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Opinion Article

The PDGFRA Reporter Activity Indicates Periosteal Progenitor Cells Essential for Skeletal Development and Trauma Regeneration

Almeida Barros*

Department of Surgery, State University of Campinas, São Paulo, Brazil

DESCRIPTION

PDGFRA, or Platelet-Derived Growth Factor Receptor A, is a receptor found on the surface of a variety of cell types. It is also known as PDGFR, or platelet-derived growth factor receptor, or CD140a, or Cluster of Differentiation 140a. This receptor binds to specific isoforms of Platelet-Derived Growth Factors (PDGFs) and becomes active in stimulating cell signaling pathways that cause responses including cell growth and differentiation. The receptor is required for the growth and maintenance of specific tissues and organs throughout life, notably hematologic tissues. PDGF-AA is mostly produced and secreted by epithelial cells, and it acts on PDGFR-expressing mesoderm-derived cells. Its primary role, in addition to angiogenesis, is to promote mesenchyme expansion. PDGFs play a crucial role in bone formation. Inhibition of PDGFR resulted in skeletal morphogenesis and development problems. PDGFs serve essential roles in wound healing and bone fracture healing in adults, where they act on fibroblasts, Pluripotent, and other cell types, suggesting that they could be used as therapeutic regents for wound healing and bone regeneration. PDGFs have been found to stimulate MSC proliferation at the cell level. While PDGF-BB has been shown to suppress osteoblast formation, the role of PDGF-AA in osteoblast differentiation remains unknown. It's still unknown how PDGFs influence MSC differentiation and proliferation.

The periosteum, or outside coverings of the skeleton, are structured in concentric layers and serve as a reservoir for tissue-specific bone progenitors. This tissue depot's cellular heterogeneity is becoming more widely known. Inducible PDGFR reporter mice were discovered to identify a population of cells within the periosteum that serve as a stem cell reservoir for periosteal appositional bone growth and fracture repair. PDGFR reporter+ progenitors give birth to Nestin+ periosteal cells before becoming osteoblasts and osteocytes during these processes. The ablation of PDGFR reporter+ cells by diphtheria toxin resulted in impairments in cortical bone production during homeostasis and a smaller hard callus after fracture repair. Both mouse PDGFR reporter+ periosteal cells and human Pdgfr+ periosteal progenitors grow, ossify, and recruit marrow more than their counterpart periosteal cells after ossicle transplantation, although PDGFR reporter periosteal cells show a susceptibility to chondrogenesis in development.

PDGFR (Platelet-Derived Growth Factor Receptor) is a member of the receptor tyrosine kinases type III protein family, which has five immunoglobulin-like domains in the extracellular ligand-binding domain, a single membrane-spanning motif, and a split intracellular tyrosine kinase domain. The interaction of a ligand causes two receptors to dimerize and autophosphorylate certain tyrosine residues in their cytoplasmic domains. Adaptor proteins that initiate signal transduction use these phosphotyrosine residues as docking sites. The Mitogen-Activated Protein Kinase (MAPK)/Extracellular Signal-Regulated Kinase (ERK) pathway, the phosphatidylinositol 3-kinase/Akt pathway, and the phospholipase C-(PLC-) pathway are three primary signal transduction pathways that PDGFR can activate. The function of PDGFR during mouse embryogenesis has also been thoroughly researched using the patch (Ph) mutant, which missing the Pdgfra gene due to the massive chromosomal 5 excision. Spina bifida and abnormalities in the development of the lung, circulatory system, and craniofacial tissue are seen in homozygous Ph/Ph embryos. PDGFR knockout mice have a phenotype that is comparable to Patch mutants, particularly when it comes to craniofacial abnormalities. As a result, PDGFR is critical for the embryonic development of the cranial mesenchyme. Conditional Pdgfra ablation in the NC causes cleft palate formation and poor ossification of neural crest cells-derived faces bones, underlining the relevance of this signaling pathway in craniofacial development. The embryonic mortality of Patch mutants and PDGFR deficient animals, on the other hand, makes elucidating the receptor's cell-autonomous roles in cerebral neural crest cells more difficult.

PDGFR signaling is critical in the formation of mesenchymal cells and connective tissue throughout development. They primarily contribute to organ connective tissue types such bone, teeth, fat, and stromal components, as well as providing an

Correspondence to: Almeida Barros, Department of Surgery, State University of Campinas, São Paulo, Brazil, E-mail: Almeida@barros.br

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architectural supportive niche for parenchymal cells. There is less evidence that PDGFR cells differentiate into parenchymal cells following organogenesis. Platelet-Derived Growth Factor (PDGF) is an important mediator during early fracture healing and a potent stimulator for skeletal precursor cells. PDGF receptor expression is found in periosteal progenitor cells during fracture repair. Whether PDGFR+ periosteal stem cells are progenitor cells for distinct subpopulations within this cellular niche and contribute to self-renewal and bone regeneration are crucial but mainly unanswered concerns.