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LncRNA MALAT1 as a Competitive Endogenous RNA in Osteoarthritis: Mechanisms and Therapeutic Prospects

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Abstract: Osteoarthritis (OA) is a complex degenerative joint disease characterized by articular cartilage degeneration, synovial inflammation, and osteophyte formation, with its exact pathogenesis not yet fully elucidated. In recent years, competing endogenous RNA (ceRNA) regulatory networks, particularly the role of long non-coding RNAs (LncRNAs) within them, have garnered increasing attention. This review focuses on LncRNA MALAT1, summarizing its ceRNA mechanism in the initiation and progression of OA. The article systematically elaborates on how MALAT1 acts as a molecular sponge to adsorb specific microRNAs (miRNAs), thereby relieving the inhibition of downstream target genes, and consequently regulating key pathological processes such as chondrocyte metabolism, extracellular matrix homeostasis, inflammatory response, and cell apoptosis. Furthermore, this review summarizes the potential value and latest research progress in targeting the MALAT1-ceRNA axis for early OA diagnosis, disease monitoring, and the development of novel therapeutic strategies, aiming to provide a new theoretical basis and perspective for the precise diagnosis and treatment of OA.

Keywords: Osteoarthritis; Competing endogenous RNA; LncRNA MALAT1; Chondrocytes; ceRNA network; Molecular mechanism

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1. Introduction

Osteoarthritis is the most common joint disease worldwide, imposing a heavy socioeconomic burden. Its pathological process involves multiple interacting factors, including chondrocyte dysfunction, imbalance between extracellular matrix degradation and synthesis, chronic low-grade inflammation, and abnormal subchondral bone remodeling [1]. Traditional treatment strategies primarily focus on symptom relief and cannot reverse disease progression; therefore, in-depth exploration of the molecular mechanisms underlying OA is crucial. In recent

years, research on non-coding RNAs has revealed their central role in gene expression regulation [2]. Among these, the ceRNA hypothesis proposes that RNA transcripts (such as LncRNAs and circular RNAs) can competitively bind miRNAs through shared miRNA response elements, thereby relieving the miRNA-induced inhibition of target messenger RNAs, forming a complex post-transcriptional regulatory network [3].

LncRNA MALAT1 (metastasis-associated lung adenocarcinoma transcript 1) has been confirmed as a key ceRNA regulator in various diseases. In the field of OA, accumulating evidence indicates that MALAT1 is aberrantly expressed and extensively participates in the pathophysiological processes of OA through ceRNA mechanisms [4]. This review will delve into the specific mechanisms of LncRNA MALAT1 as a ceRNA in OA, outline the key signaling pathways it regulates, and explore the potential of diagnostic biomarkers and novel therapeutic strategies based on this mechanism.

Studies have shown that MALAT1 expression is upregulated in the subchondral bone and chondrocytes of OA patients, closely correlating with joint inflammation and degeneration [5]. For instance, in the subchondral bone of OA patients, MALAT1 expression levels positively correlate with serum concentrations of DKK1 and galectin-1, suggesting its involvement in bone metabolism regulation [5]. Functional studies further confirmed that knocking down MALAT1 in osteoblasts from OA patients leads to significant expression changes in 155 genes, including PTGS2, and enhances basal and IL-1β-induced prostaglandin E2 secretion, indicating that MALAT1 plays a crucial role in regulating subchondral bone inflammation in OA [5]. Additionally, an allele-specific expression imbalance analysis based on a genome-wide association study identified MALAT1 as a key long intergenic non-coding RNA within an OA risk locus, where its expression imbalance is significantly associated with OA risk [6]. These findings collectively establish the central role of MALAT1 in OA pathogenesis.

As a ceRNA, MALAT1 primarily regulates downstream miRNA and target gene expression through a "sponge" adsorption effect, thereby influencing multiple pathological aspects of OA. For example, MALAT1 can competitively bind miR-146a, relieving its inhibition of target genes, ultimately reducing IL-1β-induced articular chondrocyte apoptosis and extracellular matrix degradation [7]. In human synovial mesenchymal stem cells (hSMSCs), MALAT1 has been shown to act as a ceRNA, adsorbing miR-212-5p and subsequently regulating its target gene MyD88, thereby inhibiting the chondrogenic differentiation of hSMSCs; conversely, inhibiting MALAT1 promotes chondrogenic differentiation and reduces joint degeneration in a rat OA model [8]. Another study revealed that the MALAT1/miR-15b-5p axis influences lipopolysaccharide-induced chondrocyte damage by regulating the Wnt/β-catenin signaling pathway [9]. These studies clearly outline the complex molecular landscape through which MALAT1 regulates OA progression via the ceRNA network.

Based on its ceRNA mechanism, MALAT1 demonstrates significant potential in the diagnosis and treatment of OA. In diagnostics, studies have found that MALAT1 expression levels in the serum of OA patients are altered and correlate with postoperative chronic pain, suggesting its potential as a circulating biomarker [10]. Therapeutically, targeting MALAT1 or its ceRNA network has emerged as a novel strategy. For instance, extracellular vesicles derived from human mesenchymal stem cells overexpressing MALAT1 can inhibit IL-1β-induced chondrocyte inflammation and apoptosis, alleviating cartilage degeneration in a rat OA model [11]. Traditional Chinese medicine formulas, such as Tougu Xiaotong Capsules, have also been shown to regulate the MALAT1/miR-16-5p ceRNA axis, ameliorating "cholesterol-iron" metabolic disorders in OA chondrocytes, thereby delaying OA progression [12]. Furthermore, docosahexaenoic acid (DHA) exerts anti-inflammatory, chondroprotective, and chondrogenic effects by downregulating MALAT1 levels [13]. These studies provide a solid theoretical basis and experimental foundation for developing novel OA therapies targeting MALAT1.

2. Biological characteristics of LncRNA MALAT1 and its expression regulation in OA

2.1. Basic structure and function of MALAT1

MALAT1 is a highly conserved long non-coding RNA, initially discovered in non-small cell lung cancer, with a transcript length exceeding 200 nucleotides [14]. As a nuclear-retained lncRNA, MALAT1 plays a crucial role in transcriptional regulation and alternative splicing within the nucleus [4]. In addition to its nuclear functions, MALAT1 has also been shown to shuttle to the cytoplasm, where it functions as a competing endogenous RNA (ceRNA). In the cytoplasm, MALAT1 can act as a molecular sponge, binding and inhibiting the activity of specific microRNAs (miRNAs), thereby relieving the inhibitory effect of these miRNAs on their downstream target genes, participating in the regulation of various cellular processes [15]. In the pathological context of osteoarthritis (OA), the expression level of MALAT1 is closely related to the severity of cartilage degeneration. Multiple studies have indicated that MALAT1 expression is generally significantly upregulated in OA articular tissues, suggesting its role as a molecular factor promoting OA progression [3]. For example, MALAT1 expression is upregulated in chondrocytes from OA patients, exacerbating cartilage destruction by regulating inflammatory pathways and extracellular matrix metabolism [7].

2.2. Regulation of MALAT1 expression by the OA pathological microenvironment

The pathological microenvironment of OA, particularly its various pathogenic factors, can significantly regulate MALAT1 expression in chondrocytes. Inflammatory factors constitute a core component of the OA microenvironment. Key pro-inflammatory cytokines such as interleukin-1β (IL-1β) and tumor necrosis factor-α (TNF-α) have been shown to induce the upregulation of MALAT1 expression in chondrocytes [3]. For instance, MALAT1 expression levels significantly increase in IL-1β-treated chondrocyte models [16]. Besides inflammatory factors, epigenetic modifications also participate in the transcriptional regulation of MALAT1. Mechanisms such as DNA methylation and histone modifications can affect the chromatin state of the MALAT1 locus, thereby modulating its transcriptional activity in OA chondrocytes [4]. Additionally, the hypoxic microenvironment commonly found in OA cartilage tissue is another critical factor regulating MALAT1 expression. Hypoxia can promote MALAT1 transcription by activating transcription factors like hypoxia-inducible factor-1α (HIF-1α) [3]. This upregulated MALAT1 can further exacerbate inflammatory responses and extracellular matrix degradation, forming a positive feedback loop that continuously drives cartilage destruction in OA [7].

3. Molecular mechanisms of MALAT1 as a ceRNA regulating chondrocyte metabolic homeostasis in OA

3.1. The MALAT1/miRNA/target gene axis regulating extracellular matrix synthesis and degradation

MALAT1, acting as a competing endogenous RNA, plays a key regulatory role in the balance between synthesis and degradation of the extracellular matrix in osteoarthritic chondrocytes by adsorbing specific microRNAs and relieving the inhibition of downstream target genes. Studies have shown that MALAT1 expression is upregulated in OA cartilage tissue and is closely associated with matrix degradation [7]. Specifically, MALAT1 acts as a molecular sponge for miR-146a, competitively binding to it, thereby relieving the inhibitory effect of miR-146a on its target gene, ADAMTS5. ADAMTS5 is a crucial aggrecanase; its upregulation accelerates

the degradation of the core proteoglycan in the extracellular matrix, directly promoting cartilage destruction [7]. Furthermore, another important regulatory pathway involves the adsorption of miR-150-5p by MALAT1. By sponging miR-150-5p, MALAT1 upregulates the expression of its target gene, matrix metalloproteinase 13 (MMP13), thereby enhancing the breakdown of type II collagen and compromising the integrity of the cartilage structure [17]. Under OA pathological conditions, the aberrant expression of MALAT1 also affects pro-synthesis pathways. For instance, MALAT1 can act as a ceRNA for miR-127-5p, theoretically promoting the expression of SOX9, a key transcription factor for chondrogenesis, by relieving miR-127-5p inhibition, which would favor extracellular matrix synthesis. However, in the inflammatory environment of OA, this pro-synthesis pathway is often suppressed, leading to an imbalance between synthesis and degradation, ultimately exacerbating cartilage degeneration [18].

3.2. Role of the MALAT1-ceRNA network in regulating chondrocyte apoptosis and autophagy

The MALAT1-ceRNA network also deeply participates in regulating the survival and death processes of chondrocytes, including apoptosis and autophagy. In terms of apoptosis regulation, MALAT1 sponges miR-181a-5p, upregulating the expression of its target gene Bcl-2, thereby inhibiting the mitochondrial pathway of apoptosis and exerting a protective effect on chondrocytes [19]. However, under OA pathological conditions, the aberrant high expression of MALAT1 may produce dual effects through complex networks. For example, some studies suggest that MALAT1 might promote apoptotic signals via other miRNAs (such as miR-20b), indicating the context-dependent regulation of apoptosis by MALAT1 in OA [20]. Regarding autophagy regulation, MALAT1 also plays a significant role as a ceRNA. MALAT1 acts as a molecular sponge for miR-142-3p, influencing the expression of autophagy-related genes like ATG7, thereby interfering with the autophagic flux in chondrocytes under stress conditions [21]. Autophagy is a crucial mechanism for maintaining cellular homeostasis, and dysregulation of autophagic flux is directly linked to decreased chondrocyte survival in OA. Therefore, through the ceRNA mechanism regulating miR-142-3p and its downstream targets, MALAT1 influences chondrocyte autophagic activity and thus participates in OA disease progression. These studies collectively reveal that MALAT1 plays a key role in the fine-tuning of chondrocyte apoptosis and autophagy by constructing a complex ceRNA network, and its expression imbalance is a significant factor contributing to the disruption of chondrocyte metabolic homeostasis in OA.

4. OA Synovial inflammation and subchondral bone remodeling mediated by the MALAT1-ceRNA axis

4.1. Regulation of inflammatory responses in synovial fibroblasts

In the pathological process of osteoarthritis (OA), chronic inflammation of the synovial tissue is a critical factor exacerbating joint destruction. Long non-coding RNA MALAT1 is highly expressed in OA synovial tissue and drives the inflammatory response in synovial fibroblasts by regulating downstream signaling pathways through competing endogenous RNA (ceRNA) mechanisms. Studies indicate that MALAT1 acts as a molecular sponge for microRNA-150 (miR-150). By adsorbing miR-150, it relieves the inhibition of downstream target genes, subsequently activating the nuclear factor κB (NF-κB) signaling pathway [3]. Activation of the NF-κB pathway prompts synovial fibroblasts to release large quantities of pro-inflammatory cytokines such as interleukin-6

(IL-6), interleukin-8 (IL-8), and tumor necrosis factor-α (TNF-α), significantly intensifying the inflammatory microenvironment within the joint cavity and creating a vicious cycle [3]. Furthermore, MALAT1 participates in the phenotypic transformation of synovial cells through the MALAT1/miR-145/signal transducer and activator of transcription 1 (STAT1) axis. This axis has been shown to promote abnormal proliferation of synovial cells and induce their transformation into a pro-inflammatory phenotype, thereby driving the chronic inflammatory process in OA [4]. These findings reveal that MALAT1 finely regulates synovial inflammation through a multi-dimensional ceRNA network, acting as a crucial molecular driver in the pathogenesis and progression of OA synovitis.

4.2. Influence on osteoclast differentiation and subchondral bone sclerosis

The characteristic pathological changes in OA extend beyond cartilage and include abnormal subchondral bone remodeling, with excessive osteoclast activation and subchondral bone sclerosis being central components. MALAT1 plays a significant role in this process through ceRNA mechanisms. In bone marrow mesenchymal stem cells or pre-osteoclasts, MALAT1 can act as a molecular sponge for microRNAs such as miR-214, thereby regulating the balance between the receptor activator of nuclear factor κB ligand (RANKL) and osteoprotegerin (OPG) system [3]. Dysregulation of the RANKL/OPG ratio directly promotes osteoclast differentiation and activation, leading to abnormal bone resorption, a key feature of typical subchondral bone remodeling in OA [3]. On the other hand, MALAT1 also indirectly promotes subchondral bone sclerosis by regulating osteogenesis-related signaling pathways such as Wnt/β-catenin. Aberrant activation of the Wnt/β-catenin pathway enhances osteoblast activity and accelerates bone formation, leading to thickening and sclerosis of the subchondral bone plate [3]. Such alterations in bone structure significantly impact the joint's biomechanical environment, increasing the stress on articular cartilage and further exacerbating cartilage wear and degradation. Therefore, the MALAT1-ceRNA axis profoundly participates in the complex regulatory network underlying abnormal subchondral bone remodeling in OA by simultaneously influencing both osteoclastic and osteoblastic processes.

5. Perspectives on OA diagnosis and treatment strategies targeting the MALAT1-ceRNA network

5.1. MALAT1 as a biomarker for OA diagnosis and prognosis assessment

Studies have found that the expression level of MALAT1 in the articular cartilage tissue of osteoarthritis (OA) patients is significantly higher than that in healthy controls, and its expression changes correlate with disease progression. One study showed that in OA articular cartilage, the relative expression level of MALAT1 is elevated, while the expression level of microRNA-146a (miR-146a) is decreased, with a significant negative correlation between the two [7]. This expression pattern suggests that MALAT1 may be involved in the pathological process of OA. Furthermore, another study compared the expression of MALAT1 in peripheral blood mononuclear cells (PBMCs) and fibroblast-like synoviocytes (FLSCs) from patients with rheumatoid arthritis (RA), OA, and traumatic arthritis (TA). It found that MALAT1 expression levels in PBMCs and FLSCs of OA patients were significantly higher than those in RA patients but lower than those in TA patients [22]. This disease-specific expression difference, combined with its negative correlation with the expression of inflammatory factors (such as IL-2, IL-6, TNF-α), highlights the potential value of MALAT1 as a biomarker reflecting OA disease activity and inflammatory status. Further studies using bioinformatics analysis to construct ceRNA networks suggest that MALAT1 can be linked to specific miRNAs (e.g., hsa-miR-22-3p) and their downstream target genes (e.g., PTEN,

ESR1). These networks may have specific utility in distinguishing OA from other skeletal disorders, such as osteoporosis [23]. Therefore, combining the detection of MALAT1 with key regulatory molecules within its ceRNA network could potentially constitute a more precise diagnostic panel, enhancing the specificity and sensitivity of early OA diagnosis and disease assessment.

5.2. Therapeutic intervention strategies based on the MALAT1-ceRNA axis

Gene silencing strategies represent a significant therapeutic direction targeting the MALAT1-ceRNA axis. Research has confirmed that specifically knocking down MALAT1 expression in vivo and in vitro can effectively delay OA progression. For instance, inhibiting MALAT1 expression in human synovial mesenchymal stem cells (hSMSCs) promotes their chondrogenic differentiation and exerts a chondroprotective effect via the miR-212-5p/MyD88 axis [8]. In animal models, MALAT1 inhibition similarly alleviated OA progression in rats [8]. This provides experimental evidence for gene therapy utilizing techniques such as small interfering RNA (siRNA) or short hairpin RNA (shRNA). Beyond direct MALAT1 silencing, oligonucleotide competition strategies based on its ceRNA mechanism also show promise. As MALAT1 acts as a molecular sponge for specific miRNAs (e.g., miR-146a, miR-15b-5p), thereby relieving the inhibition of their target genes [7,9], designing and delivering "decoy" sequences or anti-miRNA oligonucleotides (AMOs) that share the miRNA response elements with MALAT1 could competitively restore the function of the sponged miRNAs, correcting downstream signaling pathway abnormalities, such as excessive activation of the Wnt/β-catenin pathway [9].

To enhance the targeting and bioavailability of therapeutic agents and reduce off-target effects, developing efficient nanocarrier delivery systems is crucial. Currently, nanocarriers based on liposomes, exosomes, or polymeric materials are research hotspots. They can efficiently and specifically deliver MALAT1 inhibitors (e.g., siRNA) or protective miRNA mimics to articular chondrocytes [3]. Additionally, the modulatory effects of active ingredients from traditional Chinese medicine offer new perspectives for OA pharmacotherapy. For example, the natural compound quercetin (QUE) has been shown to directly target and downregulate MALAT1 expression, subsequently affecting the MALAT1/miR-9/NF-κB axis, thereby inhibiting cartilage damage and chondrocyte apoptosis in OA mice [16]. Similarly, Tougu Xiaotong Capsules (TGXTC) have been found to regulate the MALAT1/miR-16-5p ceRNA network, ameliorating "cholesterol-iron" metabolic disorders in OA chondrocytes, thus exerting a therapeutic effect [12,24]. These studies not only reveal novel mechanisms of action for traditional medicines but also provide valuable candidate molecules for developing new OA treatment strategies based on the MALAT1-ceRNA axis.

6. Conclusion

In summary, LncRNA MALAT1 plays a pivotal role in the initiation and progression of osteoarthritis (OA). Its upregulation is not an isolated event; rather, by acting as a competing endogenous RNA (ceRNA) that sponges specific microRNAs such as miR-140 and miR-146a, MALAT1 constructs a broad and complex regulatory network. This network acts as a sophisticated molecular hub, tightly interconnecting the three core pathological hallmarks of OA: chondrocyte metabolic imbalance, chronic synovial inflammation, and abnormal subchondral bone remodeling. Specifically, the MALAT1-ceRNA axis profoundly influences OA pathogenesis by finely regulating a series of key effector molecules and their associated signaling pathways, including ADAMTS5 and MMP13 (affecting extracellular matrix degradation), SOX9 (influencing chondrocyte differentiation and

homeostasis), NF-κB (mediating inflammatory responses), and RANKL (regulating bone remodeling). This positions MALAT1 beyond the scope of a single functional molecule, establishing it as a critical intersection point for understanding the multi-factorial, multi-stage pathogenic mechanisms of OA.

From an expert perspective, the discovery of MALAT1 signifies an important paradigm shift in OA research. Traditionally viewed primarily as a "degenerative" disease driven by mechanical wear, with treatments largely focused on symptom relief, research on MALAT1 and its ceRNA network redirects attention towards epigenetic regulation and complex molecular crosstalk. It emphasizes OA as an active disease driven by multiple cellular and molecular events. This perspective helps balance earlier viewpoints that might have overemphasized single factors (such as inflammatory cytokines or matrix-degrading enzymes), offering a more integrated systems biology view. It reveals that beneath the surface of "degeneration" lies an active, targetable dysregulation of molecular processes, laying a theoretical foundation for intervening at the root cause of the disease.

Based on its core mechanisms, targeting MALAT1 or its ceRNA network holds immense translational potential. On one hand, detecting MALAT1 expression levels or its associated ceRNA network molecular profiles in synovial fluid or peripheral blood holds promise as novel biomarkers for early OA diagnosis and monitoring disease activity, potentially complementing existing imaging and clinical assessments. On the other hand, and more importantly, it opens up new avenues for developing true disease-modifying osteoarthritis drugs (DMOADs). By specifically inhibiting MALAT1 function through techniques such as small molecule drugs, antisense oligonucleotides (ASOs), or small interfering RNA (siRNA), it is theoretically possible to simultaneously intervene in multiple pathological processes, which includes the cartilage destruction, inflammation, and abnormal bone remodeling, thus achieving a transition from "symptom management" to "disease-modifying intervention".

However, translating this potential into clinical reality faces significant challenges. Future research urgently requires employing cutting-edge technologies like single-cell sequencing and spatial transcriptomics to precisely map the dynamic spatiotemporal landscape of the MALAT1-ceRNA network across different OA stages and joint tissues (e.g., superficial vs. deep chondrocytes, synovial fibroblast subpopulations, bone cells). This would clarify the most critical intervention windows and cellular targets. Furthermore, achieving efficient, specific delivery of therapeutic agents to diseased joint tissues while ensuring long-term safety and efficacy remains a major obstacle between basic research and clinical application. Overcoming these challenges necessitates deep integration of materials science, pharmaceutical science, clinical medicine, and fundamental research.

In conclusion, LncRNA MALAT1 serves as a key ceRNA regulatory hub within the OA pathological network. Its study not only deepens our understanding of the molecular mechanisms underlying OA but also points towards new directions for diagnosis and treatment. Future efforts should focus on refining the precision of mechanistic insights and vigorously tackling the technical bottlenecks in clinical translation, potentially leading to revolutionary therapeutic options for OA patients.

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