Bai, Xue-Lian Gong, Wei-Jun Guan, and Yue-Hui Ma

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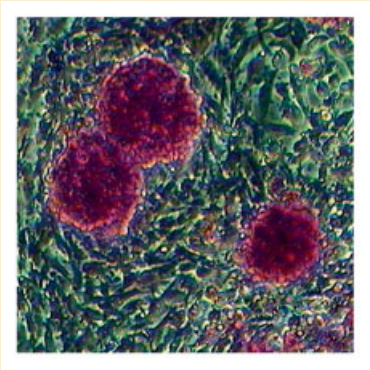

It is well known that one of the most important challenges in stem cell research is to understand and control cell-differentiation processes. To date, it has been proven that ES cells are capable of directed differentiation into neural precursors and progenies in mammals. However, similar studies on EG cells in mammals and other species are few, and directed differentiation of avian EG cells has not been fully exploited. The avian embryo represents an important model system in developmental and cell biology because of its ease of manipulation and its similarity to mammalian development. Therefore, the avian model is ideal for diverse types of neurological research. To date, prolonged propagation of undifferentiated ES cells has historically relied on the mouse embryonic fibroblast feeder layer. In order to eliminate the risk of contamination with heterogenous cells, protein, and unknown pathogenic microorganisms in culture, Li et al used duck embryonic fibroblasts as a feeder layer instead of mouse embryonic fibroblast feeder layers to support in vitro growth of EG cells. This study creates new opportunities to manipulate the duck genome for agricultural and pharmaceutical applications, and provides an in vitro model of early embryonic differentiation.

Cell Based Gene Therapy for Bone Repair

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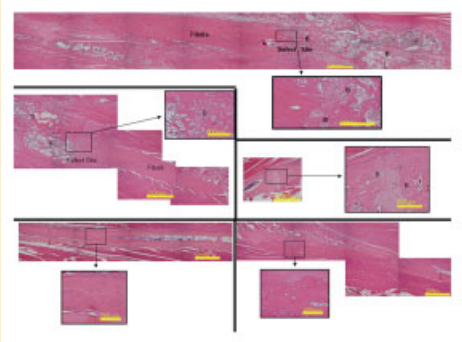

Although bone possesses the capacity to repair and remodel, often this process is ineffective when large amounts of bone must be rapidly regenerated during traumatic injury. Lazard *et al*, have developed a molecular therapy to induce bone formation, in a targeted manner for repair of large bone injuries. The system provides endogenous expression of bone morphogenic protein (BMP2) through *ex vivo* adenovirus transduction of the cells and delivery to the target site, and is able to produce heterotopic bone within two weeks, which spanned the defect introduced into a rat fibula. The model is challenging because even a simple fracture undergoes resorption rather than healing. Upon delivery of the therapy, bone healing progressed with resorption of the additional HO, and rapid fusion with the native skeleton, to restore bone health. This remodeling was completed within 4 weeks, and the bone remained effectively healed throughout the remainder of the study. This study demonstrates the ability of an injectable therapy to rapidly heal a critical size defect in rats. The benefits to this approach are in the ability to induce healing without surgical intervention, thus reducing the chances of potential infection or non-union bone healing, through the rapid nature of the mechanism.