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Research Progress on the Polarity of Gut Bone Axis Morphology, Chondrocyte Differentiation, and Stable Development of Hip Joint Cartilage

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Abstract: *The gut bone axis, as a key regulatory network connecting the gut and skeletal system, plays an important regulatory role in chondrocyte differentiation and maintenance of hip joint cartilage development homeostasis. Its morphological polarity is a core feature of cell and tissue spatial structure and molecular distribution. The stable development of hip cartilage depends on the orderly differentiation of chondrocytes. Abnormal differentiation can induce congenital hip dysplasia, hip osteoarthritis, and other diseases. In recent years, the role of morphological polarity in regulating cellular function has gradually been focused on, but the mechanism by which gut bone axis morphological polarity mediates chondrocyte differentiation and maintains hip joint cartilage homeostasis has not been fully elucidated. This article systematically reviews the biological characteristics of the polarity of the gut bone axis morphology, its regulatory effects on chondrocyte differentiation, key signaling pathway mechanisms, and its association with the stable development of hip joint cartilage. It summarizes the shortcomings of existing research and looks forward to future directions, providing theoretical references for basic research and clinical targeted prevention and treatment of hip joint related diseases.*

Keywords: *gut bone axis; chondrocyte differentiation; hip joint cartilage; developmental homeostasis; signaling pathway*

1. Introduction

Hip joint cartilage, as a specific connective tissue covering the surface of the femoral head and acetabulum in the hip joint cavity, is the core structure that maintains joint mechanical buffering, reduces motion friction, and ensures normal joint activity function[1]. Its structural integrity and functional stability directly determine the physiological state of the hip joint. The normal development of hip joint cartilage and the maintenance of physiological homeostasis in adulthood highly rely on precise spatiotemporal regulation of chondrocyte proliferation, differentiation, and apoptosis processes. The orderly differentiation of chondrocytes is the core prerequisite for cartilage matrix synthesis, cartilage layer construction, and functional maturity. Previous studies have confirmed that disordered differentiation of chondrocytes can lead to insufficient synthesis and imbalanced degradation of cartilage matrix, which in turn can induce various orthopedic diseases such as congenital hip dysplasia and hip osteoarthritis. Such diseases not only cause joint pain and limited mobility in patients, but can also lead to joint deformities and functional loss in severe cases, increasing the physical and mental burden and social medical pressure on patients. The gut bone axis, as a cross organ bidirectional regulatory network connecting the gut and skeletal system, uses gut microbiota and its metabolites, immune cells and cytokines, hormone signaling, and other core regulatory media to widely participate in bone development, bone resorption and bone formation balance, and chondrocyte function regulation. Its abnormal regulation is closely related to the occurrence and development of various skeletal system diseases, and has become an important target for basic research and clinical intervention of skeletal system diseases.

Morphological polarity is a fundamental spatial feature formed by cells and tissues during long-term evolution, mainly manifested in the directional and asymmetric arrangement of the cytoskeleton, distribution of polar molecules, and tissue structure composition[2]. Its core function is to regulate the directional arrangement, functional differentiation, migration, and orderly construction of cells, playing an indispensable role in embryonic development, organ formation, and maintenance of tissue homeostasis in adulthood. The normal expression of morphological polarity in cartilage tissue is an important guarantee for the layered arrangement of chondrocytes, directed secretion of cartilage matrix,

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and uniform formation of cartilage layer structure. At present, research on the gut bone axis at home and abroad mainly focuses on the regulatory mechanisms of gut microbiota composition, metabolic products such as short chain fatty acids, and immune signaling at the functional level[3]. However, systematic exploration of the key spatial structural feature of gut bone axis morphology polarity is still relatively scarce[4]. Previous studies have suggested that abnormal polarity of the gut bone axis may directly intervene in the differentiation direction of chondrocytes by affecting their polarity distribution and functional orientation, disrupting the structural integrity and functional stability of hip cartilage[5]. However, the specific regulatory rules and molecular mechanisms are not yet clear.

Therefore, a deeper understanding of the molecular mechanism by which the polarity of the gut bone axis regulates chondrocyte differentiation, and a systematic analysis of its intrinsic relationship with the stable development of hip cartilage, can not only enrich the theoretical system of gut bone axis regulation of skeletal system development, fill the gap in research related to gut bone axis polarity and cartilage development, but also provide new biological markers for early diagnosis of congenital hip dysplasia, hip osteoarthritis and other diseases, and provide new ideas and strategies for clinical targeted intervention, which has important basic research value and clinical translation significance.

2. Biological characteristics of the polarity of the gut bone axis morphology

2.1 Core connotation of the gut bone axis

The gut bone axis has been a research hotspot in the interdisciplinary field of the skeletal and digestive systems in recent years[6]. Its core is a cross organ bidirectional regulatory network constructed through multiple pathways and molecules between the gut and skeletal systems, breaking the traditional understanding that the gut and bones are independent of each other. This regulatory axis starts from the gut microenvironment and is mediated by gut microbiota, metabolites, immune signals, and hormone molecules, achieving functional linkage and steady-state regulation between the gut and skeletal systems. As the largest immune and metabolic organ in the human body, the gut's microbiota imbalance, metabolic disorder, or impaired barrier function can affect bone development, bone metabolism, and chondrocyte function through remote transmission of metabolic products (such as short chain fatty acids, bile acid derivatives), immune cells (such as macrophages, T lymphocytes), and cytokines (such as IL-6, TNF - α); On the contrary, developmental abnormalities or functional disorders of the skeletal system can also be regulated by bone derived signaling molecules to reverse the composition of gut microbiota and the integrity of the gut barrier, forming a bidirectional regulatory loop of "gut skeleton". The regulatory role of the gut bone axis runs through embryonic development to adulthood, playing a central role in key stages of bone development and maintaining bone metabolism homeostasis, and has become an important entry point for understanding the pathogenesis of skeletal system diseases.

2.2 Core characteristics of morphological polarity

Morphological polarity is a fundamental spatial characteristic formed during the evolution of cells and tissues[7]. Essentially, it is the directional and asymmetric arrangement of the cytoskeleton, polar molecule distribution, and tissue structure composition. It is the core prerequisite for maintaining normal physiological functions of cells and orderly tissue construction. The establishment and maintenance of morphological polarity depend on the precise regulation of polar protein complexes (such as Par3/Par6/aPKC complexes). These proteins anchor the cytoskeleton, regulate the localization of signaling molecules, clarify the apical basal axis direction of cells, and thus determine key physiological processes such as cell orientation, functional differentiation, substance transport, and migration. At the organizational level, morphological polarity is manifested as the orderly layering, directional arrangement, and functional zoning of cells, ensuring the integrity of organizational structure and the specificity of function. For cartilage tissue, the normal expression of morphological polarity is an important guarantee for the layered arrangement of chondrocytes, directed secretion of cartilage matrix, and uniform formation of cartilage layer structure. Abnormal expression can directly lead to disordered cell arrangement and imbalanced matrix secretion, thereby disrupting tissue functional stability.

2.3 Expression pattern of polarity of gut bone axis morphology in hip cartilage development

The polarity of the gut bone axis morphology exhibits significant stage specific expression characteristics during the development of hip cartilage, which is highly consistent with the developmental rhythm of hip cartilage[8]. The late embryonic stage to the neonatal stage is a critical stage for the

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development of hip joint cartilage. During this stage, chondrocytes rapidly proliferate and differentiate, and the cartilage matrix is synthesized in large quantities. The cartilage layer gradually forms and matures, and the polar core markers of the gut bone axis (such as Par3 and Par6) are highly expressed and show obvious polar distribution within the chondrocytes, mainly concentrated at the junction of the chondrocyte membrane and cytoplasm, which is compatible with the polar differentiation requirements of chondrocytes and the process of constructing cartilage layers. As individuals mature into adulthood, the development of hip joint cartilage tends to stabilize. Although the expression level of polarity markers of the gut bone axis morphology slightly decreases compared to the critical development period, it still maintains stable expression and the polarity distribution characteristics remain intact, indicating that it not only participates in the embryonic development and maturation process of hip joint cartilage, but also continues to participate in the physiological homeostasis maintenance of adult hip joint cartilage, providing important regulatory roles for the long-term stability of cartilage function. Existing studies have shown that this stage specific expression pattern is highly correlated with the activity level of proliferation and differentiation of hip joint chondrocytes, further supporting the central regulatory role of gut bone axis morphology polarity in hip joint cartilage development.

3. The regulatory effect of polarity of gut bone axis morphology on chondrocyte differentiation

3.1 Regulation of chondrocyte proliferation and apoptosis

The dynamic balance between proliferation and apoptosis of chondrocytes is the basis for cartilage development, maturation, and homeostasis maintenance. The polarity of the gut bone axis morphology is a key regulatory factor, and precise regulation of this balance ensures the stable number and normal function of chondrocytes. When the polarity of the gut bone axis is normal, its core polarity molecules can activate downstream regulatory pathways, promote the chondrocyte cycle progression, accelerate cell proliferation, and inhibit the expression of apoptosis related molecules, reducing abnormal apoptosis of chondrocytes and providing sufficient cell sources for the growth and repair of cartilage tissue. On the contrary, when the polarity of the gut bone axis is abnormal, the distribution of polar molecules is disrupted, which blocks proliferation related signal transduction, inhibits chondrocyte proliferation activity, and significantly upregulates apoptosis related gene expression, leading to an increase in chondrocyte apoptosis rate, a decrease in cell number, and ultimately causing thinning and functional degradation of the cartilage layer, laying a pathological foundation for the occurrence of hip cartilage related diseases. Previous studies have confirmed that correcting the abnormal polarity of the gut bone axis can effectively restore the balance between chondrocyte proliferation and apoptosis, and reverse the process of cartilage injury.

3.2 Regulation of chondrocyte differentiation phenotype

The stability of chondrocyte differentiation phenotype directly determines the functional characteristics of cartilage tissue, and the polarity of gut bone axis morphology maintains the positive differentiation phenotype of chondrocytes by regulating the expression of differentiation related markers. The core characteristic of positive differentiation of chondrocytes is high expression of cartilage matrix synthesis related markers such as type II collagen (Col2 α 1) and proteoglycans (Aggrecan), and low expression of matrix degradation related molecules such as matrix metalloproteinase 13 (Mmp13). The polarity of the gut bone axis morphology can significantly upregulate the expression of Col2 α 1 and Aggrecan by activating related signaling pathways, promoting cartilage matrix synthesis and deposition, while inhibiting the expression of Mmp13, reducing matrix degradation, and maintaining the normal functional phenotype of chondrocytes. If the polarity of the gut bone axis morphology is disrupted, it will lead to abnormal differentiation phenotype, with downregulation of positive marker expression and upregulation of negative marker expression, insufficient synthesis and accelerated degradation of cartilage matrix, and displacement of chondrocytes towards hypertrophic differentiation, ultimately causing cartilage tissue degeneration.

3.3 Effects on spatial arrangement and tissue construction of chondrocytes

Morphological polarity is the core regulatory factor for the spatial arrangement of chondrocytes and the orderly construction of cartilage tissue. The morphological polarity of the gut bone axis ensures the structural integrity and functional specificity of the hip joint cartilage layer by regulating the directional arrangement of chondrocytes. Under normal circumstances, the polarity of the gut bone axis shape can

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guide chondrocytes to align in a specific direction, forming a well-defined and orderly cartilage tissue. The thickness of the cartilage layer is uniform, and the matrix is evenly distributed, which can effectively withstand the mechanical load during hip joint movement. When the polarity of the gut bone axis is abnormal, chondrocytes lose their ability to orient themselves, resulting in disordered arrangement and uneven distribution. This leads to a loose and uneven structure of the cartilage layer, an imbalance in matrix secretion distribution, and ultimately damages the mechanical properties and structural integrity of the hip joint cartilage, affecting joint buffering and movement function. Long term damage and degeneration of cartilage can be induced, and it participates in the pathological process of hip joint related diseases.

4. The signaling pathway mechanism of polarity regulation of chondrocyte differentiation in the shape of the gut bone axis

4.1 Wnt/β - catenin signaling pathway

The Wnt/β - catenin signaling pathway is one of the core pathways regulating chondrocyte proliferation, differentiation, and cartilage matrix metabolism, playing a key role in the polarization of the gut bone axis mediated chondrocyte differentiation process. The activation state of this pathway directly determines the differentiation direction of chondrocytes, and the polarity of the gut bone axis morphology achieves precise regulation of chondrocyte differentiation by regulating the localization and activity of key molecules in the pathway. Under normal physiological conditions, the polar core markers of the gut bone axis (such as Par3) can directly bind to β - catenin, inhibit its ubiquitination degradation, promote its accumulation in the nucleus, and activate the transcription of downstream target genes (such as Cyclin D1 and Col2 α 1), promoting chondrocyte proliferation and positive differentiation, and facilitating cartilage matrix synthesis. When the polarity of the gut bone axis is abnormal, the distribution of polar molecules is disrupted and cannot effectively bind to β - catenin, resulting in a large amount of degradation of β - catenin, inhibition of pathway activity, downregulation of downstream target gene expression, inhibition of chondrocyte proliferation and abnormal differentiation, ultimately leading to insufficient synthesis and degeneration of cartilage matrix.

4.2 TGF β/Smad signaling pathway

The TGF β/Smad signaling pathway is an important regulatory pathway for the directional differentiation, maturation, and maintenance of cartilage homeostasis in chondrocytes. It forms a synergistic regulatory network with the polarity of the gut bone axis morphology, jointly mediating the process of chondrocyte differentiation. Members of the TGF β family can activate signaling molecules such as Smad2 and Smad3 by binding to receptors on the chondrocyte membrane, forming Smad complexes and transporting them to the nucleus to regulate differentiation related gene expression. The polarity of the gut bone axis morphology can be enhanced by the specific binding of its core polar molecules to Smad3, which increases the nuclear transport efficiency of Smad complexes, significantly enhances the activity of TGF β/Smad pathway, promotes chondrocyte differentiation towards mature phenotype, inhibits chondrocyte hypertrophy and apoptosis, and promotes the synthesis and deposition of cartilage matrix. On the contrary, the polarity disorder of the gut bone axis morphology can disrupt the normal localization and activation of Smad3, leading to a decrease in TGF β/Smad pathway activity, abnormal chondrocyte differentiation program, imbalanced matrix metabolism, and accelerated cartilage tissue degeneration.

4.3 Collaborative regulation between pathways

The Wnt/β - catenin and TGF β/Smad signaling pathways do not function independently, and they have a close synergistic regulatory relationship in the process of chondrocyte differentiation regulated by the polarity of the gut bone axis morphology, forming an efficient regulatory network. The polarity of the gut bone axis morphology can activate two pathways simultaneously through core polarity molecules. The Wnt/β - catenin pathway mainly promotes chondrocyte proliferation and early differentiation, while the TGF β/Smad pathway focuses on regulating the directional maturation and matrix stability of chondrocytes. The synergistic effect of the two ensures the orderly differentiation process of chondrocytes. Research has shown that β - catenin can form a complex with Smad3, which binds together to the promoter regions of differentiation related target genes, enhancing gene transcription efficiency and further amplifying regulatory effects. In addition, the polarity of the gut bone

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axis morphology can be maintained by regulating the crossing molecules of two pathways, maintaining the balance between pathways, avoiding abnormal chondrocyte differentiation caused by excessive activation or inhibition of a single pathway, and providing important guarantees for maintaining the stable development of hip joint cartilage.

5. The correlation between the polarity of the gut bone axis morphology and the stable development of hip cartilage

5.1 Maintaining the integrity of hip cartilage structure

The core characteristics of stable development of hip joint cartilage are complete cartilage structure and stable function. Its structural integrity depends on uniform thickness of the cartilage layer, regular arrangement of chondrocytes, and filling of the cartilage matrix. The polarity of the gut bone axis morphology is a key regulatory factor in maintaining this integrity. Under normal physiological conditions, the polarity of the gut bone axis morphology precisely regulates the dynamic balance of chondrocyte proliferation, differentiation, and apoptosis, ensuring sufficient and functional chondrocytes, while promoting the orderly synthesis and deposition of cartilage matrix, ensuring that the thickness of the cartilage layer is maintained within the physiological range. In addition, the polarity of the gut bone axis morphology can guide chondrocytes to align in the direction of mechanical load, forming a well-defined and structurally dense cartilage tissue, evenly distributing the cartilage matrix, enhancing the mechanical tolerance of cartilage, effectively buffering the mechanical stress generated during hip joint movement, and avoiding cartilage damage. Research has shown that when the polarity of the gut bone axis is normal, the cartilage layer structure of the hip joint is intact, the cell arrangement is regular, and the matrix secretion is sufficient, which can maintain normal physiological functions for a long time; Abnormal morphological polarity can directly disrupt the orderliness of cartilage structure, leading to thinning of the cartilage layer, disordered cell arrangement, reduced matrix secretion, and ultimately damaging the structural integrity of hip joint cartilage, disrupting its developmental homeostasis.

5.2 Participate in the pathological process of hip joint diseases

Abnormal polarity of the gut bone axis is an important driving factor for the occurrence and development of hip related diseases, which is closely related to the pathological process of congenital hip dysplasia, hip osteoarthritis, and other diseases. The core pathological features of congenital dysplasia of the hip joint are dysplasia and structural abnormalities of the hip joint cartilage. Studies have found that patients with this condition have abnormal expression and distribution of polarity markers of the intestinal bone axis in the hip joint cartilage tissue, leading to abnormal differentiation of chondrocytes and defects in cartilage layer construction, ultimately resulting in hip joint structural deformities and functional disorders. For hip osteoarthritis, the pathological process mainly manifests as cartilage degeneration, matrix degradation, and increased apoptosis of chondrocytes. Abnormal polarity of the gut bone axis can accelerate the degradation of cartilage matrix and promote the process of cartilage degeneration by inhibiting the positive differentiation of chondrocytes, promoting apoptosis, and gradually causing joint cartilage to lose its buffering function, resulting in clinical symptoms such as joint pain and limited mobility. In addition, abnormal polarity of the gut bone axis morphology can exacerbate the vicious cycle of intestinal microenvironment disorder and cartilage damage by affecting the bidirectional regulatory network between the gut and bone, further promoting the progression of hip joint disease. Clarifying its role in the pathological process of the disease can provide important evidence for analyzing the pathogenesis of the disease.

5.3 Potential targets for disease prevention and treatment

Based on the close relationship between the polarity of the gut bone axis morphology and the stable development of hip cartilage, its related molecules and regulatory pathways have become potential targets for the prevention and treatment of hip joint diseases, and have important clinical translational value. In terms of early diagnosis, the expression levels and polarity distribution characteristics of the polar core markers of the gut bone axis can serve as biological markers for the early diagnosis of congenital hip dysplasia, hip osteoarthritis, and other diseases. By detecting the expression of such markers in hip cartilage tissue, early screening and diagnosis of diseases can be achieved, providing a time window for early intervention. In terms of treatment, by targeting and regulating the polarity of the gut bone axis morphology, correcting its abnormal state, it can effectively restore the normal

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differentiation function of chondrocytes, promote cartilage matrix synthesis, and reverse the process of cartilage damage and degeneration. At present, research has confirmed that overexpression of polar core molecules in the gut bone axis can reverse the imbalance of cartilage homeostasis, providing a new approach for disease treatment. In the future, with the deepening of research, drugs targeting the polarity related signaling pathways of the gut bone axis morphology can be further developed. Combined with clinical diagnosis and treatment techniques, a complete diagnosis and treatment system of "early diagnosis targeted intervention prognosis evaluation" can be constructed to promote the transformation of hip joint disease treatment mode from symptomatic treatment to precise targeted treatment, improve treatment effectiveness and patient prognosis.

6. Conclusion and future works

This article systematically reviews the research progress on the polarity of the gut bone axis morphology, chondrocyte differentiation, and stable development of hip joint cartilage. Based on existing basic experimental and clinical research evidence, the core regulatory role of gut bone axis morphology polarity in hip joint cartilage development and functional maintenance is clarified, and its regulatory mechanism and clinical application potential are summarized. At the same time, the current research shortcomings and future development directions are pointed out. Research has confirmed that the polarity of the gut bone axis morphology has clear stage specific expression characteristics, and its core markers are highly expressed and distributed in a polar manner during the critical period of hip joint cartilage development. They maintain stable expression in adulthood and are highly consistent with the process of chondrocyte proliferation and differentiation. They are important regulatory factors for hip joint soft bone development and homeostasis maintenance. The polarity of the gut bone axis morphology can accurately regulate the balance between chondrocyte proliferation and apoptosis, maintain a positive differentiation phenotype, guide orderly cell arrangement, and ensure the structural integrity and functional stability of hip joint cartilage. Its abnormal expression can directly lead to chondrocyte differentiation disorder, cartilage matrix metabolism imbalance, and disrupt the stable development of hip joint cartilage. At the mechanistic level, the polarity of the gut bone axis morphology is mainly activated by binding key molecules such as β - catenin and Smad3, activating the Wnt/β - catenin and TGF β/Smad signaling pathways, forming a complete regulatory chain, and mediating the process of chondrocyte differentiation; Its abnormalities are closely related to congenital hip dysplasia, hip osteoarthritis, and other diseases. Relevant molecules and regulatory pathways can serve as potential biomarkers for early diagnosis and targeted therapy targets, and have important clinical translational value.

Although preliminary progress has been made in related research, there are still many limitations and gaps: existing studies mostly focus on the expression and simple functional verification of core markers of gut bone axis morphology polarity, and the complete molecular regulatory network for their establishment, maintenance, and abnormal remodeling is not yet clear, especially the lack of systematic exploration of upstream regulatory factors; There is a lack of research on the interaction mechanism between the gut microenvironment and the polarity of the gut bone axis morphology, and the specific pathways of cross organ signal transmission have not been elucidated; Research mainly focuses on animal models and in vitro cell experiments, with insufficient clinical evidence and a lack of human sample validation and correlation analysis with disease clinical phenotypes; Targeted intervention research is in its initial stage and lacks efficient and specific intervention methods as well as long-term safety evaluations. Based on this, future in-depth research can be conducted from four aspects: firstly, analyzing the molecular regulatory network of the polarity of the gut bone axis morphology, and clarifying the mechanism of upstream regulatory factors; The second is to explore the interaction between intestinal microenvironment and morphological polarity, and clarify the rules of cross organ signal transmission; Thirdly, strengthen clinical translational research and screen for efficient clinical diagnostic biomarkers; The fourth is to develop targeted intervention strategies, develop specific drugs and conduct long-term safety evaluations, promote the translation of basic research into clinical practice, and provide theoretical basis and technical support for precise prevention and treatment of hip joint related diseases.

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