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Commentary

Pluripotent Stem Cells as a Source of Osteoblasts for Bone Tissue Regeneration

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DESCRIPTION

Generally bone is capable of self-repair, for example following a fracture. However, in some cases such as a critical-sized fault or tumor-mediated damage, localized bone regeneration is reduced. In such cases, a source of bone-forming osteoblasts in combination with tissue engineering could offer a regenerative option for bony defects. Osteoblasts have been difficult to harvest in large numbers, because they are found in mineralized tissues. Traditional methods of obtaining primary osteoblasts trust upon cell outgrowth from surgically resected bone fragments, or differentiation from bone marrow Mesenchymal Stem Cells (MSCs).

Pluripotent stem cells differentiate into any tissue and can self-renew therefore signify a theoretically unlimited source of osteoblasts. Pluripotent stem cells contain Embryonic Stem Cells (ESCs) derived from the inner cell mass of the blastocyst, and induced Pluripotent Stem Cells (iPSCs) derived from somatic cell by the outline of four transcription factors. Both ESCs and iPSCs can give rise to osteoblasts in *in vivo* using a skeletal complementation model, and that these osteoblasts can further save a defective hematopoietic bone marrow microenvironment. However, since in vivo transplantation of ESCs and iPSCs transmits the risk of teratoma formation, osteoblasts derived from ESC and iPSC differentiation *in vitro* might be favoured for bone tissue engineering purposes. Numerous recent studies have focused on differentiating both ESCs and iPSCs into osteoblasts. ESCs and iPSCs can be differentiated to the osteoblast lineage by first forming Emryoid Bodies (EBs), in which mesoderm lineage cells differentiate to osteoblast lineage cells under osteogenic factors including ascorbic acid, ß-glycerophosphate, and dexamethasone. ESCs and iPSCs can also be distinguished to osteoblast lineage cells through monolayer culture without first forming suspended EBs.

Osteoblast diversity in culture is often assayed by expression of osteoblast-related genes, formation of bony nodules, and mineralization of the surrounding extracellular matrix. Differentiation of pluripotent stem cells naturally results in heterogeneous cellular populations, and even the existence of only a small fraction of osteoblasts can yield positive results in assays of osteoblast gene expression and mineralization. *In vitro* osteoblast assays do not precisely predict bone formation *in vivo*, and osteoblast gene expression and mineralization are not enough to demonstrate osteogenic maturation. Furthermore, these assays do not allow for quantitation of osteoblast frequency within a combined population, making it difficult to directly compare the efficiency of various differentiation protocols.

The ability to selectively develop for live mature osteoblasts within heterogeneous pluripotent stem cell-derived cell populations would be a vital step to elevating osteoblast differentiation from ESCs and iPSCs, but has been delayed by the lack of cell surface markers that uniquely distinguish osteoblasts.

CONCLUSION

Once osteoprogenitor cells start to differentiate into osteoblasts, they initiate to express a level of genetic markers: they secrete collagen I which is important for future mineralisation of hydroxyapatite. The collagen excreted forms osteoids, the osteoblasts cause calcium salts and phosphorous to precipitate from the blood and bond with the newly formed osteoid to mineralise the bone tissue.

Osteoblasts also produce alkaline phosphatase which is an enzyme that is involved in the mineralisation of bone. It is a primary marker of osteoblast differentiation and its augmented expression is associated with the advanced differentiation of osteoblasts. Osteoblasts have oestrogen receptors that permit them to promote the number of osteoblasts in order to increase collagen production.

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