

World Journal of *Stem Cells*

World J Stem Cells 2024 July 26; 16(7): 739-772



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The *WJSC* is now abstracted and indexed in Science Citation Index Expanded (SCIE, also known as SciSearch®), Journal Citation Reports/Science Edition, PubMed, PubMed Central, Scopus, Biological Abstracts, BIOSIS Previews, Reference Citation Analysis, China Science and Technology Journal Database, and Superstar Journals Database. The 2024 Edition of Journal Citation Reports® cites the 2023 journal impact factor (JIF) for *WJSC* as 3.6; JIF without journal self cites: 3.5; 5-year JIF: 4.2; JIF Rank: 105/205 in cell biology; JIF Quartile: Q3; and 5-year JIF Quartile: Q2. The *WJSC*'s CiteScore for 2023 is 7.8 and Scopus CiteScore rank 2023: Histology is 11/62; Genetics is 78/347; Genetics (clinical) is 19/99; Molecular Biology is 131/410; Cell Biology is 104/285.

RESPONSIBLE EDITORS FOR THIS ISSUE

Production Editor: *Xiang-Di Zhang*; Production Department Director: *Xu Guo*; Cover Editor: *Jia-Ru Fan*.

NAME OF JOURNAL

World Journal of Stem Cells

ISSN

ISSN 1948-0210 (online)

LAUNCH DATE

December 31, 2009

FREQUENCY

Monthly

EDITORS-IN-CHIEF

Shengwen Calvin Li, Carlo Ventura

EDITORIAL BOARD MEMBERS

<https://www.wjgnet.com/1948-0210/editorialboard.htm>

PUBLICATION DATE

July 26, 2024

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INSTRUCTIONS TO AUTHORS

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ARTICLE PROCESSING CHARGE

<https://www.wjgnet.com/bpg/gerinfo/242>

STEPS FOR SUBMITTING MANUSCRIPTS

<https://www.wjgnet.com/bpg/GerInfo/239>

ONLINE SUBMISSION

<https://www.f6publishing.com>

Additional comments on extracellular vesicles derived from mesenchymal stem cells mediate extracellular matrix remodeling in osteoarthritis

Hang Pei, Yi Zhang, Chao Wang, Bang-Jian He

Specialty type: Cell and tissue engineering

Provenance and peer review: Invited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's classification

Scientific Quality: Grade C, Grade C

Novelty: Grade B, Grade B

Creativity or Innovation: Grade B, Grade B

Scientific Significance: Grade B, Grade B

P-Reviewer: Gallone A; Wong CY

Received: March 22, 2024

Revised: June 3, 2024

Accepted: June 27, 2024

Published online: July 26, 2024

Processing time: 124 Days and 21.4 Hours



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Abstract

Recently, we read an article published by the Yang *et al.* The results of this study indicated that engineered exosomes loaded with microRNA-29a (miR-29a) alleviate knee inflammation and maintain extracellular matrix stability in Sprague Dawley rats. The study's results provide useful information for treating knee osteoarthritis (KOA). This letter, shares our perspectives on treating KOA using engineered exosomes for miR-29a.

Key Words: Exosomes; Intra-articular injection; Mesenchymal stem cells; MicroRNA-29a; Osteoarthritis

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Core Tip: We recently reviewed a study that treated knee osteoarthritis using engineered exosomes. The authors found that this novel treatment approach is more effective than standard exosomes at symptom alleviation. This letter summarizes the advantages and limitations of this therapy and clarifies aspects of the original article.

Citation: Pei H, Zhang Y, Wang C, He BJ. Additional comments on extracellular vesicles derived from mesenchymal stem cells mediate extracellular matrix remodeling in osteoarthritis. *World J Stem Cells* 2024; 16(7): 739-741

URL: <https://www.wjgnet.com/1948-0210/full/v16/i7/739.htm>

DOI: <https://dx.doi.org/10.4252/wjsc.v16.i7.739>

INTRODUCTION

We recently read an article titled “Extracellular vesicles derived from mesenchymal stem cells mediate extracellular matrix remodeling in osteoarthritis through the transport of microRNA-29a”. In this study, Yang *et al*[1] used engineered exosomes loaded with microRNA-29a (miR-29a) to treat early knee osteoarthritis (KOA) in rats. The study demonstrated superior pain reduction and joint improvement compared to standard exosomes. It provided a glimmer of hope for novel and exciting approach for treating KOA without side effects. We hope this piques your curiosity and encourages further exploration of this fascinating topic.

MSCS IN OSTEOARTHRITIS

OA is a severe degenerative joint disease characterized by articular cartilage degeneration due to the loss of extracellular matrix, extensive fibrosis, and fissure formation, ultimately leading to the complete loss of the cartilage surface[2]; current first-line drugs for the treatment of OA improve symptoms by suppressing inflammation (using nonsteroidal anti-inflammatory drugs) and lubricating and nourishing the cartilage (using hyaluronic acid); however, these treatments are ineffective at halting cartilage degeneration.

In recent years, the emergence of biologics has provided new insights into the treatment of OA. Our previous studies have identified the excellent potential of platelet-rich plasma and fat microfragments for treating OA[3,4]. Mesenchymal stem cells (MSCs) have garnered significant attention as a promising therapy for delaying or controlling arthritis due to their ability to differentiate into various cell types and their impact on tissue repair and immunomodulation[5]. The paracrine regulatory function of MSCs plays a crucial role in promoting cartilage repair. Jia *et al*[6] discovered that undifferentiated MSCs were more effective in regenerating hyaline cartilage than *in vitro* preformed chondrogenic differentiated MSCs. Exosomes, which are secreted by MSCs, have been widely studied for their role in paracrine regulation. Qian *et al*[7] found that exosomal miR-26b-5p can transform pro-inflammatory M1 macrophages into anti-inflammatory M2 types by targeting the toll-like receptor 3 signaling pathway to alleviate OA. Additionally, Qiu *et al*[8] found that exosomal miR-485-3p can alleviate cartilage damage in OA by targeting the NRP1-mediated PI3K/Akt pathway. Furthermore, researchers integrated the less commonly used miR-29a into exosomes, presenting a new approach for treating OA.

CONCLUSION

The use of stem cell exosome miR-29a has produced promising results in reducing the symptoms of OA and promoting cartilage repair and joint function recovery. However, like any new treatment, certain limitations need to be addressed. For example, the mechanism of action of miR-29a requires further research. Similarly, stem cell exosome preparation and application methods need to be optimized. This highlights the importance of additional research to confirm and build upon the results of the discussed study.

ACKNOWLEDGEMENTS

We would like to thank Hang Pei, Yi Zhang, Chao Wang, and Bang-Jian He for their valuable contributions to this editorial.

FOOTNOTES

Author contributions: Pei H and Zhang Y contributed to this manuscript equally. Pei H was responsible for the conception and design of the editorial; Zhang Y and Wang C contributed to the drafting and revising of the manuscript; He BJ provided critical revisions and gave final approval of the version to be published. All authors read and approved the final manuscript.

Supported by National Natural Science Foundation of China, No. 82074469.

Conflict-of-interest statement: All the authors report no relevant conflicts of interest for this article.

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S-Editor: Wang JJ

L-Editor: A

P-Editor: Zhang L

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