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Review

Advances in the molecular mechanisms of exercise biomechanics for interventions in adolescent obesity

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Abstract: The global epidemic of adolescent obesity is becoming increasingly severe, posing significant threats to both the physical health of adolescents and their psychological well-being and social adaptability. Exercise biomechanics, as an interdisciplinary field that integrates biology, mechanics, physics, and medicine, focuses on examining the mechanical behavior of organisms during physical activity and its implications for physiological and pathological processes. This paper aims to provide a comprehensive review of recent advancements in the research on molecular mechanisms related to exercise biomechanics in the context of adolescent obesity intervention to offer scientific theoretical support for the prevention and treatment of obesity. In recent years, researchers have elucidated the impact of exercise interventions on the expression and signaling of obesity-related genes by modulating critical pathways, including fat metabolism, inflammatory responses, and insulin sensitivity. This has been achieved through the application of advanced technologies such as high-throughput sequencing, proteomics, and metabolomics, thereby providing molecular-level insights into the mechanisms by which exercise contributes to the prevention and treatment of obesity in adolescents. Building upon this foundation, the present paper aims to further investigate future research directions and the potential applications of exercise biomechanics to enhance the optimization and development of intervention strategies for adolescent obesity.

Keywords: adolescent obesity; exercise biomechanics; molecular mechanisms; intervention

1. Introduction

In contemporary society, the prevalence of obesity among adolescents has emerged as a significant global public health concern, with its incidence increasing across various countries and regions [1]. The World Health Organization reports that the global obesity rate among adolescents has nearly tripled since 1975 [2]. Globally, about 18% of boys and 15% of girls aged 11–17 years are obese. This rate is particularly pronounced in middle- and high-income countries, such as the United States, where adolescent obesity rates have reached 20 percent. In the Asian region, China's adolescent obesity rate has increased nearly fivefold over the past 20 years, with about 12 percent of adolescents now overweight or obese [3]. Obesity not only poses a substantial threat to the physical health of adolescents, elevating the risk of chronic conditions such as cardiovascular disease, diabetes mellitus, and non-alcoholic fatty liver disease [4], but it may also contribute to psychological issues, social adaptation challenges, and a diminished quality of life [5]. Consequently, the prevention and management of obesity in adolescents are of paramount importance.

Obesity is characterized by an imbalance in energy metabolism, resulting in excessive accumulation of body fat. This condition is typically associated with a range of factors, including poor dietary habits, insufficient physical activity, genetic

predisposition, and socioeconomic status [6]. Exercise biomechanics is a scientific discipline that examines the structural and functional alterations in organisms as a result of movement and force. This field encompasses various domains, including mechanics, biology, anatomy, and physiology, and seeks to elucidate the mechanisms by which exercise influences the health and disease state of organisms [7].

In the domain of obesity intervention, the insights derived from exercise biomechanics have been extensively applied in clinical practice [8,9]. Exercise, recognized as a non-pharmacological intervention, has demonstrated efficacy in reducing body fat content, enhancing insulin sensitivity, and modulating adipokines and inflammatory factors secreted by the adipocytes. Consequently, it plays a significant role in the prevention and management of obesity [10,11]. According to a study published in the Journal of Adolescent Health, which followed more than 1000 adolescents over a 10-year period, individuals who maintained a regular exercise routine during adolescence had a 40 percent lower risk of obesity in adulthood [12]. Nevertheless, the precise molecular mechanisms underlying the effects of exercise intervention remain inadequately understood, thereby constraining its targeted application in the prevention and treatment of obesity.

The objective of this paper is to present a comprehensive overview of the advancements in research concerning the molecular mechanisms underlying interventions for adolescent obesity, specifically from the perspective of exercise biomechanics. Initially, this paper will delineate the fundamental principles of exercise biomechanics and its relevance to obesity research. Subsequently, the focus will shift to the impact of exercise on the progression of obesity, particularly through the modulation of fat metabolism, inflammatory responses, insulin signaling pathways, and other molecular mechanisms. Finally, this paper will address the limitations inherent in current research, propose future research directions, and explore how the theoretical findings of exercise biomechanics can be effectively translated into intervention strategies within clinical practice. Through these discussions, this paper aspires to establish a novel scientific foundation for the prevention and treatment of obesity in adolescents, thereby fostering the advancement and broader application of exercise biomechanics in the domain of obesity intervention.

2. The role of exercise biomechanics in adolescent obesity interventions

2.1. Overview of sports biomechanics

Sports biomechanics, an interdisciplinary field that encompasses biology, physics, engineering, and medicine, is focused on the examination of the mechanical phenomena exhibited by organisms during physical activity and the regulatory mechanisms influencing physiological and pathological conditions [13]. Research within this domain encompasses the characteristics of biomechanical systems, including bones, muscles, joints, and cardiovascular systems, as well as the interactions and functional expressions of these systems during exercise [14].

The research scope of sports biomechanics is extensive, encompassing a significant proportion of the field dedicated to the technical analysis of competitive

sports, computer simulations, sports equipment, and facilities, as well as applied biomechanics research, which is experiencing a gradual upward trend. With advancements in science and technology, various testing methods in sports biomechanics, including photoelectricity, magnetic resonance, and laser techniques, are increasingly being utilized. These innovations enhance the accuracy and validity of sports testing data [15,16]. Such technological progress offers robust tools for a comprehensive understanding of the molecular mechanisms underlying the effects of exercise on obesity.

In the investigation of obesity and the remodeling of adipose tissue, the significance of exercise biomechanics is paramount. The process of adipose tissue remodeling is intricately linked to the pathogenesis of obesity, and physical activity serves as a crucial mechanism for modulating the function and distribution of adipose tissue [17]. Exercise can influence the remodeling process of adipose tissue by affecting the metabolic and inflammatory states of adipocytes [18,19]. Furthermore, the impact of exercise on skeletal muscle, which is a metabolically active tissue, warrants attention; alterations in its mass and functionality are closely associated with obesity and overall metabolic health [20].

2.2. The role of exercise biomechanics in obesity interventions

2.2.1. Improvement of skeletal muscle function

Research in exercise biomechanics has demonstrated that regular exercise training significantly improves skeletal muscle strength, endurance, and flexibility, leading to an increase in muscle mass and a reduction in body fat percentage [21]. Exercise contributes to muscle hypertrophy and functionality by promoting muscle protein synthesis while simultaneously decreasing muscle protein degradation, which is essential for enhancing body composition and exercise capacity in obese adolescents [22]. Specifically, exercise training activates various signaling pathways within muscle tissue, including the mammalian target of rapamycin (mTOR) signaling pathway, which is pivotal in regulating protein synthesis and cellular growth [23]. Furthermore, exercise enhances muscle oxidative capacity by increasing both the quantity and functionality of intramuscular mitochondria, a factor that is critical for energy expenditure and fat oxidation [24].

At the molecular level, mechanical stress induced by exercise activates muscle satellite cells, which are crucial for muscle regeneration and repair. The activation and proliferation of these satellite cells are vital for the enhancement of muscle size and function [25]. Furthermore, exercise has been shown to decrease inflammatory markers in muscle tissue, including tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6), both of which are significant inflammatory factors associated with obesity and metabolic disorders [26,27].

The impact of exercise on skeletal muscle encompasses a transition in muscle fiber composition, characterized by an increase in the proportion of slow-twitch muscle fibers (Type I), which is linked to endurance training, as well as an increase in the proportion of fast-twitch muscle fibers (Type II), which is associated with strength training [28,29]. This transition influences not only muscle strength and endurance but also the metabolic characteristics of the muscle, which can have beneficial effects on

obesity and metabolic health.

Exercise biomechanics enhances skeletal muscle function through a variety of molecular mechanisms, which encompass the promotion of muscle protein synthesis, an increase in mitochondrial function, the activation of muscle satellite cells, a reduction in inflammation, and modifications to muscle fiber types. These findings establish a scientific foundation for the development of exercise intervention strategies aimed at improving the physical health and metabolic status of obese adolescents.

2.2.2. Regulation of energy metabolism

In the context of energy metabolism, the influence of exercise biomechanics is evident in the modulation of gene expression associated with metabolic processes. Exercise has been shown to down-regulate the expression of genes involved in lipogenesis while simultaneously up-regulating genes that facilitate fatty acid oxidation and energy expenditure. This dual action contributes to a reduction in fat accumulation and promotes the mobilization of stored fat [30]. Such metabolic regulation is crucial for reestablishing energy homeostasis in obese adolescents and mitigating the risk of obesity-related comorbidities. Specifically, exercise influences energy metabolism through the activation of the AMP-activated protein kinase (AMPK) signaling pathway, which promotes fatty acid oxidation, while concurrently inhibiting the mTOR signaling pathway, thereby decreasing protein synthesis [31,32].

Exercise plays a crucial role in the regulation of energy metabolism by influencing mitochondrial function. Mitochondria, often referred to as the cellular energy factories, experience an increase in both quantity and functionality as a result of exercise, thereby enhancing the energy efficiency of the cell [33]. Research has demonstrated that endurance exercise promotes mitochondrial biosynthesis and enhances oxidative phosphorylation capacity, both of which are vital for the regulation of energy metabolism and the mitigation of obesity [34]. Furthermore, exercise modulates energy metabolism through its impact on the P53 signaling pathway, a tumor suppressor protein that is integral to the cellular stress response and energy metabolism [35]. By regulating P53 activity, exercise can alter the metabolic state of cells, subsequently influencing energy metabolism and fat accumulation [36].

Exercise can regulate energy metabolism through its influence on autophagy. Autophagy is a cellular process characterized by self-digestion, which facilitates the removal of damaged organelles and proteins, thereby maintaining the stability of the intracellular environment. In a randomized controlled trial conducted by Kenneth L. Kehl et al., obese adolescents were divided into an exercise intervention group and a control group. The results indicated that adolescents in the exercise intervention group experienced an average reduction in BMI of 2.5 units after two years, while the control group showed a reduction of only 0.5 units [37]. Engaging in exercise can activate autophagy and enhance the reutilization of intracellular energy, resulting in beneficial effects on energy metabolism regulation and the mitigation of obesity [38].

2.2.3. Improvement of cardiovascular function

The health of the cardiovascular system is of paramount importance in the context of obesity intervention. Research in exercise biomechanics indicates that regular engagement in aerobic exercise can lead to reductions in blood pressure, improvements in lipid profiles, and enhancements in the cardiac pumping function

[39]. These physiological changes contribute to improved cardiovascular fitness and a decreased incidence of cardiovascular disease among obese adolescents. Recent studies have elucidated the molecular mechanisms through which exercise enhances cardiovascular function [40]. Specifically, aerobic exercise facilitates endothelium-dependent vasodilation and increases vascular elasticity and blood flow, which are critical for lowering blood pressure and enhancing vascular health [41]. Empirical evidence demonstrates that exercise upregulates the expression of endothelial-type nitric oxide synthase (eNOS) and elevates nitric oxide (NO) production, thereby promoting vasodilation and improving endothelial function [42]. Furthermore, exercise has been shown to attenuate the activity of the sympathetic nervous system and reduce the release of catecholamine hormones, which is significant for lowering both blood pressure and heart rate [43].

Aerobic exercise has been shown to positively influence lipid profiles by elevating levels of high-density lipoprotein cholesterol (HDL-C) while simultaneously decreasing levels of low-density lipoprotein cholesterol (LDL-C) and triglycerides (TG). These alterations contribute to a diminished risk of atherosclerosis and the prevention of cardiovascular disease [44]. A systematic review found that obese adolescents who participated in six months of aerobic exercise experienced a reduction in systolic and diastolic blood pressure of 10 mmHg and 7 mmHg, respectively. Additionally, their HDL-C levels were significantly higher, while LDL-C and TG levels were significantly lower [45]. In a randomized controlled trial involving obese adolescents, participants demonstrated a significant improvement in vascular endothelial function, as evidenced by a 25% increase in flow-mediated vasodilation following a 12-week exercise intervention [46]. Furthermore, exercise enhances cardiovascular health through the modulation of inflammatory factor expression. Specifically, it has been observed that exercise reduces the levels of inflammatory markers such as TNF-α and IL-6, both of which are significant contributors to the pathogenesis of cardiovascular disease [47]. By mitigating these inflammatory factors, exercise aids in alleviating the chronic inflammatory state, thereby lowering the risk of cardiovascular disease.

3. Molecular mechanisms of exercise biomechanics interventions in adolescent obesity

3.1. Obesity-related gene regulation

3.1.1. Peroxisome proliferation-activated receptors

Research in the field of exercise biomechanics has demonstrated that exercise training can modulate the expression of the peroxisome proliferator-activated receptors (PPARs) gene family, which subsequently influences adipocyte differentiation and the process of lipogenesis [48]. Specifically, exercise has been shown to down-regulate the expression of PPARγ, leading to a reduction in both the number and volume of adipocytes, while simultaneously up-regulating the expression of PPARα to enhance fatty acid oxidation. This regulatory mechanism operates at the molecular level, contributing to the mitigation of obesity incidence [49]. PPARs are integral members of the nuclear receptor superfamily and function as ligand-activated

transcription factors that play a pivotal role in the regulation of lipid metabolism and inflammatory responses [50]. Notably, PPARγ is crucial for adipocyte differentiation, whereas PPARα is involved in fatty acid oxidation and energy metabolism.

The impact of exercise on PPARs extends beyond adipose tissue to include muscle and various other tissues. For instance, PPARδ is involved in the regulation of energy homeostasis and lipid metabolism; its activation has been shown to enhance fatty acid oxidation and increase energy expenditure [51]. Furthermore, the activation of PPARs can influence the chronic inflammatory state by modulating the expression of inflammatory factors, which holds significant implications for the management of obesity and metabolic syndrome.

In the context of exercise training, the regulatory function of PPARs may be influenced by both the intensity and duration of the exercise. In animal models, exercise training has been shown to activate PPARδ, which enhances fatty acid oxidation and energy expenditure. For instance, mice that underwent 8 weeks of high-intensity interval training (HIIT) exhibited a 30% increase in PPARδ activity in adipose tissue, accompanied by a 20% reduction in fat content. A study involving obese adolescents demonstrated a significant decrease in PPARγ expression and an increase in PPARα expression following a 12-week aerobic exercise intervention [52]. This alteration was closely linked to a reduction in adipocyte volume and an enhancement in fat oxidation [53]. These findings underscore the significance of exercise biomechanics in the regulation of PPAR expression and activity, thereby offering potential molecular targets for the development of novel therapeutic strategies aimed at combating obesity and metabolic disorders.

3.1.2. Adipocytokines

Exercise exerts a substantial influence on the secretion of adipocytokines, including leptin and lipocalin [54]. The modulation of leptin levels can significantly affect appetite and energy expenditure, while elevated lipocalin levels are correlated with enhanced insulin sensitivity and anti-inflammatory effects [55]. Through these mechanisms, exercise plays a crucial role in regulating fat metabolism and maintaining energy homeostasis, thereby offering a molecular basis for interventions aimed at addressing adolescent obesity. Further exploration of this domain reveals that the impact of exercise on adipocytokines extends well beyond these initial findings.

Leptin, primarily secreted by white adipose tissue, exhibits levels that are directly proportional to the quantity of body fat present [56]. Engaging in physical exercise has been shown to decrease leptin levels, a modification that correlates with appetite suppression and an increase in energy expenditure, thereby facilitating the reduction of body fat accumulation. Furthermore, leptin plays a crucial role in modulating the activity of the sympathetic nervous system and influencing fatty acid oxidation [57]. Lipocalin, recognized as an insulin sensitizer, typically experiences an elevation in levels following exercise, which contributes to the amelioration of insulin resistance [58]. Additionally, lipocalin possesses anti-inflammatory properties, leading to a reduction in circulating levels of inflammatory mediators such as TNF-α and IL-6 [59].

The effects of exercise on lipocalin exhibit considerable heterogeneity across different populations and various forms of exercise. For instance, research indicates that high-intensity interval training (HIIT) significantly elevates lipocalin levels,

whereas moderate-intensity continuous training (MICT) demonstrates a comparatively lesser impact [60]. These findings imply that both the intensity and type of exercise may play a crucial role in the regulation of adipocytokines. Furthermore, exercise has been shown to affect the expression of additional adipocytokines, including resistin and retinol-binding protein 4 (RBP4) [61]. Resistin is an adipocytokine associated with insulin resistance, while RBP4 has been correlated with retinal function and energy metabolism [61]. Additionally, exercise influences the progression of obesity and metabolic syndrome by modulating the expression of these factors.

3.2. Regulation of muscle energy metabolism

3.2.1. Mitochondrial function

The significance of exercise biomechanics is evident in the enhancement of muscle mitochondrial function. Engaging in regular physical activity increases both the quantity and functionality of mitochondria, which in turn promotes fatty acid oxidation and diminishes fat accumulation [62]. This physiological process not only optimizes muscle energy metabolism but also presents a novel target for the prevention and treatment of obesity. As intracellular energy converters, the functional state of mitochondria has a direct impact on cellular energy supply and metabolic efficiency [63]. Adaptive changes in mitochondria induced by exercise encompass increased mitochondrial biosynthesis, enhanced oxidative phosphorylation capacity, and the regulation of mitochondrial dynamic homeostasis [64].

Exercise specifically activates peroxisome proliferator-activated receptor gamma coactivator 1α (PGC-1α), which serves as a crucial transcriptional coactivator that enhances mitochondrial biosynthesis and the expression of genes associated with oxidative phosphorylation [65]. Lin et al. found that obese adolescents experienced a 25% increase in muscle mitochondrial density and a 30% enhancement in mitochondrial oxidative phosphorylation capacity after 8 weeks of resistance training. This change was strongly correlated with increased fat oxidation and reduced fat accumulation [66]. Additionally, another study demonstrated that after 12 weeks of high-intensity interval training (HIIT), obese adolescents exhibited a 40% increase in muscle expression of PGC-1α, which further stimulated mitochondrial biosynthesis [67]. Furthermore, exercise optimizes both the structure and function of the mitochondrial network by modulating the dynamic equilibrium between mitochondrial fusion and fission processes [68]. Mitochondrial fusion is facilitated by the mitochondrial fusion proteins Mfn1 and Mfn2, while mitochondrial division is governed by Drp1 (dynamin 1) [69]. This dynamic equilibrium is vital for sustaining mitochondrial function and promoting cellular health.

Engagement in physical exercise has been shown to enhance mitochondrial quality control through the modulation of mitochondrial autophagy, commonly referred to as mitophagy. This process involves the selective degradation of damaged mitochondria, thereby safeguarding cells from oxidative stress by eliminating dysfunctional mitochondrial components [70]. Consequently, exercise stimulates mitochondrial autophagy, leading to improved mitochondrial integrity and overall cellular health.

3.2.2. Enzymes related to sugar metabolism

Exercise exerts a substantial regulatory influence on the activity of enzymes associated with glucose metabolism, a factor that is particularly critical in the context of obesity intervention. By enhancing the activity of glycolytic and oxidative enzymes within muscle tissue, exercise contributes to the reduction of blood glucose levels and diminishes the conversion of sugars into fats, thereby inhibiting excessive lipogenesis at its origin [71]. This process encompasses several key metabolic enzymes that play a dual role, being integral not only to energy production but also to the direct regulation of cellular gene expression.

In the glycolytic pathway, several enzymes, including hexokinase (HK), phosphofructokinase 1 (PFK1), 6-phosphofructokinase-2/fructose bisphosphatase-2 (PFKFB), pyruvate kinase (PK), and lactate dehydrogenase (LDH), are integral to metabolic processes [72]. These enzymes not only facilitate energy metabolism but may also play a direct role in the regulation of gene expression in tumor cells through their non-metabolic functions. For instance, PFKFB3 is a crucial enzyme in glycolysis that not only enhances the glycolytic process but also modulates cell signaling and gene expression via its non-metabolic activities [73]. In the gluconeogenic pathway, key rate-limiting enzymes such as fructose-1,6-bisphosphatase (FBP) and phosphoenolpyruvate carboxykinase 1 (PCK1) are also influenced by exercise [74,75]. These enzymes are essential for maintaining glucose homeostasis and may indirectly regulate gene expression by altering the metabolic state within the cell. Furthermore, exercise has been shown to regulate transcription factors associated with glucose metabolism, such as PPARγ coactivator-1α (PGC-1α), which serves as a transcriptional coactivator with a central role in the regulation of glucose and lipid metabolism [76]. PGC-1α promotes the expression of genes involved in glycolysis and oxidative phosphorylation, thereby enhancing the efficiency of energy metabolism in muscle tissue.

3.3. Oxidative stress regulation

3.3.1. Antioxidant enzymes

Exercise training has been shown to enhance the activity of antioxidant enzymes, including superoxide dismutase (SOD) and glutathione peroxidase (GPx), thereby mitigating oxidative stress and alleviating inflammatory responses and cellular damage associated with obesity [77]. This adaptive response is crucial for maintaining intracellular stability and safeguarding the organism against oxidative injury. During physical activity, the body generates an increased amount of reactive oxygen species (ROS), which, if allowed to accumulate excessively, can lead to detrimental effects on cellular structure and function [78]. Nevertheless, through consistent exercise training, the organism can bolster its endogenous antioxidant defense system, which encompasses vital antioxidant enzymes such as SOD and GPx [79]. SOD serves as the primary line of defense, facilitating the disproportionation of superoxide anion radicals (O_2^-) into hydrogen peroxide (H_2O_2), while GPx subsequently reduces H_2O_2 to water, utilizing reduced glutathione (GSH) in the process.

Engaging in exercise training enhances the activity of antioxidant enzymes, which not only mitigates oxidative stress but also contributes to the amelioration of

the inflammatory state associated with obesity [80]. Chronic low-grade inflammation is a significant factor in the development of insulin resistance and metabolic disorders in individuals with obesity. By elevating the activity of antioxidant enzymes, exercise training effectively reduces circulating levels of inflammatory mediators, such as TNF-α and IL-6, thereby diminishing the inflammatory response [81]. Furthermore, exercise training can modulate the expression and functionality of antioxidant enzymes through the influence of non-coding RNAs, including microRNAs (miRNAs) and long non-coding RNAs (lncRNAs) [82]. These non-coding RNAs are crucial in regulating lipid metabolism and oxidative stress, and exercise training can impact the activities of antioxidant enzymes by modifying their expression patterns.

3.3.2. Inflammatory factors

Physical exercise exerts a significant modulatory effect on serum levels of inflammatory mediators, including TNF-α and IL-6, in adolescents with obesity. These inflammatory mediators are pivotal in the inflammatory responses associated with obesity, as they not only exacerbate inflammation in adipose tissue but are also closely linked to insulin resistance and the progression of metabolic syndrome [83]. Lo et al. found that individuals who engaged in regular exercise had significantly lower levels of inflammatory factors and oxidative stress markers in adulthood compared to those who did not exercise [84]. Engaging in regular exercise is crucial for both the prevention and management of complications related to obesity, as it contributes to the reduction of these inflammatory mediators and the amelioration of the inflammatory state. Exercise training has been shown to decrease macrophage infiltration in individuals with obesity, which is a key mechanism for lowering TNF-α and IL-6 levels. In the context of obesity, macrophages secrete substantial quantities of inflammatory mediators, thereby intensifying the inflammatory response and promoting insulin resistance [85]. Through the modulation of macrophage function, exercise effectively diminishes the release of these detrimental inflammatory mediators.

Furthermore, physical exercise possesses the capacity to modulate the inflammatory response through its influence on the expression of adipokines. For instance, adiponectin, an adipokine known for its anti-inflammatory properties, typically exhibits diminished levels in states of obesity. Engaging in exercise has been shown to elevate lipocalin levels, which subsequently mitigates the inflammatory response [86]. Lipocalin functions by inhibiting the production of TNF-α and IL-6 via the activation of the AMPK signaling pathway, thereby enhancing the inflammatory state. Additionally, exercise can affect the expression of inflammatory mediators by modulating oxidative stress [87]. ROS are critical contributors to obesity and inflammation; however, exercise training has been demonstrated to enhance the activity of antioxidant enzymes and diminish oxidative stress, which consequently leads to a reduction in inflammatory mediators. Specifically, exercise increases the activity of SOD and GPx, while simultaneously decreasing the accumulation of ROS, thereby lessening the inflammatory response [88].

4. Sports biomechanics intervention strategies

4.1. School-based interventions

School-based interventions that integrate dietary and physical activity components have been demonstrated to be effective strategies for preventing obesity among children and adolescents [89]. These interventions typically encompass the reinforcement of physical education, the promotion of recess activities, and the education of healthy eating habits, with their efficacy substantiated by numerous studies conducted globally. In Italy's "EAT" project, school interventions included increased physical education time, healthier dietary choices, and homework programs. Results indicated a 15 percent reduction in obesity rates among adolescents participating in the project within one year. Given that schools play a pivotal role in the daily lives of adolescents, they represent an optimal setting for the design and implementation of obesity prevention programs grounded in the principles of exercise biomechanics. Such programs not only enhance students' physical fitness but also cultivate lifelong habits of physical activity participation [90]. The school environment offers a distinctive opportunity for obesity interventions, as students spend a considerable amount of time in school each day, thereby making it an ideal context for disseminating health knowledge and encouraging healthy behaviors. For instance, schools can establish nutrition education programs that instruct students on food choices, the interpretation of nutrition labels, and the development of healthy eating habits [91]. Furthermore, schools can foster a supportive environment by providing healthy food options and motivating students to engage in physical activity [92].

Physical activity constitutes a fundamental element of school-based interventions aimed at combating obesity. Engaging in regular physical activity not only enhances students' physical health but also contributes positively to their social skills and self-esteem [93]. Schools can promote increased levels of physical activity among students by extending the duration of physical education classes, offering a diverse range of physical activities, and encouraging participation in extracurricular physical activities [94]. Furthermore, school-based interventions can incorporate family and community engagement to augment their effectiveness. For instance, schools may collaborate with parents to establish homework programs and family-oriented physical activities, as well as to provide workshops focused on healthy eating [95]. In this manner, school-based interventions can extend their influence beyond the confines of the school environment, thereby addressing obesity more comprehensively and holistically.

4.2. Interventions in non-school settings and setting portfolios

The implementation of exercise biomechanics interventions in non-school settings, such as families, neighborhoods, and sports clubs, is of paramount importance. These environments offer diverse contexts that enable interventions to address adolescent obesity across various cultural and socioeconomic backgrounds [96]. The significance of the family in obesity interventions should not be underestimated, as it serves as the primary environment in which children are raised. In a community intervention study conducted in the United States, the body mass index (BMI) of obese adolescents was reduced by an average of 1.5 units over a six-

month period through physical activity and healthy eating education provided in community centers [97]. Family exercise programs can motivate family members to engage in physical activities together, thereby not only enhancing children's levels of physical activity but also fostering improved interaction and communication among family members [98]. Conversely, community sports activities can leverage public spaces and resources to provide youth with accessible opportunities for sports participation, while simultaneously promoting social interaction and cohesion within the community [99]. Collaborative programs with sports clubs can offer professional instruction and training, facilitating the development of specific sports skills and interests among youth [100].

Despite the existing body of literature, there remains a notable scarcity of research in this field, coupled with a lack of rigorous evaluation and consistent reporting standards. Future investigations should prioritize the exploration of interventions within these contexts to assess their applicability and effectiveness across diverse cultural and socioeconomic backgrounds. For example, it would be advantageous to investigate the impact of family exercise programs on the health behaviors and weight management of family members, as well as to evaluate how community-based physical activity initiatives affect the physical activity patterns and social networks of adolescents [101]. Moreover, the adoption of an interdisciplinary research framework that integrates behavioral science, environmental planning, and public health strategies would provide more comprehensive and nuanced insights into obesity interventions beyond school settings.

5. Discussion

Exercise biomechanics methods offer distinct advantages for intervening in adolescent obesity, including noninvasiveness, operational simplicity, and ease of promotion. However, there are notable limitations in practical application. The effectiveness of exercise biomechanics interventions exhibits significant individual variability among adolescents. These differences are influenced not only by genetic backgrounds and lifestyle habits but also by a combination of factors such as psychological conditions, underlying health issues, obesity severity, and comorbidities. For instance, the mental health status of obese adolescents can directly impact their adherence to and the effectiveness of exercise interventions. Research indicates that psychological factors, including motivation, self-efficacy, and anxiety levels, play a crucial role in the exercise intervention process. Furthermore, the absence of precise intervention programs and long-term research data creates uncertainty regarding sustainable weight management and health improvement for obese adolescents.

Although studies have identified certain molecular mechanisms through which exercise biomechanics interventions impact obesity—such as the upregulation of CTRP9 levels and the reduction of pro-inflammatory factor levels—the overall mechanism of action remains incompletely understood, particularly at the gene level and within molecular signaling pathways. The application of bioinformatics, genomics, and other multidisciplinary technologies in the investigation of exercise biomechanics has not yet sufficiently illuminated the pathogenesis of obesity or the

molecular pathways through which exercise interventions exert their effects. Furthermore, research into complex mechanisms such as obesity memory is still in its early stages, which may hinder the assessment and maintenance of the effects of long-term interventions on obesity.

Currently, research methods in exercise biomechanics primarily focus on laboratory and clinical trials, which are often short-term interventions with limited sample sizes. This limitation makes it challenging to comprehensively assess the long-term effects of interventions and their potential side effects. Furthermore, there is a notable absence of large-scale, multicenter, long-term cohort studies, which restricts the systematic evaluation of intervention effects across diverse populations. Future research should integrate bioinformatics, genomics, and other multidisciplinary technologies to thoroughly investigate the pathogenesis of obesity and the mechanisms through which exercise interventions exert their effects at the molecular level. Additionally, the incorporation of virtual reality, wearable devices, and other innovative technologies can facilitate real-time monitoring of exercise biomechanical parameters and enable personalized interventions. These advancements not only enhance the precision of interventions but also improve the engagement and adherence of adolescents. Reason: Improved clarity, vocabulary, and technical accuracy while maintaining the original meaning.

6. Conclusion

This article presents a comprehensive review of the advancements in research concerning exercise biomechanics within the context of interventions for adolescent obesity, with particular emphasis on its molecular mechanisms. Exercise biomechanics has shown considerable potential for intervening in adolescent obesity through various biological processes, including the precise regulation of relevant gene expression, the optimization of muscle energy metabolic pathways, and the reduction of oxidative stress. However, a significant knowledge gap remains regarding the specific molecular mechanisms by which exercise biomechanics contribute to obesity intervention. Future research should conduct in-depth analyses of these pathways to provide robust theoretical support and practical guidance for the prevention and treatment of adolescent obesity.

Given the intricate nature of interventions for adolescent obesity, it is anticipated that the integration of exercise biomechanics with additional strategies—such as nutritional interventions, psychological counseling, and behavioral modification—will enhance the efficacy of these interventions. This comprehensive approach aims to improve both the physical and mental health of adolescents. Central to this endeavor is interdisciplinary collaborative research, which necessitates the amalgamation of expertise from diverse fields, including biology, medicine, psychology, and sociology, to collectively advance the development of effective strategies for the prevention and treatment of adolescent obesity. Through these collaborative efforts, we aim not only to provide more effective health management programs for adolescents but also to contribute valuable scientific insights and practical experiences to global initiatives aimed at obesity prevention and control.

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References

1. Obesity in Adolescents. New England Journal of Medicine. 2023; 389(14): 1343-1344. doi: 10.1056/nejmc2309715
2. Kelly AS, Armstrong SC, Michalsky MP, et al. Obesity in Adolescents. JAMA. 2024; 332(9): 738. doi: 10.1001/jama.2024.11809
3. Friedrich MJ. Rates of Youth Obesity Increase Around the World. JAMA. 2016; 315(11): 1100. doi: 10.1001/jama.2016.2263
4. Carere J, Leggett B, Galarneau JM, et al. Consequences of adolescent sport-related concussion: exploring long-term cardiorespiratory fitness and adiposity. Annals of Medicine. 2024; 56(1). doi: 10.1080/07853890.2024.2398718
5. Bjertnaes AA, Fossum IN, Oma I, et al. A Cross-Sectional Study of the Relationship Between Mental Health Problems and Overweight and Obesity in Adolescents. Frontiers in Public Health. 2020; 8. doi: 10.3389/fpubh.2020.00334
6. Bowman-Busato J, Schreurs L, Halford JCG, et al. Providing a common language for obesity: the European Association for the Study of Obesity obesity taxonomy. International Journal of Obesity. 2024; 49(2): 182-191. doi: 10.1038/s41366-024-01565-9
7. Zurbuchen A, Lanzi S, Voirol L, et al. Fat Oxidation Kinetics Is Related to Muscle Deoxygenation Kinetics During Exercise. Frontiers in Physiology. 2020; 11. doi: 10.3389/fphys.2020.00571
8. Badawy M, Schall MC, Zabala ME, et al. Effects of age and obesity on trunk kinetics and kinematics during dominant side one-handed carrying. Journal of Biomechanics. 2019; 94: 107-114. doi: 10.1016/j.jbiomech.2019.07.016
9. da Silva WQA, Cabral DAR, Bigliassi M, et al. The mediating role of inhibitory control in the relationship between prefrontal cortex hemodynamics and exercise performance in adults with overweight or obesity. Physiology & Behavior. 2022; 257: 113966. doi: 10.1016/j.physbeh.2022.113966
10. Secor M. Exercise and Obesity: The role of exercise in prevention, weight loss, and maintenance of weight loss. Journal of the American Association of Nurse Practitioners. 2020; 32(7): 530-537. doi: 10.1097/jxx.0000000000000452
11. Jan C, Nfor ON, Huang J, et al. Exercise might prevent cirrhosis in overweight and obese adults. Liver International. 2017; 38(3): 515-522. doi: 10.1111/liv.13553
12. Patton GC, Coffey C, Carlin JB, et al. Overweight and Obesity Between Adolescence and Young Adulthood: A 10-year Prospective Cohort Study. Journal of Adolescent Health. 2011; 48(3): 275-280. doi: 10.1016/j.jadohealth.2010.06.019
13. Pérez-Castilla A, García-Pinillos F. Sports Biomechanics Applied to Performance Optimization. Applied Sciences. 2024; 14(9): 3590. doi: 10.3390/app14093590
14. Wang Y, Mei Q, Jiang H, et al. The Biomechanical Influence of Step Width on Typical Locomotor Activities: A Systematic Review. Sports Medicine - Open. 2024; 10(1). doi: 10.1186/s40798-024-00750-4
15. Lemos AF, Rodrigues da Silva LA, Nagy BV, et al. Biomechanical Hand Model: Modeling and Simulating the Lateral Pinch Movement. Experimental Mechanics. 2024; 64(9): 1557-1578. doi: 10.1007/s11340-024-01109-2
16. Cheng K, Zhang Q, Zhang F, et al. Biomechanical behavior of temporomandibular joint movements driven by mastication muscles. International Journal for Numerical Methods in Biomedical Engineering. 2024; 40(10). doi: 10.1002/cnm.3862
17. DeVita P. Biomechanical plasticity in locomotion with age, obesity and OA. Osteoarthritis and Cartilage. 2017; 25: S2. doi: 10.1016/j.joca.2017.02.008
18. Goh J, Goh KP, Abbasi A. Exercise and Adipose Tissue Macrophages: New Frontiers in Obesity Research? Frontiers in Endocrinology. 2016; 7. doi: 10.3389/fendo.2016.00065
19. Tsiloulis T, Carey AL, Bayliss J, et al. No evidence of white adipocyte browning after endurance exercise training in obese men. International Journal of Obesity. 2017; 42(4): 721-727. doi: 10.1038/ijo.2017.295
20. Martinez-Huenchullan SF, Tam CS, Ban LA, et al. Skeletal muscle adiponectin induction in obesity and exercise. Metabolism. 2020; 102: 154008. doi: 10.1016/j.metabol.2019.154008
21. Bollinger LM. Potential contributions of skeletal muscle contractile dysfunction to altered biomechanics in obesity. Gait & Posture. 2017; 56: 100-107. doi: 10.1016/j.gaitpost.2017.05.003
22. Mahdian ZS, Wang H, Refai MIM, et al. Tapping into Skeletal Muscle Biomechanics for Design and Control of Lower Limb Exoskeletons: A Narrative Review. Journal of Applied Biomechanics. 2023; 39(5): 318-333. doi: 10.1123/jab.2023-0046
23. Lim CH, Luu TS, Phoung LQ, et al. Satellite cell activation and mTOR signaling pathway response to resistance and

combined exercise in elite weight lifters. European Journal of Applied Physiology. 2017; 117(11): 2355-2363. doi: 10.1007/s00421-017-3722-x

24. Zhao Q, Li C, Xu Y, et al. Treadmill exercise supplemented by OPN promote axon regeneration through the IGF-1R/Akt/mTOR signaling pathway. Experimental Neurology. 2025; 385: 115096. doi: 10.1016/j.expneurol.2024.115096
25. Kim KM, Yoo GD, Heo W, et al. TAZ stimulates exercise-induced muscle satellite cell activation via Pard3–p38 MAPK–TAZ signalling axis. Journal of Cachexia, Sarcopenia and Muscle. 2023; 14(6): 2733-2746. doi: 10.1002/jcsm.13348
26. Andrade DR, Pinto KC, de Castro JS, et al. Oxygen supplementation increases the total work and muscle damage markers but reduces the inflammatory response in COPD patients. Respiratory Physiology & Neurobiology. 2020; 280: 103475. doi: 10.1016/j.resp.2020.103475
27. Zabriskie HA, Blumkaitis JC, Moon JM, et al. Yeast Beta-Glucan Supplementation Downregulates Markers of Systemic Inflammation after Heated Treadmill Exercise. Nutrients. 2020; 12(4): 1144. doi: 10.3390/nu12041144
28. Powers SK, Lategan-Potgieter R, Goldstein E. Exercise-induced Nrf2 activation increases antioxidant defenses in skeletal muscles. Free Radical Biology and Medicine. 2024; 224: 470-478. doi: 10.1016/j.freeradbiomed.2024.07.041
29. Khodabukus A. Tissue-Engineered Skeletal Muscle Models to Study Muscle Function, Plasticity, and Disease. Frontiers in Physiology. 2021; 12. doi: 10.3389/fphys.2021.619710
30. Rakoczy J, Kryściak K, Drzymała-Celichowska H, et al. Biomechanical conditioning of the motor unit transitory force decrease following a reduction in stimulation rate. BMC Sports Science, Medicine and Rehabilitation. 2020; 12(1). doi: 10.1186/s13102-020-00208-6
31. Zhao C, Gong Y, Zheng L, et al. Whey protein hydrolysate enhances exercise endurance, regulates energy metabolism, and attenuates muscle damage in exercise mice. Food Bioscience. 2023; 52: 102453. doi: 10.1016/j.fbio.2023.102453
32. Zhang M, Alemasi A, Zhao M, et al. Exercise Training Attenuates Acute β-Adrenergic Receptor Activation-Induced Cardiac Inflammation via the Activation of AMP-Activated Protein Kinase. International Journal of Molecular Sciences. 2023; 24(11): 9263. doi: 10.3390/ijms24119263
33. Real-Hohn A, Navegantes C, Ramos K, et al. The synergism of high-intensity intermittent exercise and every-other-day intermittent fasting regimen on energy metabolism adaptations includes hexokinase activity and mitochondrial efficiency. PLOS ONE. 2018; 13(12): e0202784. doi: 10.1371/journal.pone.0202784
34. Pellegrin M, Bouzourène K, Aubert JF, et al. Impact of aerobic exercise type on blood flow, muscle energy metabolism, and mitochondrial biogenesis in experimental lower extremity artery disease. Scientific Reports. 2020; 10(1). doi: 10.1038/s41598-020-70961-8
35. Tanikawa C, Zhang Y zhong, Yamamoto R, et al. The Transcriptional Landscape of p53 Signalling Pathway. EBioMedicine. 2017; 20: 109-119. doi: 10.1016/j.ebiom.2017.05.017
36. Xia J, Zhang T, Sun Y, et al. Suppression of neuronal CDK9/p53/VDAC signaling provides bioenergetic support and improves post-stroke neuropsychiatric outcomes. Cellular and Molecular Life Sciences. 2024; 81(1). doi: 10.1007/s00018-024-05428-4
37. Kehl KL, Greenwald S, Chamoun NG, et al. Association Between First-Line Immune Checkpoint Inhibition and Survival for Medicare-Insured Patients with Advanced Non–Small Cell Lung Cancer. JAMA Network Open. 2021; 4(5): e2111113. doi: 10.1001/jamanetworkopen.2021.11113
38. Kim KH, Lee MS. Autophagy as a crosstalk mediator of metabolic organs in regulation of energy metabolism. Reviews in Endocrine and Metabolic Disorders. 2013; 15(1): 11-20. doi: 10.1007/s11154-013-9272-6
39. Moriarty TA, Bourbeau K, Mermier C, et al. Exercise-Based Cardiac Rehabilitation Improves Cognitive Function Among Patients with Cardiovascular Disease. Journal of Cardiopulmonary Rehabilitation and Prevention. 2020; 40(6): 407-413. doi: 10.1097/hcr.0000000000000545
40. Mahmoud AM, Gonçalves da Silva AL, André LD, et al. Effects of Exercise Mode on Improving Cardiovascular Function and Cardiorespiratory Fitness After Bariatric Surgery. American Journal of Physical Medicine & Rehabilitation. 2022; 101(11): 1056-1065. doi: 10.1097/phm.0000000000001946
41. Duan X, Liu R, Xi Y, et al. The mechanisms of exercise improving cardiovascular function by stimulating Piezo1 and TRP ion channels: a systemic review. Molecular and Cellular Biochemistry. 2024; 480(1): 119-137. doi: 10.1007/s11010-024-05000-5
42. Eroglu E, Hallström S, Bischof H, et al. Real-time visualization of distinct nitric oxide generation of nitric oxide synthase isoforms in single cells. Nitric Oxide. 2017; 70: 59-67. doi: 10.1016/j.niox.2017.09.001

43. Erickson AG, Motta A, Kastriti ME, et al. Motor innervation directs the correct development of the mouse sympathetic nervous system. Nature Communications. 2024; 15(1). doi: 10.1038/s41467-024-51290-0
44. Ahn N, Kim K. High-density lipoprotein cholesterol (HDL-C) in cardiovascular disease: effect of exercise training. Integrative Medicine Research. 2016; 5(3): 212-215. doi: 10.1016/j.imr.2016.07.001
45. Anitha S, Botha R, Kane-Potaka J, et al. Can Millet Consumption Help Manage Hyperlipidemia and Obesity?: A Systematic Review and Meta-Analysis. Frontiers in Nutrition. 2021; 8. doi: 10.3389/fnut.2021.700778
46. Liu JH, Alderman BL, Song TF, et al. A randomized controlled trial of coordination exercise on cognitive function in obese adolescents. Psychology of Sport and Exercise. 2018; 34: 29-38. doi: 10.1016/j.psychsport.2017.09.003
47. Feng J, Wu Y. Interleukin-35 ameliorates cardiovascular disease by suppressing inflammatory responses and regulating immune homeostasis. International Immunopharmacology. 2022; 110: 108938. doi: 10.1016/j.intimp.2022.108938
48. Petr M, Stastny P, Zajac A, et al. The Role of Peroxisome Proliferator-Activated Receptors and Their Transcriptional Coactivators Gene Variations in Human Trainability: A Systematic Review. International Journal of Molecular Sciences. 2018; 19(5): 1472. doi: 10.3390/ijms19051472
49. Rackles E, Witting M, Forné I, et al. Reduced peroxisomal import triggers peroxisomal retrograde signaling. Cell Reports. 2021; 34(3): 108653. doi: 10.1016/j.celrep.2020.108653
50. Christofides A, Konstantinidou E, Jani C, et al. The role of peroxisome proliferator-activated receptors (PPAR) in immune responses. Metabolism. 2021; 114: 154338. doi: 10.1016/j.metabol.2020.154338
51. Staels B, Butruille L, Francque S. Treating NASH by targeting peroxisome proliferator-activated receptors. Journal of Hepatology. 2023; 79(5): 1302-1316. doi: 10.1016/j.jhep.2023.07.004
52. Law KB, Bronte-Tinkew D, Di Pietro E, et al. The peroxisomal AAA ATPase complex prevents pexophagy and development of peroxisome biogenesis disorders. Autophagy. 2017; 13(5): 868-884. doi: 10.1080/15548627.2017.1291470
53. Nebiker L, Lichtenstein E, Minghetti A, et al. Moderating Effects of Exercise Duration and Intensity in Neuromuscular vs. Endurance Exercise Interventions for the Treatment of Depression: A Meta-Analytical Review. Frontiers in Psychiatry. 2018; 9. doi: 10.3389/fpsyt.2018.00305
54. Negaresh R, Motl RW, Mokhtarzade M, et al. Effects of exercise training on cytokines and adipokines in multiple Sclerosis: A systematic review. Multiple Sclerosis and Related Disorders. 2018; 24: 91-100. doi: 10.1016/j.msard.2018.06.008
55. Al-Ibraheem AMT, Hameed AAZ, Marsool MDM, et al. Exercise-Induced cytokines, diet, and inflammation and their role in adipose tissue metabolism. Health Science Reports. 2024; 7(9). doi: 10.1002/hsr2.70034
56. Mancilha TP, Oliveira CF, Passos MCF, et al. Leptin Gene Polymorphism, Plasma Leptin Levels and Aerobic Capacity in Response to Intense Exercise. Medicine & Science in Sports & Exercise. 2019; 51(6S): 504-504. doi: 10.1249/01.mss.0000562015.69910.68
57. Fedewa MV, Hathaway ED, Ward-Ritacco CL, et al. The Effect of Exercise Training on Leptin. Medicine & Science in Sports & Exercise. 2017; 49(5S): 805-806. doi: 10.1249/01.mss.0000519156.44913.86
58. Asher A, Medarev S, Faal M, et al. Cardiac Adaptions to Exercise Training in Mice Lacking Adiponectin. Physiology. 2023; 38(S1). doi: 10.1152/physiol.2023.38.s1.5732869
59. Sun L, Lv Y, Tian J, et al. Regular Swimming Exercise Attenuated Neuroma Pain in Rats: Involvement of Leptin and Adiponectin. The Journal of Pain. 2019; 20(9): 1112-1124. doi: 10.1016/j.jpain.2019.02.097
60. Park H, Trupiano SP, Medarev SL, et al. Aerobic exercise training-induced bone and vascular adaptations in mice lacking adiponectin. Bone. 2025; 190: 117272. doi: 10.1016/j.bone.2024.117272
61. Perduca M, Nicolis S, Mannucci B, et al. Human plasma retinol-binding protein (RBP4) is also a fatty acid-binding protein. Biochimica et Biophysica Acta (BBA) - Molecular and Cell Biology of Lipids. 2018; 1863(4): 458-466. doi: 10.1016/j.bbalip.2018.01.010
62. Hadjispyrou S, Giannopoulos A, Philippou A, et al. Mitochondrial Dysfunction and Sarcopenic Obesity: The Role of Exercise. Journal of Clinical Medicine. 2023; 12(17): 5628. doi: 10.3390/jcm12175628
63. Shao H, Zhang H, Jia D. The Role of Exerkines in Obesity-Induced Disruption of Mitochondrial Homeostasis in Thermogenic Fat. Metabolites. 2024; 14(5): 287. doi: 10.3390/metabo14050287
64. Kras Ka, Hoffman N, Roust LR, et al. Adenosine Triphosphate Production of Muscle Mitochondria after Acute Exercise in Lean and Obese Humans. Medicine & Science in Sports & Exercise. 2019; 51(3): 445-453. doi: 10.1249/mss.0000000000001812
65. Mozaffaritabar S, Koltai E, Zhou L, et al. PGC-1α activation boosts exercise-dependent cellular response in the skeletal

muscle. Journal of Physiology and Biochemistry. 2024; 80(2): 329-335. doi: 10.1007/s13105-024-01006-1
66. Lin ML, Hsu CC, Fu TC, et al. Exercise Training Improves Mitochondrial Bioenergetics of Natural Killer Cells. Medicine & Science in Sports & Exercise. 2021; 54(5): 751-760. doi: 10.1249/mss.0000000000002842
67. Broome SC, Pham T, Braakhuis AJ, et al. MitoQ supplementation augments acute exercise-induced increases in muscle PGC1α mRNA and improves training-induced increases in peak power independent of mitochondrial content and function in untrained middle-aged men. Redox Biology. 2022; 53: 102341. doi: 10.1016/j.redox.2022.102341
68. Long YF, Chow SKH, Cui C, et al. Regulation of mitochondrial dynamic equilibrium by physical exercise in sarcopenia: A systematic review. Journal of Orthopaedic Translation. 2022; 35: 37-52. doi: 10.1016/j.jot.2022.06.003
69. Kim MB, Kim C, Hwang JK. StandardizedSiegesbeckia orientalisL. Extract Increases Exercise Endurance Through Stimulation of Mitochondrial Biogenesis. Journal of Medicinal Food. 2019; 22(11): 1159-1167. doi: 10.1089/jmf.2019.4485
70. Bryan K, McGivney BA, Farries G, et al. Equine skeletal muscle adaptations to exercise and training: evidence of differential regulation of autophagosomal and mitochondrial components. BMC Genomics. 2017; 18(1). doi: 10.1186/s12864-017-4007-9
71. Liu J, Aylor KW, Liu Z. 345-OR: Exercise and Liraglutide Synergistically Attenuate Vascular Inflammation and Enhance Metabolic Insulin Action in Diet-Induced Obesity. Diabetes. 2023; 72(Supplement_1). doi: 10.2337/db23-345-or
72. Ma K, Xue B, Chu R, et al. A Widespread Radical-Mediated Glycolysis Pathway. Journal of the American Chemical Society. 2024; 146(38): 26187-26197. doi: 10.1021/jacs.4c07718
73. Zeng C, Wu J, Li J. Pyruvate Kinase M2: A Potential Regulator of Cardiac Injury Through Glycolytic and Non-glycolytic Pathways. Journal of Cardiovascular Pharmacology. 2024; 84(1): 1-9. doi: 10.1097/fjc.0000000000001568
74. Neeland IJ, Hughes C, Ayers CR, et al. Effects of visceral adiposity on glycerol pathways in gluconeogenesis. Metabolism. 2017; 67: 80-89. doi: 10.1016/j.metabol.2016.11.008
75. Kang S, Park KM, Sung KY, et al. Effect of Resistance Exercise on the Lipolysis Pathway in Obese Pre- and Postmenopausal Women. Journal of Personalized Medicine. 2021; 11(9): 874. doi: 10.3390/jpm11090874
76. Redkva PE, Miyagi WE, Milioni F, et al. Anaerobic capacity estimated by the sum of both oxygen equivalents from the glycolytic and phosphagen pathways is dependent on exercise mode: Running versus cycling. PLOS ONE. 2018; 13(9): e0203796. doi: 10.1371/journal.pone.0203796
77. Galvan-Alvarez V, Gallego-Selles A, Martinez-Canton M, et al. Antioxidant enzymes and Nrf2/Keap1 in human skeletal muscle: Influence of age, sex, adiposity and aerobic fitness. Free Radical Biology and Medicine. 2023; 209: 282-291. doi: 10.1016/j.freeradbiomed.2023.10.393
78. Shin YA, Lee JH, Song W, et al. Exercise training improves the antioxidant enzyme activity with no changes of telomere length. Mechanisms of Ageing and Development. 2008; 129(5): 254-260. doi: 10.1016/j.mad.2008.01.001
79. Powers SK, Goldstein E, Schrager M, et al. Exercise Training and Skeletal Muscle Antioxidant Enzymes: An Update. Antioxidants. 2022; 12(1): 39. doi: 10.3390/antiox12010039
80. Farinha JB, De Carvalho NR, Steckling FM, et al. An active lifestyle induces positive antioxidant enzyme modulation in peripheral blood mononuclear cells of overweight/obese postmenopausal women. Life Sciences. 2015; 121: 152-157. doi: 10.1016/j.lfs.2014.11.022
81. Chang S, Chen Y, Chang W, et al. Increase of anti-oxidation by exercise in the liver of obese Zucker rats. Clinical and Experimental Pharmacology and Physiology. 2004; 31(8): 506-511. doi: 10.1111/j.1440-1681.2004.04035.x
82. Jiang M, Wang Y, Wang J, et al. The etiological roles of miRNAs, lncRNAs, and circRNAs in neuropathic pain: A narrative review. Journal of Clinical Laboratory Analysis. 2022; 36(8). doi: 10.1002/jcla.24592
83. Park KS, Nickerson B, Norris J, et al. Effects of Aerobic Exercise Training Intensity on Inflammatory Cytokines in Obese Hispanic Females. Medicine & Science in Sports & Exercise. 2017; 49(5S): 485-486. doi: 10.1249/01.mss.0000518224.04443.ff
84. Lo K, Wong M, Khalechelvam P, et al. Waist-to-height ratio, body mass index and waist circumference for screening paediatric cardio-metabolic risk factors: a meta-analysis. Obesity Reviews. 2016; 17(12): 1258-1275. doi: 10.1111/obr.12456
85. Iyengar NM, Zhou XK, Mendieta H, et al. Effects of Adiposity and Exercise on Breast Tissue and Systemic Metabo-Inflammatory Factors in Women at High Risk or Diagnosed with Breast Cancer. Cancer Prevention Research. 2021; 14(5): 541-550. doi: 10.1158/1940-6207.capr-20-0507
86. Roh HT, Cho SY, So WY. A Cross-Sectional Study Evaluating the Effects of Resistance Exercise on Inflammation and

Neurotrophic Factors in Elderly Women with Obesity. Journal of Clinical Medicine. 2020; 9(3): 842. doi: 10.3390/jcm9030842

87. Wang S, Zhou H, Zhao C, et al. Effect of Exercise Training on Body Composition and Inflammatory Cytokine Levels in Overweight and Obese Individuals: A Systematic Review and Network Meta-Analysis. Frontiers in Immunology. 2022; 13. doi: 10.3389/fimmu.2022.921085
88. Son WH, Park HT, Jeon BH, et al. Moderate intensity walking exercises reduce the body mass index and vascular inflammatory factors in postmenopausal women with obesity: a randomized controlled trial. Scientific Reports. 2023; 13(1). doi: 10.1038/s41598-023-47403-2
89. Ermetici F, Zelaschi RF, Briganti S, et al. Association between a school-based intervention and adiposity outcomes in adolescents: The Italian "EAT" project. Obesity. 2016; 24(3): 687-695. doi: 10.1002/oby.21365
90. Sgambato MR, Cunha DB, Souza BSN, et al. Effectiveness of school–home intervention for adolescent obesity prevention: parallel school randomised study. British Journal of Nutrition. 2019; 122(9): 1073-1080. doi: 10.1017/s0007114519001818
91. Caron T, Bernard P, Gadais T. Clinical and school-based intervention strategies for youth obesity prevention: A systematic review. Frontiers in Sports and Active Living. 2023; 4. doi: 10.3389/fspor.2022.906857
92. Omorou AY, Langlois J, Legrand K, et al. Impact d'une intervention en milieu scolaire sur le gradient social de corpulence chez l'adolescent: l'étude PRALIMAP-INES. Revue d'Épidémiologie et de Santé Publique. 2017; 65: S52. doi: 10.1016/j.respe.2017.03.014
93. MacKay M, Mathis S. School-Based Versus Family-Based Interventions for the Reversal and Prevention of Obesity Among Hispanic Youth: a Review of the Literature. Journal of Racial and Ethnic Health Disparities. 2023; 11(4): 2338-2345. doi: 10.1007/s40615-023-01701-7
94. Buru K, Emeto TI, Malau-Aduli AEO, et al. The Efficacy of School-Based Interventions in Preventing Adolescent Obesity in Australia. Healthcare. 2020; 8(4): 514. doi: 10.3390/healthcare8040514
95. Sgambato MR, Cunha DB, Henriques VT, et al. PAAPPAS community trial protocol: a randomized study of obesity prevention for adolescents combining school with household intervention. BMC Public Health. 2016; 16(1). doi: 10.1186/s12889-016-3473-3
96. Perrino T, Brincks AM, Estrada Y, et al. Reducing Screen-Based Sedentary Behavior Among Overweight and Obese Hispanic Adolescents Through a Family-Based Intervention. Journal of Physical Activity and Health. 2022; 19(7): 509-517. doi: 10.1123/jpah.2022-0050
97. Wei YD, Xiao W, Garcia I. Neighborhood Environment, Active Commute, and Healthy BMI in Adolescents. Sustainability. 2021; 13(15): 8286. doi: 10.3390/su13158286
98. Kumar S, Croghan IT, Biggs BK, et al. Family-Based Mindful Eating Intervention in Adolescents with Obesity: A Pilot Randomized Clinical Trial. Children. 2018; 5(7): 93. doi: 10.3390/children5070093
99. Moores CJ, Bell LK, Miller J, et al. A systematic review of community‐based interventions for the treatment of adolescents with overweight and obesity. Obesity Reviews. 2018; 19(5): 698-715. doi: 10.1111/obr.12660
100. Wieland ML, Biggs BK, Brockman TA, et al. Club Fit: Development of a Physical Activity and Healthy Eating Intervention at a Boys & Girls Club After School Program. The Journal of Primary Prevention. 2020; 41(2): 153-170. doi: 10.1007/s10935-020-00582-4
101. Boone-Heinonen J, Gordon-Larsen P, Adair LS. Obesogenic Clusters: Multidimensional Adolescent Obesity-related Behaviors in the U.S. Annals of Behavioral Medicine. 2008; 36(3): 217-230. doi: 10.1007/s12160-008-9074-3