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Review Article

A New Insight on Targeting SDF-1 As an Emerging Therapeutic Potential Target Against Osteoarthritis

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Abstract

Osteoarthritis (OA), a primary contributor to musculoskeletal disability, remains without disease-modifying therapies despite extensive investigation into inflammation and catabolism over the years. Stromal cell-derived factor-1 (SDF-1/CXCL12) has been identified as a significant therapeutic target, playing a crucial role in cartilage degradation, synovial inflammation, osteoclast recruitment, and subchondral bone remodelling through CXCR4/CXCR7 signalling pathways. This review consolidates recent findings indicating that increased levels of synovial and serum SDF-1 are associated with the severity of OA. Additionally, mechanistic studies have shown that SDF-1 induces MMP-3/9/13, ADAMTS-4/5, and COX-2 in chondrocytes via the PI3K/AKT, MAPK/ERK, and JAK/STAT pathways. SDF-1 enhances the infiltration of CD^{4+} T-cells and macrophages, stimulates angiogenesis through VEGF, and facilitates osteoclastogenesis, thereby creating a pathological interaction among cartilage, synovium, and bone. The use of CXCR4 antagonists (AMD3100 analogues), SDF-1 neutralizing agents, and miR-146a-5p modulation in preclinical settings demonstrates a reduction in matrix degradation, maintenance of aggrecan/collagen II levels, and inhibition of bone sclerosis in ACLT/DMM models. CXCR7-biased signalling and SDF-1-guided MSC homing offer regenerative potential when combined with catabolic suppression. The results indicate that modulating the SDF-1/CXCR4/CXCR7 axis represents a complex strategy for disease-modifying osteoarthritis drugs, highlighting the need for clinical translation via targeted nanoparticles and the selection of patients guided by biomarkers.

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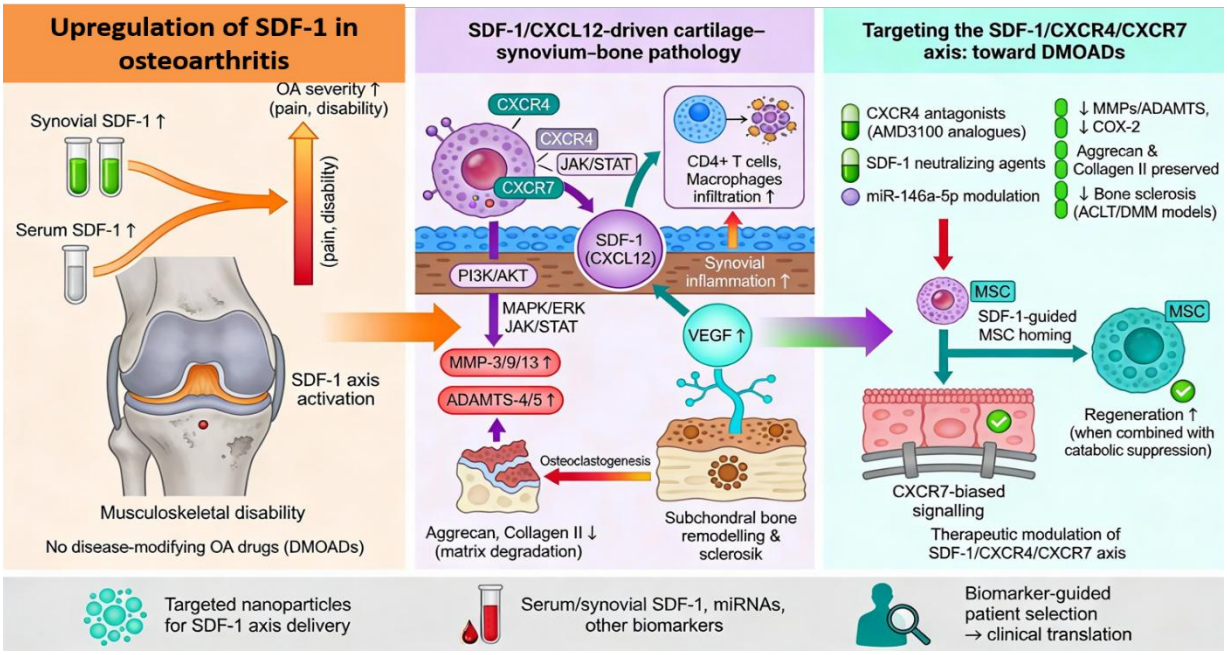

Graphical abstract: This graphical abstract illustrates the upregulation of SDF-1 in osteoarthritis, resulting in the pathological infiltration of macrophages that leads to bone degeneration and osteoclastogenesis. Targeting SDF-1, developing antagonists against CXCR4, and applying miR-146-5p may ameliorate the condition of osteoarthritis patients by promoting bone tissue regeneration and suppressing catabolic processes.

1. INTRODUCTION

Osteoarthritis (OA) is a common degenerative joint condition marked by the gradual breakdown of cartilage, inflammation of the synovial membrane, the development of bone spurs, and changes in the underlying bone structure, resulting in persistent pain and disability among older individuals. The existing pharmacological treatments primarily address symptoms and do not prevent structural progression, highlighting the critical necessity for disease-modifying osteoarthritis drugs (DMOADs) aimed at core molecular drivers. Among the emerging pathways, the chemokine stromal cell-derived factor-1 (SDF-1, also known as CXCL12) and its receptors CXCR4 and CXCR7 have garnered increasing interest as crucial regulators of joint inflammation, cartilage catabolism, and pathological bone turnover.[1–3].

SDF-1 is an 8-kDa chemokine that was first identified in bone marrow stromal cells. It plays a crucial role in regulating hematopoietic stem cell retention, angiogenesis, and tissue repair through its interaction with the G-protein-coupled receptor CXCR4 and the atypical chemokine receptor CXCR7. In the joint, SDF1 is prominently expressed by synovial fibroblasts, macrophages, endothelial cells, and subchondral bone stromal cells, whereas CXCR4 and CXCR7 are found on chondrocytes, osteoclast precursors, and immune cells that infiltrate the synovium. The activation of CXCR4 by SDF-1 initiates canonical Gi-dependent signalling through the PI3K-AKT, ERK/MAPK, and PLC-β pathways. Additionally, it engages β-arrestin–biased signalling through CXCR4-CXCR7 heteromers, which together facilitate chemotaxis, proliferation, matrix metalloproteinase (MMP) secretion, and angiogenesis.[1,4,5].

Clinical and experimental findings are progressively highlighting the role of the SDF-1/CXCR4 axis in the development of OA. Consistent findings indicate that elevated SDF-1 levels are present in the synovial fluid and plasma of individuals with osteoarthritis and rheumatoid arthritis, showing a correlation with disease activity and markers of cartilage degradation. The process of synovectomy or the pharmacological inhibition of CXCR4 leads to a decrease in circulating levels of SDF-1, MMP-9, and MMP-13. This suggests that synovial SDF-1 plays a significant role in driving catabolic signalling within articular cartilage. In vitro, SDF-1 stimulation of chondrocytes leads to an increase in the secretion of MMP-3, MMP-9, and MMP-13, while also promoting hypertrophic differentiation, which in turn accelerates the breakdown of the extracellular matrix. In subchondral bone, the activation of mTORC1 in pre-osteoblasts leads to an upregulation of Cxcl12, subsequently promoting abnormal bone sclerosis and cartilage degeneration. The neutralisation of SDF-1 using specific antibodies mitigates these alterations and reduces the progression of OA in mice.[3,5,6].

In addition to its role in cartilage, SDF-1 plays a crucial role in the recruitment and activation of various immune cell subsets within arthritic joints. The gradients of CXCL12 play a crucial role in directing the migration and retention of $CD4^+$ memory T cells, B cells, monocytes, and macrophages within the synovium. In this environment, the stimulation of fibroblast-like synoviocytes by chemokines enhances the production of IL-6, IL-8, TNF, and various other inflammatory mediators,

contributing to a self-reinforcing cycle of synovitis. In bone, CXCL12 produced by CXCL12-abundant reticular (CAR) cells plays a crucial role in regulating the chemotaxis, differentiation, and survival of osteoclast precursors, thus linking SDF-1/CXCR4 signalling to pathological bone resorption and structural joint damage. The integration of these actions establishes SDF-1 as a central chemokine that connects synovial inflammation, cartilage degradation, angiogenesis, and osteoclast-driven bone erosion in osteoarthritis[1,3,7].

Recent studies have broadened this idea by showing that specific modulation of the CXCL12/CXCR4 axis can reduce experimental arthritis and OA-like pathology. Selective CXCR4 antagonists and Neutraligands significantly reduce inflammatory cytokine production, immune cell infiltration, and joint destruction in models of collagen-induced arthritis and juvenile idiopathic arthritis, confirming CXCR4 as a viable target within chemokine networks. In OA-relevant systems, the natural compound Astragaloside IV has been identified as a novel CXCR4 antagonist that blocks CXCL12-induced ADAMTS-4/5 upregulation and protects cartilage and subchondral bone in monosodium iodoacetate-induced OA rats, indicating its potential for repurposing as a joint-protective agent. On the other hand, the protective functions of SDF-1 that depend on context have been documented, such as maintaining mitochondrial homeostasis and supporting chondrocyte survival in particular OA models, highlighting the intricate nature of this chemokine in musculoskeletal biology.[7–10].

In summary, the growing body of evidence from the past three years indicates that SDF-1/CXCL12 and its receptors CXCR4/CXCR7 represent promising, albeit complex, therapeutic targets for OA. Integrating chemokine biology with advancements in small-molecule antagonists, biologics, and nanomedicine-based delivery could provide a novel disease-modifying strategy targeting SDF-1. This approach may effectively tackle cartilage degradation, synovial inflammation, and abnormal bone remodelling simultaneously. This review offers a comprehensive update on the biology of SDF1 in joint tissues, summarises the latest preclinical and translational studies focused on targeting CXCL12/CXCR4 in osteoarthritis, and explores future perspectives and challenges in utilising this pathway for clinical intervention.

2. The cytokine SDF-1 system

Following the activation of CXCR4 by SDF-1/CXCL12, various signalling pathways initiate pathological responses within the rheumatoid arthritis synovium. The majority of pathways exhibit sensitivity to pertussis toxin (PTX), suggesting a reliance on Gi proteins. In this context, activated Gi inhibits adenylyl cyclase, while Gβγ subunits initiate the activation of phospholipase C-β (PLC-β), phosphoinositide-3 kinase (PI3K), and mitogen-activated protein kinase (MAPK) [8]. The PI3K/AKT and MAPK/ERK pathways enhance chemotaxis, drive the migration of synovial fibroblasts, and stimulate the secretion of matrix metalloproteinases (MMPs), specifically MMP-2 and MMP-9. This process aids in the invasion of immune cells through basement membranes and contributes to the formation of pannus.[11].

The MMPs also promote the expression of vascular endothelial growth factor (VEGF), which is crucial for driving synovial angiogenesis necessary for the progression of RA. [12]. The inhibition of PI3K or MAPK pathways through pharmacological means effectively prevents CXCR4-mediated migration in RA fibroblast-like synoviocytes and T cells.[8]. The SDF-1/CXCR4 signalling pathway plays a crucial role in coordinating cell proliferation, chemotaxis, MMP production, and angiogenesis, all of which are essential elements of destructive synovitis.[3].

CXCR4 additionally initiates G-protein-independent JAK/STAT signalling via the temporary recruitment of JAK2/JAK3, facilitating the nuclear translocation of STAT and the expression of inflammatory genes. Although mainly mediated by β-arrestin, PTX pretreatment extends the association between JAK and CXCR4, indicating that Gi coupling influences receptor recycling and the persistence of STAT activation.[1].

The molecular mechanisms place SDF-1/CXCR4 modulation within the developing framework of chemokine network-targeted therapies for rheumatoid arthritis, necessitating clinical exploration of intra-articular CXCR4 antagonists and combinatorial approaches with current DMARDs to attain disease modification beyond mere symptomatic alleviation.

3. Mechanism of action of SDF-1 in cartilage degradation

In Osteoarthritis, SDF-1 (CXCL12) facilitates the degradation of cartilage by directly activating chondrocytes and exerting indirect influences on inflammation and bone remodelling processes[13,14]. Direct Chondrocyte Catabolism via CXCR4 SDF-1 interacts with CXCR4 on chondrocytes, leading to the activation of PI3K/AKT, MAPK/ERK, and JAK/STAT pathways.

Increases the levels of MMP-3, MMP-9, MMP-13, and ADAMTS-4/5, leading to the breakdown of type II collagen, aggrecan, and proteoglycans (Figure 1). Stimulates the expression of COX-2/PGE2, leading to increased matrix degradation and heightened pain levels[13,15]. The recruitment of CD^{4+} T cells, monocytes, and macrophages through CXCR4 leads to the establishment of a pro-inflammatory microenvironment. Induces fibroblast-like synoviocytes (FLS) to produce IL-6, IL-8, and TNF-α, subsequently activating chondrocytes. Facilitates the formation of new blood vessels through the induction of VEGF from endothelial cells and chondrocytes[13,14].

Subchondral Bone-Cartilage Crosstalk SDF-1 released by subchondral osteoblasts diffuses into the cartilage, facilitating the recruitment of osteoclasts through CXCR4 on precursor cells. Increases MMP-9 activity and facilitates collagen transmigration, establishing a connection between bone sclerosis and the erosion of overlying cartilage[16,17]. CXCR7 influences this process by removing surplus SDF-1 or favouring β-arrestin signalling pathways.

The compromised matrix emits damage-associated molecular patterns (DAMPs), which in turn lead to an increased expression of SDF-1/CXCR4, perpetuating a harmful cycle. The inhibition of the SDF-1 pathway by miR-146a-5p

safeguards cartilage, underscoring its essential role in degradation[17,18]. In conclusion, SDF-1 plays a crucial role in the destruction of cartilage by inducing catabolic enzymes. (MMPs/ADAMTS), promoting the infiltration of inflammatory cells, facilitating angiogenesis, and contributing to a vicious cycle between bone and cartilage. This positions CXCR4 blockade as a logical target for therapeutic intervention.

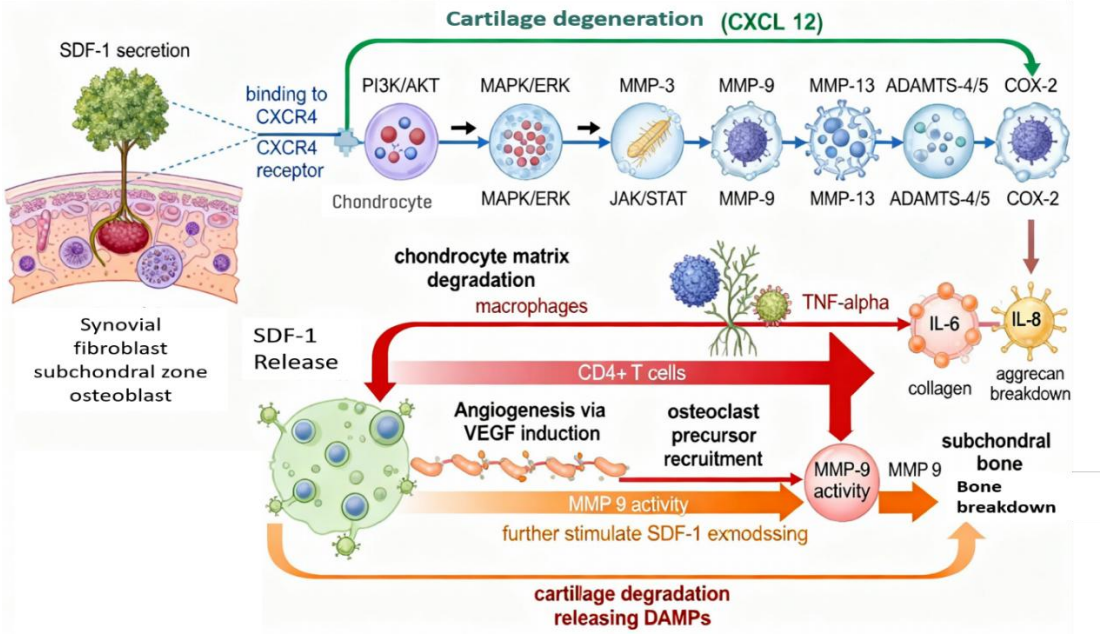

Figure 1. This figure represents the release of SDF-1 with the development of osteoarthritis. Upregulation of different metalloproteinases, including MMP-9 and MMP-13, proinflammatory cytokines such as IL-6 and TNF, and influx of macrophages and osteoclasts, leads to the degradation of the cartilage matrix and subchondral bone.

4. Therapeutic strategies targeting the SDF-1/CXCR4/CXCR7 axis

Therapeutic approaches aimed at the SDF-1/CXCR4/CXCR7 axis in osteoarthritis (OA) concentrate on diminishing catabolic and inflammatory signalling, while also maintaining or utilising regenerative functions [14].

4.1. CXCR4 Inhibitors

Small-molecule or peptide CXCR4 antagonists can inhibit SDF-1 binding, which leads to a decrease in the production of MMP-3, MMP-9, and MMP-13, as well as a reduction in cartilage matrix degradation and osteoclast recruitment.[15,19]. The administration of CXCR4 blockers, whether systemic or intra-articular, such as AMD3100 analogues and optimised CXCR4 inhibitors, has shown a reduction in OA-like changes and has protected subchondral bone in preclinical models.[11,20]. Local delivery methods involving hydrogels and nanoparticles are suggested to enhance joint targeting while reducing hematopoietic side effects.[16]. Therefore, Recent developments in the clinical application of therapies aimed at bone targets are discussed, along with an analysis of the challenges faced in implementing these therapies in clinical settings and the anticipated future directions in this field.

4.2. Inhibition of SDF-1 or Gene Suppression

Reducing SDF-1 levels in joint tissues can disrupt chemotactic and catabolic signalling pathways. Neutralising antibodies or aptamers targeting SDF1 diminish the availability of CXCL12, leading to a reduction in inflammatory cell infiltration and angiogenesis.[3]. The intra-articular administration of SDF-1-targeted siRNA or shRNA through either viral or non-viral vectors results in the downregulation of CXCL12 expression in the synovium and subchondral bone.[21]. This process subsequently leads to a decrease in MMPs and helps in the preservation of type II collagen and aggrecan.

4.3. Modulation of CXCR7 and Signalling

Given that CXCR7 functions both as a scavenger and a signalling receptor, the modulation of CXCR7 presents an opportunity to alter SDF-1 signalling without completely inhibiting it.

Improving the “decoy” function of CXCR7 leads to increased uptake and degradation of SDF-1, which in turn reduces CXCR4-mediated catabolic signalling.[3,14]. Creating ligands that steer SDF-1 signalling towards β-arrestin-dependent, tissue-protective pathways (such as pro-survival and pro-repair) while minimising G-protein-mediated catabolism is a suggested approach for precise regulation of the axis[14].

4.4. SDF-1-Directed Regenerative Approaches

The chemotactic properties of SDF-1 may be utilised to improve cartilage repair, provided that catabolic signalling is regulated. The modification of mesenchymal stem cells (MSCs) to overexpress CXCR4 aims to enhance their targeting to SDF-1-rich osteoarthritic joints. When paired with a local catabolic blockade[22], this approach has demonstrated improved cartilage regeneration and matrix deposition. Innovative

biomaterials, such as scaffolds or microspheres, that release low, controlled doses of SDF-1 can effectively attract endogenous progenitors to defects. Additionally, they can co-deliver anti-catabolic agents or CXCR4 modulators to inhibit degradation[16].

4.5. Integrative Strategies and Precise Delivery

Considering SDF-1's involvement in both damage and repair processes, the use of combination strategies is especially appealing. Utilising dual-target strategies, such as the blockade of SDF-1/CXCR4 in conjunction with anti-inflammatory or anti-osteoclast agents like bisphosphonates or RANKL inhibitors, has the potential to synergistically mitigate bone erosion and cartilage degradation[16,23]. Systems utilising nanoparticles for the co-delivery of CXCR4 antagonists and chondrogenic growth factors, such as TGF-β and IGF-1, are currently under investigation for their potential to inhibit catabolism while simultaneously promoting regeneration [11].

The collective strategies seek to reprogram the SDF-1/CXCR4/CXCR7 axis, transforming it from a contributor to inflammation, angiogenesis, and matrix degradation into a regulated facilitator of repair. This approach presents a promising avenue for developing genuine disease-modifying therapies for osteoarthritis.

5. CONCLUSION

The SDF-1/CXCL12 axis serves as a crucial intersection in the development of osteoarthritis, bringing together the activation of catabolic chondrocytes, the movement of inflammatory cells, the process of pathological angiogenesis, and the remodelling of bone mediated by osteoclasts into a cohesive mechanistic framework. Recent studies present significant clinical-translational evidence: increased serum/synovial SDF-1 is associated with radiographic severity and pain, while targeted blockade maintains cartilage matrix and subchondral microarchitecture in preclinical models, meeting essential DMOAD criteria. Therapeutic strategies that utilise CXCR4 antagonism, SDF-1 neutralisation, CXCR7 modulation, and SDF-1-guided regeneration provide a sophisticated approach to managing this complex pathway, steering clear of the drawbacks associated with widespread immunosuppression. Local delivery systems, such as nanoparticles and hydrogels, effectively tackle concerns related to systemic toxicity. Meanwhile, circulating SDF-1 has been identified as a dynamic biomarker for monitoring disease progression and therapeutic response.

There are ongoing difficulties in refining receptor bias (between G-protein and β-arrestin signalling), coordinating interventions with the stages of OA, and confirming effectiveness through human trials. Future investigations should focus on Phase II studies of intra-articular CXCR4 inhibitors, combination therapies with anti-RANKL agents, and tailored approaches that stratify patients based on SDF-1/CXCR4 expression profiles. Effectively leveraging SDF-1 signalling has the potential to shift OA from a state of ongoing degeneration to one of manageable repair, addressing a significant gap in musculoskeletal medicine.

Future Prospect

Future investigations into the SDF-1/CXCR4/CXCR7 axis in osteoarthritis (OA) must transition from preclinical potential to clinically applicable, targeted treatments. This necessitates comprehensive mechanistic analysis, strategic medication development, and biomarker-guided patient selection, included into early-phase studies. Methodically enhance CXCR4 antagonists (AMD3100 analogues) for targeted joint delivery, pharmacokinetics, and safety, encompassing local (intra-articular) vs systemic administration and prolonged-release depots or hydrogels.

Enhance SDF-1 neutralisation techniques (antibodies, aptamers, decoy receptors) and miR-146a-5p-based methodologies, emphasising off-target profiling, longevity of catabolic inhibition, and reversibility in the event of adverse reactions. Design nanoparticle platforms (liposomes, polymeric nanoparticles, exosomes, or stimuli-responsive carriers) that selectively concentrate in inflammatory synovium and injured cartilage, delivering CXCR4 antagonists, SDF-1 inhibitors, or miRNA payloads to enhance effectiveness and reduce systemic exposure.

Utilise SDF-1-guided MSC homing and CXCR7-biased signalling alongside catabolic inhibition to formulate "two-arm" treatments that initially suppress matrix breakdown and then enhance endogenous or cell-based cartilage repair. Investigate combination therapies that integrate SDF-1 axis modulation with current osteoarthritis treatments (viscosupplementation, PRP, MSC injections, physical rehabilitation) to see if chemokine reprogramming may transform predominantly palliative approaches into really disease-modifying techniques. Further scientific investigation into the role of SDF-1 in cartilage tissue degeneration, as well as the suppression of this process using antagonists and the use of nanotechnology, is necessary for the development of successful treatment techniques.

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