ISSN 2618-1584 Vol. 6, Issue 12: 37-43, DOI: 10.25236/FMSR.2024.061206

Effect of mangiferin on anti-aging of skeletal muscle and its influence on glycolysis

Hanjie Jin[1,2,a], Haojie Tang[1,2,b], Chaoyue Zhao[1,2,c], Xuting Zhu[1,2,d], Jinhua Wang[1,e], Zhikun Bai[1,3,f,*]

[1]School of Basic Medical Sciences, Youjiang Medical University for Nationalities, Baise, China
[2]Graduate School, Youjiang Medical University for Nationalities, Baise, China
[3]Guangxi Database Construction and Application Engineering Research Center for Intracorporal Pharmacochemistry of TCM, Youjiang Medical University for Nationalities, Baise, China
[a]810566517@qq.com, [b]1017574040@qq.com, [c]136374466@qq.com, [d]3358462191@qq.com, [e]28516758@qq.com, [f]bai.zhikun@ymun.edu.cn
[*]Corresponding author

Abstract: *This review aims to explore the potential role of Mangiferin, a natural bioactive compound, in skeletal muscle anti-aging, with a particular focus on its effects on glycolysis. As aging progresses, skeletal muscle mass and function decline, leading to reduced mobility and quality of life. Recently, Mangiferin has garnered attention for its antioxidant, anti-inflammatory, and metabolic regulatory properties. This article will summarize the bioactivity of Mangiferin, its protective mechanisms in skeletal muscle, its role in the anti-aging process, and its impact on glycolysis, providing a theoretical foundation and practical guidance for future research.*

Keywords: *Mangiferin, skeletal muscle, anti-aging, glycolysis, natural active ingredient*

1. Introduction

Skeletal muscle aging is a significant concern for public health, particularly as populations around the world continue to age. The decline in skeletal muscle mass and function, also known as sarcopenia, contributes to frailty and various health issues, including metabolic diseases and decreased quality of life in older adults[1]. This age-related muscle deterioration is characterized by a loss of muscle fibers, changes in muscle composition, and a reduced capacity for regeneration[2]. As the global population aged over 60 is expected to reach 1.4 billion by 2030, understanding the mechanisms behind skeletal muscle aging is critical for developing interventions to improve healthspan alongside lifespan[2].

Glycolysis plays a pivotal role in energy metabolism, particularly in skeletal muscle, where it provides the necessary ATP for muscle contraction and recovery[3]. In aging muscles, glycolytic pathways may become dysregulated, leading to impaired energy production and contributing to muscle weakness and atrophy[4]. This metabolic shift towards reduced glycolytic activity can exacerbate the effects of aging on muscle function, emphasizing the need for research into how to maintain or restore glycolytic efficiency in older adults.

Mangiferin, a bioactive compound derived from Mangifera indica (mango), has garnered attention for its potential health benefits, including antioxidant, anti-inflammatory, and muscle-protective properties[5]. Recent studies have indicated that mangiferin may enhance energy metabolism and promote muscle health, making it a promising candidate for addressing age-related muscle decline[6]. Given its diverse biological activities and potential to influence metabolic pathways, investigating the effects of mangiferin on skeletal muscle aging and glycolysis is of significant importance.

The purpose of this review is to explore the relationship between skeletal muscle aging, energy metabolism with a focus on glycolysis, and the therapeutic potential of mangiferin. By synthesizing current knowledge and identifying gaps in research, we aim to highlight the significance of these areas for developing effective strategies to combat sarcopenia and improve the health of aging populations.

ISSN 2618-1584 Vol. 6, Issue 12: 37-43, DOI: 10.25236/FMSR.2024.061206

2. Effect of mangiferin on anti-aging of skeletal muscle

2.1 Chemical Structure and Sources of Mangiferin

2.1.1 Chemical Properties of Mangiferin

Mangiferin, a naturally occurring glucosylxanthone, is predominantly found in the mango plant (Mangifera indica) and exhibits a complex chemical structure characterized by its xanthone core and multiple hydroxyl groups. This molecular configuration contributes to its diverse biological activities, including antioxidant, anti-inflammatory, and anticancer properties. The structural formula of mangiferin is $C_{17}H_{18}O_{11}$, featuring a unique arrangement that enhances its solubility and bioavailability. However, its low aqueous solubility poses significant challenges for therapeutic applications. Recent studies have focused on enhancing the solubility of mangiferin through glycosylation, which has shown to significantly increase its water solubility and bioactivity. For instance, the transglycosylation of mangiferin using cyclodextrin glycosyltransferase resulted in derivatives with solubility improved by over 5,000 times compared to the parent compound, while also retaining or enhancing its antioxidant capacity[7]. Such modifications not only improve the pharmacokinetic properties of mangiferin but also expand its potential applications in both food and pharmaceutical industries.

2.1.2 Natural Sources and Extraction Methods of Mangiferin

Mangiferin is primarily extracted from the bark, leaves, and fruits of Mangifera indica, as well as from other plants in the Anacardiaceae family. Traditional extraction methods often involve solvent extraction, which can be inefficient and time-consuming. Recent advancements have introduced more effective techniques such as high-speed counter-current chromatography (HSCCC) and supercritical antisolvent (SAS) methods, which enhance the yield and purity of mangiferin. For instance, HSCCC has been utilized to isolate mangiferin glycosides with purities exceeding 99%, demonstrating its efficacy in purifying bioactive compounds from complex mixtures[8]. Moreover, enzymatic methods employing recombinant maltogenic amylase have been shown to facilitate the glycosylation of mangiferin, yielding derivatives with significantly enhanced solubility and biological activity[9]. These innovative extraction and modification techniques not only improve the availability of mangiferin but also pave the way for its incorporation into various therapeutic and nutraceutical formulations, highlighting its significance as a valuable bioactive compound in modern medicine.

2.2 Bioactivity of Mangiferin

2.2.1 Antioxidant Activity

Mangiferin, a polyphenolic xanthone glycoside primarily derived from mango (Mangifera indica L.) leaves, exhibits significant antioxidant properties, which are crucial for mitigating oxidative stress and related diseases. Various studies have demonstrated that mangiferin can scavenge free radicals, thereby preventing cellular damage caused by oxidative stress. For instance, the antioxidant activity of mangiferin has been evaluated using several methods, including DPPH and ABTS assays, which measure the ability of a compound to neutralize free radicals. The results indicate that mangiferin's structure, particularly the presence of multiple hydroxyl groups, enhances its radical scavenging capacity, making it a potent natural antioxidant. Additionally, mangiferin has been shown to upregulate endogenous antioxidant enzymes, such as superoxide dismutase and catalase, further supporting its role in cellular defense mechanisms against oxidative damage[5]. Furthermore, its ability to modulate oxidative stress pathways suggests potential therapeutic applications in conditions associated with oxidative stress, such as neurodegenerative diseases and cardiovascular disorders.

2.2.2 Anti-inflammatory Activity

The anti-inflammatory properties of mangiferin are well-documented and contribute to its therapeutic potential. Mangiferin has been shown to inhibit the production of pro-inflammatory cytokines and mediators such as TNF-α, IL-1β, and nitric oxide in various cell models, indicating its capability to modulate inflammatory responses. For example, studies have revealed that mangiferin can suppress the activation of NF-κB, a key transcription factor involved in inflammatory processes, thereby reducing the expression of inflammatory genes[10]. Additionally, mangiferin's action on macrophages has demonstrated its efficacy in attenuating inflammation by inhibiting the expression of COX-2 and iNOS, which are crucial for the inflammatory response. The mechanisms underlying these effects include the modulation of signaling pathways that regulate inflammation, such as MAPK and PI3K/Akt pathways. This multifaceted approach to controlling inflammation highlights mangiferin's

ISSN 2618-1584 Vol. 6, Issue 12: 37-43, DOI: 10.25236/FMSR.2024.061206

potential as a therapeutic agent in treating inflammatory diseases, including arthritis and cardiovascular diseases[11].

2.2.3 Metabolic Regulation

Mangiferin also plays a significant role in metabolic regulation, particularly in glucose metabolism and lipid homeostasis. Research indicates that mangiferin can enhance insulin sensitivity and improve glucose uptake in skeletal muscle cells, which is beneficial for managing conditions such as diabetes. The underlying mechanism involves the activation of the AMPK pathway, which is crucial for energy homeostasis and metabolic regulation[5]. Furthermore, mangiferin has been reported to exert lipid-lowering effects by modulating lipid metabolism pathways, reducing triglyceride accumulation in the liver, and promoting fatty acid oxidation. This dual action of mangiferin in regulating both glucose and lipid metabolism positions it as a promising candidate for developing functional foods and nutraceuticals aimed at preventing and managing metabolic disorders such as obesity and type 2 diabetes[12]. The ongoing research into the metabolic effects of mangiferin emphasizes its potential utility in clinical settings, particularly in the context of metabolic syndrome and related diseases.

2.3 Biological Mechanisms of Skeletal Muscle Aging

Skeletal muscle aging, often referred to as sarcopenia, is characterized by a progressive decline in muscle mass and function, significantly impacting the quality of life in older adults. This phenomenon is not merely a consequence of aging but results from a complex interplay of biological mechanisms. The decline in skeletal muscle mass is primarily driven by an imbalance between muscle protein synthesis and degradation, influenced by factors such as hormonal changes, inflammation, and oxidative stress. Mitochondrial dysfunction is another critical aspect, as impaired mitochondrial function leads to decreased energy production and increased oxidative stress, further exacerbating muscle atrophy. Research indicates that the accumulation of pro-inflammatory cytokines, such as TNF-α, plays a significant role in promoting muscle degradation pathways, including the ubiquitin-proteasome system and autophagy, which are vital for muscle protein turnover[13,14]. Additionally, the role of microRNAs in regulating gene expression related to muscle growth and differentiation is emerging as a crucial area of study, offering potential therapeutic targets for mitigating the effects of aging on skeletal muscle[15,16].

2.3.1 Causes of Skeletal Muscle Mass Reduction

The reduction of skeletal muscle mass in aging individuals is a multifaceted issue influenced by various factors. One primary cause is the decrease in physical activity levels, which is often observed as people age. Sedentary lifestyles lead to muscle disuse, contributing to atrophy and loss of muscle fibers, particularly type II fibers, which are essential for strength and power[17]. Furthermore, hormonal changes, including decreased levels of anabolic hormones like testosterone and growth hormone, play a significant role in muscle mass reduction[18]. The presence of chronic low-grade inflammation, often termed "inflammaging," is another critical contributor, as it promotes catabolic processes that lead to muscle breakdown[13]. Additionally, mitochondrial dysfunction, characterized by reduced mitochondrial biogenesis and increased oxidative stress, has been implicated in the pathogenesis of sarcopenia, as it affects energy metabolism and contributes to muscle fiber degeneration[19,20]. Lastly, nutritional deficiencies, particularly in protein intake and essential micronutrients, can exacerbate muscle loss, highlighting the importance of dietary interventions in combating sarcopenia[21].

2.3.2 Factors Influencing Skeletal Muscle Dysfunction

Skeletal muscle dysfunction in aging is influenced by a myriad of factors that interact in complex ways. One significant factor is the decline in neuromuscular function, which affects the ability of the nervous system to effectively stimulate muscle contraction[22]. This decline is often exacerbated by conditions such as diabetes and cardiovascular diseases, which further impair muscle performance and regeneration. Additionally, the role of systemic inflammation cannot be overlooked; elevated levels of inflammatory markers are associated with muscle wasting and weakness, contributing to a cycle of dysfunction[23]. Oxidative stress, resulting from an imbalance between reactive oxygen species production and antioxidant defenses, significantly impacts muscle health by promoting cellular damage and impairing muscle regeneration[24]. Furthermore, changes in the extracellular matrix and muscle satellite cell function during aging hinder the muscle's ability to repair and regenerate, leading to a decline in muscle mass and strength[24,25]. Understanding these factors is crucial for developing targeted interventions aimed at preserving muscle function and mitigating the impact of aging on skeletal muscle health.

ISSN 2618-1584 Vol. 6, Issue 12: 37-43, DOI: 10.25236/FMSR.2024.061206

2.4 The Protective Effects of Mangiferin on Skeletal Muscle

2.4.1 Promoting Muscle Cell Proliferation and Differentiation

Mangiferin, a polyphenolic compound derived from mangoes, has been shown to exert significant effects on muscle cell proliferation and differentiation. Recent studies indicate that mangiferin can enhance the proliferation of myoblasts, which are critical for muscle regeneration and repair. This is particularly important in the context of muscle injuries, where the regeneration of muscle fibers is necessary for recovery. For instance, research has demonstrated that mangiferin stimulates the expression of key myogenic regulatory factors, such as MyoD and Myogenin, which are essential for myoblast differentiation and muscle fiber formation. Furthermore, mangiferin has been found to activate signaling pathways that promote cell cycle progression, thereby facilitating the transition of myoblasts from a quiescent state to an actively proliferating state. This mechanism not only supports muscle growth but also contributes to the overall health of skeletal muscle by maintaining an appropriate balance between cell proliferation and differentiation, which is crucial for muscle homeostasis and function[26,27].

2.4.2 Reducing Muscle Atrophy and Injury

Mangiferin also demonstrates protective effects against muscle atrophy and injury, conditions that can severely impair muscle function. Muscle atrophy can result from various factors, including disuse, aging, and chronic diseases. Studies have shown that mangiferin can mitigate the effects of oxidative stress, a major contributor to muscle degradation. By acting as an antioxidant, mangiferin reduces the levels of reactive oxygen species (ROS) that can damage muscle fibers and promote atrophy. Additionally, mangiferin has been observed to downregulate the expression of muscle-specific E3 ubiquitin ligases, such as Atrogin-1 and MuRF1, which are involved in the proteolytic degradation of muscle proteins. This action helps to preserve muscle mass and function in conditions that typically lead to muscle wasting. Moreover, in models of muscle injury, mangiferin has been shown to enhance the repair processes by promoting satellite cell activation and proliferation, thereby facilitating muscle regeneration and reducing the extent of damage[28,29].

2.4.3 Improving Muscle Function and Strength

The functional benefits of mangiferin extend to the enhancement of muscle strength and overall performance. Research indicates that mangiferin supplementation can lead to improvements in muscle strength and endurance, making it a potential therapeutic agent for conditions characterized by muscle weakness. The compound has been linked to the increased synthesis of contractile proteins, which are crucial for muscle function. Additionally, mangiferin may enhance mitochondrial biogenesis and function within muscle cells, contributing to improved energy metabolism and endurance capacity. This is particularly relevant for athletes and individuals engaged in physical training, as enhanced muscle function and strength can lead to better performance outcomes. Furthermore, studies suggest that mangiferin can improve neuromuscular transmission, thereby enhancing muscle contraction efficiency. Collectively, these effects position mangiferin as a promising natural compound for supporting muscle health and function, particularly in populations at risk for muscle-related disorders[30,31].

2.5 The Effects of Mangiferin on Glycolysis

2.5.1 Overview of Glycolytic Pathway

Glycolysis is a fundamental metabolic pathway that converts glucose into pyruvate, generating ATP and NADH in the process. This pathway occurs in the cytoplasm and consists of ten enzymatic reactions that can be grouped into two phases: the energy investment phase and the energy payoff phase. In the energy investment phase, two ATP molecules are consumed to phosphorylate glucose and its derivatives. The energy payoff phase produces four ATP molecules, resulting in a net gain of two ATP molecules per glucose molecule. Glycolysis is crucial for cellular energy production and is tightly regulated by various factors, including substrate availability, hormonal signals, and energy demands of the cell. Dysregulation of glycolysis is implicated in numerous metabolic disorders, including obesity, diabetes, and cancer[32].

2.5.2 The Regulatory Role of Mangiferin in Carbohydrate Metabolism

Mangiferin, a polyphenolic compound found in mangoes, has been shown to exert beneficial effects on glucose metabolism. Studies indicate that mangiferin can enhance insulin sensitivity and glucose uptake in skeletal muscle cells, thereby promoting glycolysis. The mechanism underlying this effect

ISSN 2618-1584 Vol. 6, Issue 12: 37-43, DOI: 10.25236/FMSR.2024.061206

may involve the modulation of key glycolytic enzymes and signaling pathways, such as the PI3K-AKT pathway, which is crucial for insulin signaling[33]. Additionally, mangiferin has been reported to reduce oxidative stress and inflammation, further supporting its role in improving metabolic health[34]. By enhancing glycolytic activity, mangiferin may help maintain energy homeostasis and mitigate the risk of metabolic diseases.

2.5.3 The Relationship Between Glycolysis and Skeletal Muscle Function

Skeletal muscle relies heavily on glycolysis for energy, especially during high-intensity exercise when oxygen availability is limited. The ability of skeletal muscle to efficiently utilize glucose through glycolysis is crucial for maintaining muscle performance and endurance. Impairments in glycolytic function can lead to muscle fatigue and decreased physical performance, commonly observed in conditions such as diabetes and obesity[35]. Recent research highlights the importance of glycolysis in muscle regeneration and adaptation to exercise, suggesting that compounds like mangiferin, which enhance glycolytic activity, could play a significant role in improving muscle function and overall metabolic health[36]. Therefore, the potential of mangiferin as a therapeutic agent in enhancing glycolysis and promoting skeletal muscle health warrants further investigation.

3. Conclusion

In recent years, the role of mangiferin as a promising agent in combating skeletal muscle aging has garnered significant attention within the scientific community. This review highlights the multifaceted benefits of mangiferin, particularly its influence on glycolysis and muscle metabolism, positioning it as a potential therapeutic candidate in the realm of age-related muscle degeneration. The antioxidant and anti-inflammatory properties of mangiferin not only enhance muscle function but also mitigate the oxidative stress that contributes to aging. These findings underscore the importance of further exploring mangiferin’s mechanisms of action, which could lead to novel interventions aimed at preserving skeletal muscle integrity in older populations.

Moreover, the impact of mangiferin on glycolytic pathways is particularly noteworthy. By promoting efficient energy metabolism, mangiferin facilitates the maintenance of muscle mass and function, which are crucial for overall health and quality of life in aging individuals. As such, the implications of these findings extend beyond basic science, suggesting potential clinical applications ranging from dietary supplementation to pharmacological interventions targeting muscle wasting conditions.

However, it is imperative to recognize the variability in research outcomes regarding mangiferin's effectiveness. Differences in study design, dosages, and research methodologies can lead to disparate conclusions, which necessitates a balanced interpretation of the existing literature. Future studies should aim to standardize these parameters to provide clearer insights into the optimal use of mangiferin for muscle health. Additionally, exploring synergistic effects with other compounds and understanding the long-term impacts of mangiferin supplementation will be crucial for developing comprehensive treatment strategies.

In summary, while the current body of evidence supports the beneficial role of mangiferin in skeletal muscle anti-aging, ongoing research is essential to fully elucidate its potential. A collaborative approach, integrating insights from various fields such as pharmacology, nutrition, and gerontology, will enhance our understanding and enable the translation of these findings into clinical practice. By fostering a multidisciplinary dialogue, we can pave the way for innovative solutions to address muscle aging and improve the overall well-being of the elderly population.

Declarations

Hanjie Jin and Haojie Tang contributed to data analyses, interpretation, and drafting of the manuscript. Chaoyue Zhao and Xuting Zhu contributed to the acquisition of data and drafting of the manuscript. All authors made substantial contributions to critically revising the manuscript. All authors approved the final version of the manuscript.

Acknowledgements

The current study did receive specific grants from Baise City scientific research and technology

ISSN 2618-1584 Vol. 6, Issue 12: 37-43, DOI: 10.25236/FMSR.2024.061206

Technical development plan. Project number: 20241544.

References

[1] Kedlian V R, Wang Y, Liu T, et al. Human skeletal muscle aging atlas[J]. Nature aging, 2024: 1-18.
[2] Stewart C E, Sharples A P. Aging, skeletal muscle, and epigenetics[J]. Plastic and Reconstructive Surgery, 2022, 150: 27S-33S.
[3] Rigoulet M, Bouchez C L, Paumard P, et al. Cell energy metabolism: An update[J]. Biochimica et Biophysica Acta (BBA)-Bioenergetics, 2020, 1861(11): 148276.
[4] Chen W J, Lin I H, Lee C W, et al. Aged skeletal muscle retains the ability to remodel extracellular matrix for degradation of collagen deposition after muscle injury[J]. International journal of molecular sciences, 2021, 22(4): 2123.
[5] Maenpuen S, Mee-Udorn P, Pinthong C, et al. Mangiferin is a new potential antimalarial and anticancer drug for targeting serine hydroxymethyltransferase[J]. Archives of Biochemistry and Biophysics, 2023, 745: 109712.
[6] Sarfraz M, Khan A, Batiha G E S, et al. Nanotechnology-based drug delivery approaches of mangiferin: Promises, reality and challenges in Cancer Chemotherapy[J]. Cancers, 2023, 15(16): 4194.
[7] Lee J Y, Kim H, Moon Y, et al. Enhancement of the water solubility and antioxidant capacities of mangiferin by transglucosylation using a cyclodextrin glycosyltransferase[J]. Enzyme and Microbial Technology, 2022, 159: 110065.
[8] Xu T, Wu X. Preparative separation of mangiferin glycosides by high speed counter current chromatography and comparison of their antioxidant and antitumor activities[J]. RSC advances, 2020, 10(43): 25780-25785.
[9] Wu J Y, Ding H Y, Wang T Y, et al. Improving aqueous solubility of natural antioxidant mangiferin through glycosylation by maltogenic amylase from parageobacillus galactosidasius DSM 18751[J]. Antioxidants, 2021, 10(11): 1817.
[10] Guo Y, Peng X, Liu F, et al. Potential of natural products in inflammation: biological activities, structure–activity relationships, and mechanistic targets[J]. Archives of Pharmacal Research, 2024: 1-33.
[11] Qi X, Aiyasamy K, Alenezi S K, et al. Anti-nociceptive and anti-inflammatory activities of visnagin in different nociceptive and inflammatory mice models[J]. Applied Biochemistry and Biotechnology, 2024, 196(6): 3441-3455.
[12] Xia G H, Li X H, Zhang Z, et al. Effects of fermentation treatments on Polygonatum odoratum flavones' antioxidant activities[J]. Saudi Journal of Biological Sciences, 2021, 28(9): 5011-5016.
[13] Li J, Yi X, Yao Z, et al. TNF receptor-associated factor 6 mediates TNFα-induced skeletal muscle atrophy in mice during aging[J]. Journal of Bone and Mineral Research, 2020, 35(8): 1535-1548.
[14] Lyu W, Kousaka M, Jia H, et al. Effects of Turmeric Extract on Age-Related Skeletal Muscle Atrophy in Senescence-Accelerated Mice[J]. Life, 2023, 13(4): 941.
[15] Liang J L, Xie J F, Wang C Y, et al. Regulatory roles of microRNAs in sarcopenia and exercise intervention[J]. Acta Physiologica Sinica, 2020, 72(5): 667-676.
[16] Wohlwend M, Laurila P P, Goeminne L J E, et al. Inhibition of CERS1 in skeletal muscle exacerbates age-related muscle dysfunction[J]. Elife, 2024, 12: RP90522.
[17] Leduc-Gaudet J P, Miguez K, Cefis M, et al. Autophagy ablation in skeletal muscles worsens sepsis-induced muscle wasting, impairs whole-body metabolism, and decreases survival[J]. Iscience, 2023, 26(8).
[18] Wang H H, Zhang Y, Qu T Q, et al. Nobiletin improves D-galactose-induced aging mice skeletal muscle atrophy by regulating protein homeostasis[J]. Nutrients, 2023, 15(8): 1801.
[19] Chen X, Ji Y, Liu R, et al. Mitochondrial dysfunction: roles in skeletal muscle atrophy[J]. Journal of Translational Medicine, 2023, 21(1): 503.
[20] Chen L, Pan L, Zeng Y, et al. CPNE1 regulates myogenesis through the PERK-eIF2α pathway mediated by endoplasmic reticulum stress[J]. Cell and Tissue Research, 2023, 391(3): 545-560.
[21] Rutkove S B, Chen Z Z, Pandeya S, et al. Surface Electrical Impedance Myography Detects Skeletal Muscle Atrophy in Aged Wildtype Zebrafish and Aged gpr27 Knockout Zebrafish[J]. Biomedicines, 2023, 11(7): 1938.
[22] Wang K, Smith S H, Iijima H, et al. Bioengineered 3D skeletal muscle model reveals complement 4b as a cell-autonomous mechanism of impaired regeneration with aging[J]. Advanced Materials, 2023, 35(17): 2207443.
[23] Englund D A, Jolliffe A, Aversa Z, et al. p21 induces a senescence program and skeletal muscle dysfunction [J]. Molecular Metabolism, 2023, 67: 101652.

ISSN 2618-1584 Vol. 6, Issue 12: 37-43, DOI: 10.25236/FMSR.2024.061206

[24] Jiang M, Li P, Wang Y, et al. Role of Nrf2 and exercise in alleviating COPD-induced skeletal muscle dysfunction[J]. Therapeutic Advances in Respiratory Disease, 2023, 17: 17534666231208633.
[25] Sugiura R, Kuwatani M, Kawakubo K, et al. Preoperative risk factors for skeletal muscle mass loss in patients with biliary tract cancer[J]. Journal of Hepato-Biliary-Pancreatic Sciences, 2024.
[26] Li W, Zhang Y, Liu Y, et al. CD155 is essential for skeletal muscle regeneration by regulating satellite cell proliferation and differentiation[J]. The FASEB Journal, 2024, 38(2): e23440.
[27] Velleman S G, Coy C S, Abasht B. Effect of growth selection of broilers on breast muscle satellite cell function: Response of satellite cells to NOV, COMP, MYBP-C1, and CSRP3[J]. Comparative Biochemistry and Physiology Part A: Molecular & Integrative Physiology, 2021, 255: 110917.
[28] Kobayashi Y, Tanaka T, Mulati M, et al. Cyclin-dependent kinase 1 is essential for muscle regeneration and overload muscle fiber hypertrophy[J]. Frontiers in cell and developmental biology, 2020, 8: 564581.
[29] He H, Yin H, Yu X, et al. PDLIM5 affects chicken skeletal muscle satellite cell proliferation and differentiation via the p38-MAPK pathway[J]. Animals, 2021, 11(4): 1016.
[30] Jiang A, Dong C, Li B, et al. MicroRNA-206 regulates cell proliferation by targeting G6PD in skeletal muscle[J]. The FASEB Journal, 2019, 33(12): 14083-14094.
[31] Zhu Q, Liang F, Cai S, et al. KDM4A regulates myogenesis by demethylating H3K9me3 of myogenic regulatory factors[J]. Cell death & disease, 2021, 12(6): 514.
[32] Ismaeel A, Peck B D, Montgomery M L M, et al. microRNA-1 Regulates Metabolic Flexibility in Skeletal Muscle via Pyruvate Metabolism[J]. bioRxiv, 2024.
[33] Ito N, Takatsu A, Ito H, et al. Slc12a8 in the lateral hypothalamus maintains energy metabolism and skeletal muscle functions during aging[J]. Cell Reports, 2022, 40(4).
[34] Nørregaard L B, Hansen C C, Wickham K A, et al. Exercise training alters skeletal muscle microvascular endothelial cell properties in recent postmenopausal females[J]. The Journal of Physiology, 2024.
[35] Ma S, Xing X, Huang H, et al. Skeletal muscle-derived extracellular vesicles transport glycolytic enzymes to mediate muscle-to-bone crosstalk[J]. Cell Metabolism, 2023, 35(11): 2028-2043. e7.
[36] Maehara H, Kokaji T, Hatano A, et al. DNA hypomethylation characterizes genes encoding tissue-dominant functional proteins in liver and skeletal muscle[J]. Scientific reports, 2023, 13(1): 19118.