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Editorial

Platelet derived growth factor (PDGF)

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1. Introduction

Growth factors, which are proteins and polypeptides, have grown in significance in numerous physiologic contexts. Numerous polypeptide growth factors have been discovered as a result of research over the past few decades, and the majority of these have been found to play important roles in basic biological processes such cell growth, migration, and differentiation. These growth factors are easily split into three groups and can be classified into several families based on their structural and functional features. One category consists of substances that promote the growth or division of diverse cell types; examples include nerve growth factor, insulin-like growth factor I, and epidermal growth factor (EGF), and more than 20 have been identified. The second category consists of the cytokines, which are immune system regulators produced by lymphocytes and macrophages. Colony-stimulating elements, which control the maturation and proliferation of red and white blood cells, make up the third group.

2. Sources

osteoblastic cells that have not been activated. A variety of tumour cell lines, as well as macrophages, endothelial cells, fibroblasts, glial cells, astrocytes, myoblasts, and smooth muscle cells, are examples of additional cells. IL1, IL6, INF-

alpha, IGF-beta, and EGF can all stimulate the production of PDGF.

2.1. PDGF ISO forms

The disulfide-bonded homodimers of polypeptide chains A, B, C, and D as well as the heterodimer PDGF-AB make up the PDGF family. Inside the producer cells, PDGF-AA, -AB, and -BB are already cleaved and stored in secretory vesicles. The N-terminal CUB-domains of PDGF-CC and -DD, in contrast, must be removed in order to activate the growth factors once they are secreted as active precursor molecules. In the case of PDGF-CC, tissue-type plasminogen activator (tPA) or plasmin is responsible for this cleavage, while in the case of PDGF-DD, urokinase-type PA (uPA) or matriptase (MT-Sp1) fulfil the function.

3. Signalling Via PDGF Receptors

PDGF isoforms bind to the tyrosine kinase receptors and to have an effect on cells. The two PDGF receptors PDGFR α and PDGFR β , share structural similarities and are made up of intracellular kinase domains with distinctive inserts of roughly 100 amino acid residues that have no resemblance to kinases and extracellular domains with five immunoglobulin (Ig)-like domains.

Receptor dimerization, which is further stabilised by direct receptor-receptor contacts involving Ig-like domain 4, is caused by ligand binding, which primarily involves Ig-like domains 2 and 3. The receptors also bind and

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activate transcription factors known as STATs, which are then transported to the nucleus where they function as transcription factors. Finally, the adaptor molecules that the receptors bind can form complexes with other signalling molecules despite lacking intrinsic enzymatic activity.

4. Clinical Significance

4.1. PDGF and the bone remodelling cycle

The ability of the skeleton to regenerate both during homeostasis and after injury is strong and innate. The remodelling cycle, in which cell populations are attracted and differentiated for the objectives of bone resorption or bone creation, is what distinguishes this amazing regeneration process. An intricate system of growth factors and cytokines orchestrates and controls these actions. PDGF is one of the essential biological elements in charge of reparative osseous activity. The majority of mesenchymal-derived cells have cell-surface receptors that PDGF binds to in order to activate the reparative processes in various tissue types. As a powerful mitogen and chemoattractant with the capacity to stimulate angiogenesis, PDGF serves as a crucial mediator in tissue repair.

4.2. PDGF expression and function in bone healing

The four genes that encode the four members of the PDGF polypeptide growth factor family—PDGF-A, B, C, and D—are spread across many chromosomes. While PDGF-C and PDGF-D only exist as homodimers, PDGF-A and PDGF-B can form both homodimers and heterodimers. Given that PDGF has a half-life of around 30 minutes when circulating in the blood, it is likely that successful therapeutic treatment will depend heavily on local distribution of the growth factor. Bone repair is characterised by the activation of the coagulation cascade and creation of a blood clot at the site of trauma after damage and haemorrhage. PDGF-AB, PDGF-AA, PDGF-

BB, and PDGF-CC are among the cytokines released by aggregating platelets when they enter the growing blood clot. By initially recruiting and activating neutrophils and macrophages, the PDGFs function early in the wound-healing cascade, by initially attracting and activating neutrophils and macrophages, which are key cell mediators of early tissue repair.

Following the formation of granulation tissue, the next stage in endochondral bone repair, these cells act as a constant source of PDGFs and other growth factors. The local release of PDGF into the wound-healing milieu also promotes the chemotaxis and mitogenesis of a variety of mesenchymal derived cells, including fibroblasts, osteoblasts, chondrocytes, and smooth muscle cells.


5. Conclusion

A dimeric glycoprotein known as platelet-derived growth factor (PDGF) can have two A subunits (PDGF-AA), two B subunits (PDGF-BB), or one of each (PDGF-AB). Although platelets synthesise, store, and release PDGF (in the alpha granules of platelets), other cells, such as smooth muscle cells, activated macrophages, and endothelial cells, also generate it. One of the many growth factors that control cell division and growth is platelet-derived growth factor (PDGF).

6. Conflict of Interest

None.

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