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Revisiones

Molecular mechanisms of the role of aerobic and resistance exercise in increasing adiponectin levels in humans as a control of energy expenditure: a systematic review

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ABSTRACT

Background: Insulin resistance, chronic inflammation, and impaired energy balance are closely linked to obesity and type 2 diabetes mellitus, two increasingly common metabolic diseases. In these conditions, decreased adiponectin levels contribute to metabolic dysfunction by disrupting glucose regulation and fatty acid oxidation. Exercise has been shown to be a non-pharmacological therapy that increases adiponectin.

Objectives: The aim of this study was to determine the effect of aerobic and resistance physical exercise on adiponectin levels.

Methods: A number of journal databases, including Science Direct, Web of Science, Pubmed, and Scopus, were examined for this study. Among the factors taken into account in this analysis were studies on the effects of resistance and aerobic exercise on adiponectin levels that were published between 2020 and 2025. Papers that did not fit the inclusion criteria such as non-experimental research or articles not found in our pre-established databases were not included in this systematic review. Using databases from Pubmed, Science Direct, Web of Science, and Scopus, 689 publications in all were located. Ten carefully chosen, peer-reviewed studies discuss the necessity of this systemic change. Standard operating procedures for this inquiry were established using the Preferred Standards for Reporting Systematics and Meta-Analysis (PRISMA).

Results: The study's findings demonstrated both resistance and aerobic exercise raised people's adiponectin levels noticeably.

Conclusion: Resistance training and aerobic exercise have been demonstrated to significantly raise human adiponectin levels. Improving insulin sensitivity, controlling blood sugar, and having anti-inflammatory properties are all made possible by increasing adiponectin. It has been demonstrated that both forms of exercise have a beneficial effect on general metabolic health. Thus, consistent aerobic and resistance training could be a useful tactic for managing and preventing metabolic diseases.

Keywords: Aerobic; Resistance; Physical Exercise; Adiponectin; Good Health and Well-being.

Mecanismos moleculares del papel del ejercicio aeróbico y de resistencia en el aumento de los niveles de adiponectina en humanos como control del gasto energético: revisión sistemática

RESUMEN

Antecedentes: La resistencia a la insulina, la inflamación crónica y el deterioro del equilibrio energético están estrechamente relacionados con la obesidad y la diabetes mellitus tipo 2, dos enfermedades metabólicas cada vez más frecuentes. En estas condiciones, la disminución de los niveles de adiponectina contribuye a la disfunción metabólica al alterar la regulación de la glucosa y la oxidación de ácidos grasos. Se ha demostrado que el ejercicio es una terapia no farmacológica capaz de aumentar la adiponectina.

Objetivos: El objetivo de este estudio fue determinar el efecto del ejercicio físico aeróbico y de resistencia sobre los niveles de adiponectina.

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Métodos: Para este estudio se revisaron varias bases de datos científicas, entre ellas ScienceDirect, Web of Science, PubMed y Scopus. Entre los factores considerados en este análisis se incluyeron estudios sobre los efectos del ejercicio de resistencia y aeróbico sobre los niveles de adiponectina publicados entre 2020 y 2025. Los artículos que no cumplían los criterios de inclusión, como investigaciones no experimentales o trabajos no encontrados en las bases de datos previamente establecidas, no fueron incluidos en esta revisión sistemática. A través de las bases de datos PubMed, ScienceDirect, Web of Science y Scopus se localizaron un total de 689 publicaciones. Diez estudios cuidadosamente seleccionados y revisados por pares abordaron la necesidad de este cambio sistémico. Los procedimientos operativos estándar de esta investigación se establecieron siguiendo las directrices Preferred Reporting Items for Systematic Reviews and Meta-Analyses, PRISMA.

Resultados: Los hallazgos del estudio demostraron que tanto el ejercicio de fuerza como el ejercicio aeróbico aumentaron de forma notable los niveles de adiponectina en humanos.

Conclusión: Se ha demostrado que el entrenamiento de resistencia y el ejercicio aeróbico aumentan significativamente los niveles de adiponectina en humanos. El incremento de la adiponectina contribuye a mejorar la sensibilidad a la insulina, controlar la glucemia y ejercer propiedades antiinflamatorias. Se ha demostrado que ambas formas de ejercicio tienen un efecto beneficioso sobre la salud metabólica general. Por tanto, la práctica regular de ejercicio aeróbico y de resistencia podría constituir una estrategia útil para el manejo y la prevención de enfermedades metabólicas.

Palabras clave: aeróbico; resistencia; ejercicio físico; adiponectina; salud y bienestar.

Mecanismos moleculares do papel do exercício aeróbio e de resistência no aumento dos níveis de adiponectina em humanos como controlo do gasto energético: revisão sistemática

RESUMO

Antecedentes: A resistência à insulina, a inflamação crónica e o comprometimento do equilíbrio energético estão estreitamente associados à obesidade e à diabetes mellitus tipo 2, duas doenças metabólicas cada vez mais frequentes. Nestas condições, a diminuição dos níveis de adiponectina contribui para a disfunção metabólica ao alterar a regulação da glicose e a oxidação dos ácidos gordos. O exercício tem demonstrado ser uma terapia não farmacológica capaz de aumentar a adiponectina.

Objetivos: O objetivo deste estudo foi determinar o efeito do exercício físico aeróbio e de resistência nos níveis de adiponectina.

Métodos: Para este estudo foram analisadas várias bases de dados científicas, incluindo ScienceDirect, Web of Science, PubMed e Scopus. Entre os fatores considerados nesta análise incluíram-se estudos sobre os efeitos do exercício de resistência e aeróbio nos níveis de adiponectina publicados entre 2020 e 2025. Os artigos que não cumpriam os critérios de inclusão, como estudos não experimentais ou artigos não encontrados nas bases de dados previamente estabelecidas, não foram incluídos nesta revisão sistemática. Através das bases de dados PubMed, ScienceDirect, Web of Science e Scopus, foram localizadas 689 publicações no total. Dez estudos cuidadosamente selecionados e revistos por pares abordaram a necessidade desta mudança sistémica. Os procedimentos operacionais padrão desta investigação foram estabelecidos de acordo com as diretrizes Preferred Reporting Items for Systematic Reviews and Meta-Analyses, PRISMA.

Resultados: Os resultados do estudo demonstraram que tanto o exercício de resistência como o exercício aeróbio aumentaram de forma evidente os níveis de adiponectina em humanos.

Conclusão: Foi demonstrado que o treino de resistência e o exercício aeróbio aumentam significativamente os níveis de adiponectina em humanos. O aumento da adiponectina contribui para melhorar a sensibilidade à insulina, controlar a glicemia e exercer propriedades anti-inflamatórias. Demonstrou-se que ambas as formas de exercício têm um efeito benéfico na saúde metabólica geral. Assim, a prática regular de exercício aeróbio e de resistência poderá constituir uma estratégia útil para a gestão e prevenção de doenças metabólicas.

Palavras-chave: aeróbio; resistência; exercício físico; adiponectina; saúde e bem-estar.

Introduction

Currently the fifth most prevalent primary cause of death worldwide, obesity is a rapidly expanding global health concern that affects people of all ages and genders[1]. One of the most urgent global public health issues of the twenty-first century is obesity. Approximately 43% of adults are overweight or obese by 2022, and over 1 billion people worldwide roughly one in eight adults are obese (body mass index ≥30)[2]. Since 1990, the prevalence of obesity has more than doubled in adults and more than quadrupled in youngsters[2]. Experts have described obesity as a global epidemic or perhaps a "pandemic" due to these striking patterns[3]. In other words, people in almost every part of the world are now affected by excess body fat; it is no longer a problem that only exists in a select few nations.

One of the biggest organs in the human body is adipose tissue. Abnormal adipose deposition and adipocyte expansion in a number of anatomical places, including the subcutaneous and visceral areas like the intra-abdominal region, are characteristics of obesity[4,5]. In addition to storing extra energy as fat, visceral adipose tissue (VAT) has a significant role in energy regulation[6]. An imbalance in the availability of substrates can cause mitochondrial malfunction, which may have an effect on oxidative respiration and energy production[7]. Despite having relatively low mitochondrial density, adipocytes are crucial for a number of cellular metabolic processes[7]. Therefore, via changing adipogenesis as well as the lipogenic and lipolytic pathways, mitochondrial dysfunction invariably impacts adipocyte dynamics[8]. When compared to those who are not obese, adipocytes in obese people have mitochondria with an ambiguous inner membrane, a lesser capacity to produce energy, and a lower capacity to oxidize fatty acids[9].

Apart from storing energy, adipose tissue also functions as an active endocrine organ. By secreting bioactive adipokines like leptin, resistin, and adiponectin, this tissue controls energy balance[10], which functions by signaling pathways that are endocrine, paracrine, and autocrine[11]. Additionally, these adipokines affect a number of physiological functions, including immune system and

Table 1. Inclusion criteria

Web search engines	**Pubmed, Science Direct, Scopus, and Web of Science**
Publishing period	2020 – 2025
Keyword	Aerobic and resistance training, adiponectin, and obesity
Language	English
Type of article	Original research article
Full Text	Articles matched the purpose and/or topic of the research.

cardiovascular function, metabolic regulation, and inflammatory response modulation[12]. Four separate research teams identified adiponectin, a protein hormone released by adipose tissue, in the late 1990s[12]. Its clinical significance was initially unknown. When it was shown that low serum levels were associated with obesity and increased visceral fat, interest in this adipokine grew[13]. This correlation highlights the preventive effect of adiponectin and its possible involvement in the emergence of metabolic diseases associated with obesity[14]. A higher risk of metabolic syndrome components is linked to lower adiponectin levels, and genetic factors seem to be crucial in controlling these concentrations[15]. One of the main causes of obesity and associated metabolic diseases is the dysregulation of adipocytokines, including resistin, leptin, and adiponectin[16]. Adiponectin is one of these factors that has positive regulatory effects on obesity and associated metabolic syndrome. These effects include coordinating the expansion and vascularization of adipose tissue, lowering inflammation, boosting metabolic flexibility, improving insulin sensitivity, modulating skeletal muscle, controlling cardiovascular disease, controlling liver function, and more[16].

It is well recognized that exercise and physical activity are crucial for enhancing general health and physical fitness[17,18]. Both are well known to be successful therapeutic and preventative strategies for a range of illnesses. Regular, moderate exercise in particular seems to be a particularly useful strategy for preventing metabolic disorders linked to obesity[19]. High-intensity exercise, either by itself or in conjunction with weight training, has been demonstrated in a number of recent studies to enhance inflammatory, immunological, and metabolic processes[20,21]. Specifically, it has been noted that during acute physical activity, tumor necrosis factor alpha expression declines while interleukin 6 and interleukin 10 levels ris[22]. The literature has a number of scientific research about changes in the expression of different cytokines after exercise regimens. These findings suggest that numerous organs and tissues contribute to the physiological changes brought on by exercise by secreting cytokines[22].

Additionally, certain adipocytokines that are secreted by adipose tissue play a role in controlling inflammation and energy metabolism[23]. Indeed, alterations in the expression of adipokines, particularly adiponectin, indicate the involvement of endocrine function, according to data from the literature[24]. Adipose tissue is the specialized site for the synthesis and release of adiponectin, a polypeptide of 244 amino acids, in the form of trimers (low molecular weight, LMW), hexamers (middle molecular weight, MMW), and high molecular weight oligomers (HMW)[25]. Adiponectin is an endocrine mediator that enhances energy metabolism and insulin sensitivity[25]. According to data from the literature, HMW oligomers are the most potent type of adipocytokines, which are involved in energy balance and body weight regulation[26]. Adiponectin, which is frequently assessed in research involving obese participants, has been found to be negatively correlated with inflammation, body fat percentage, and body mass index (BMI)[25].

According to the majority of research, in healthy men and women, serum adiponectin levels tend to rise with age[27,28], however, some research has indicated a decline[28] or no change[29]. In a very large population, Obata et al. also found a significant positive association between serum adiponectin levels and age in healthy subjects[26]. Numerous studies have demonstrated that both acute and regular exercise can raise blood levels of adiponectin[30]. There is ongoing discussion over the significance of the association between adiponectin levels and exercise. Furthermore, studies continue to disagree about the precise molecular processes by which regular exercise influences adiponectin levels. Thus, the purpose of this research was to ascertain how resistance and aerobic training affected adiponectin levels. Additionally, we sought to thoroughly investigate the underlying chemical pathways.

Materials and methods

Study Design

This study design is a systematic review, reviewing scientific papers according to criteria established by the researchers. The selection of papers regarding the effects of aerobic and resistance training on adiponectin levels in humans was determined by reviewing the scientific literature. The following search engines were used to locate scientific literature: Web of Science, Pubmed, Science Direct, and Scopus. The search terms used were aerobic and resistance training, adiponectin, and obesity. Publications were selected based on the following inclusion criteria: year of publication, experimental study, and articles related to humans.

Eligibility criteria

The study inclusion criteria were established by searching existing databases for material published between 2020 and 2025. Experimental studies on increasing adiponectin levels after aerobic and resistance exercise were also included. Among the search terms used was adiponectin levels. Furthermore, our study excluded papers that did not meet scientific validity standards or were not included in leading search indexes such as Scopus, Web of Science, PubMed, or Science Direct. Therefore, we screened the selected papers using our pre-established inclusion criteria.

Procedure

The full text, abstract, and title of each publication were added to the Mendeley database after review and confirmation. Using Scopus, Science Direct, Pubmed, and Web of Science, 689 publications were identified during the initial screening phase. 370 eligible papers were selected for the second screening phase after identifying duplicate articles and addressing the reasons behind title discrepancies. In the next phase, 180 papers were identified based on the concordance of the reviewed titles, abstracts, and keywords. After reviewing each paper, we determined that the study must be experimental in nature, the parameter used must be the biomarker adiponectin, the intervention must be aerobic or resistance exercise, and the samples used must be human. This was the final step in the process. We screened these publications to identify those that met our pre-defined inclusion criteria. After a rigorous review and observation process, ten papers that met the inclusion criteria were selected for analysis. The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) criteria were followed for this study. This study reviewed previous literature that met bioethical and ethical standards.

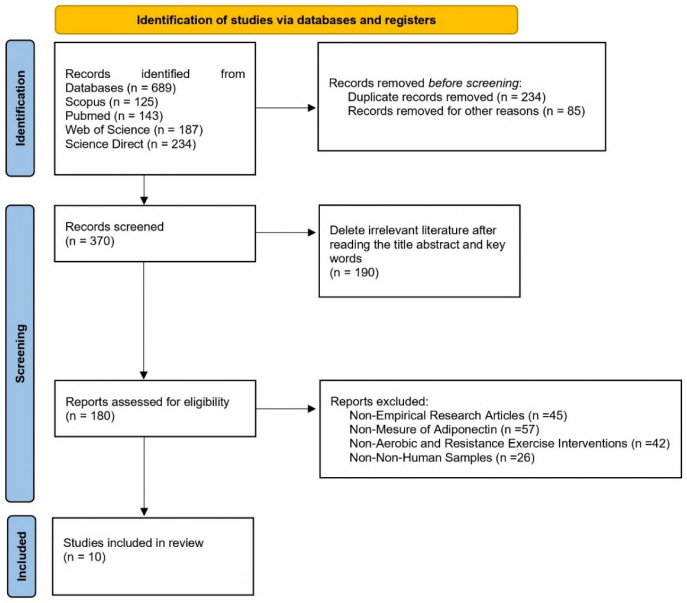

Figure 1. PRISMA flowchart of the article selection process

Results

Discussion

This study sought to ascertain how aerobic exercise affected the rise in adiponectin levels in people. The study's findings showed that adiponectin levels were significantly raised by aerobic activity. Previous studies have demonstrated a considerable increase in adiponectin levels in athletes who received cardiopulmonary exercise therapies and worked out on a bicycle ergometer until exhaustion[19]. Furthermore, following a 12-week intervention, other research has demonstrated that both HIIT and MICT exercise have an effect on raising adiponectin levels[31]. These results contradict the findings of Swisher et al.[39], They found that after 12 weeks of moderate-intensity aerobic exercise, women with breast cancer had a non-significant increase in blood adiponectin. Dieli-Conwright et al., however[40], demonstrated a noteworthy rise in adiponectin levels following a 16-week intervention in breast cancer that included both aerobic and strength training. Additionally, they looked at leptin levels, which also significantly decreased. This also has a beneficial effect on obese patients; Specifically, a 12-week resistance training regimen consisting of three sessions per week markedly raised adiponectin levels. Thus, it can be suggested that aerobic exercise helps avoid metabolic syndrome in obese people by raising adiponectin levels[32].

Additionally, resistance training has a beneficial therapeutic impact, especially for patients who are fat. Adiponectin levels were shown to be substantially elevated in obese patients who underwent three 50-minute resistance training sessions per week for a period of 12 weeks[33]. Additionally, the results of other studies on obese patients showed that walking, running, or a combination of the two, done three times a week for 24 weeks in a row, had a significant effect on raising adiponectin levels in addition to VO2max[30]. In addition to obesity, it was discovered that three times a week for four weeks, patients with type 2 diabetes mellitus who had 30-minute moderate-intensity treadmill workouts with rising pace and progressive incline significantly increased their levels of adiponectin[34]. Resistance training performed three times a week for 12 weeks with a protocol that included resistance training to increase endurance (low intensity), increase volume (moderate intensity), or increase strength (high intensity) had a significant impact on raising adiponectin levels, according to another study in patients with type 2 diabetes mellitus[35]. For individuals who are obese, exercise is an alternative form of treatment. Prior studies have demonstrated a considerable increase in adiponectin levels following a six-month course of aerobic resistance exercise[36].

Elderly adults over 70 years of age were also significantly impacted, in addition to obesity. Over the course of a year, participants engage in a typical exercise regimen that includes resistance and aerobic training at home and at a fitness facility. Adiponectin levels increased significantly as a result of the intervention[37]. The hormone adiponectin, which is mostly released by adipocytes, is linked to the regulation of insulin, glucose consumption, fatty acid oxidation, and inflammation[41,42]. According to a prior study, individuals with diabetic nephropathy had higher urine adiponectin levels than healthy controls, which may indicate that this molecule serves as a marker of impaired kidney function[43]. Urinary adiponectin levels rose following the final strength training session in our study, but not immediately following the first. These findings imply that a longer time period is required for the systemic synthesis and excretion of adiponectin in the urine in response to strength exercise. According to our long-term findings, a 10-week time is required to increase adiponectin production, which in turn leads to increased urine excretion. Our hypothesis is that a strength training regimen can increase adiponectin production both temporarily and over time[38].

Molecular Mechanisms of Aerobic and Resistance Exercise Increase Adiponectin

The metabolic effects of exercise have been found to be significantly mediated by adiponectin and its high molecular weight isoforms[44]. Numerous physiological processes, such as exercise-induced acute muscle signaling, adipose tissue response, and hormonal control, may be responsible for the observed alterations in adiponectin isoforms. The AMP-activated protein kinase (AMPK) and peroxisome proliferator-activated receptor gamma (PPARγ) pathways are both triggered by exercise and are crucial modulators of the release of adiponectin from skeletal muscle and adipose tissue[45]. Exercise is a potent physiological stimulus for AMPK activation, particularly aerobic exercises such as cycling, swimming, and running[46,47]. We can understand the different metabolic advantages since ATP is quickly used up in muscle cells, increasing AMP and AMPK activation[48].

AMPK's function in boosting glucose absorption by encouraging the translocation of GLUT4 to the muscle cell membrane and boosting fatty acid oxidation by facilitating the transport of fatty acids to the mitochondria[49,50]. The path to human fitness requires an understanding of these metabolic advantages. Exercise-induced AMPK activation is a life-changing process that motivates us to reach our fitness objectives and push ourselves to the limit. Our endurance is greatly increased, which is evidence of the ability of AMPK activation to motivate us to pursue greater levels of fitness[51]. AMPK is often referred to as the 'master switch of metabolism' because it helps coordinate the body's response to changes in energy levels[52]. Exercise is a potent and organic AMPK activator that enhances metabolic efficiency, mitochondrial health, and body composition[47]. Following activation, AMPK will additionally promote a rise in PGC-1(α)[53].

Generally found in high-energy-demanding tissues such as the heart, muscle, and brown adipose tissue, peroxisome proliferator-activated receptor coactivator (PPAR)-γ-1α (PGC-1α) has been identified as a key regulator of metabolic regulatory pathways such as the adenosine monophosphate-activated protein kinase (AMPK)-sirtuin 1 (SIRT1)-PGC-1α pathway[54]. Without a doubt, exercise modifies the AMP-to-ATP ratio, which activates AMPK in vivo[55]. PGC-1α is mostly involved in cellular respiration and mitochondrial biosynthesis, but it has also been shown to be an essential regulator of cell proliferation and differentiation[56]. In both

Table 2. Summary of the design and intervention of the studies

Author	Design	Participants	Participants Age	Intervention	Outcome
(Mallardo, et al., 2024)[19]	Randomized controlled trials	25 male amateur athlete	20–65 years	Exercise Intervention 1. A cardiopulmonary exercise test was used to determine maximal aerobic capacity. 2. An incremental exercise test on a bicycle ergometer intended to cause volitional exhaustion. 3. During riding, respiratory gasses were constantly monitored.	Following a physical activity session, adiponectin levels dramatically increased.
(Eskandari et al., 2021)	Randomized controlled trials	45 participants	57 years	Exercise Intervention 1. For 12 weeks, the intervention groups (HIIT and MICT) used a bicycle ergometer three days a week to complete their respective training regimens. 2. The CON group kept up their way of life.	Following an exercise regimen, adiponectin levels rise dramatically.
(Bagheri et al., 2024)	Randomized controlled trials	Sixty overweight and obese men	30-31 years	Exercise Intervention 1. For 12 weeks, the resistance training regimen includes three training sessions per week.	Following a physical activity regimen, adiponectin levels dramatically increased.
(Ataeinostrat & Vandusseldrop, 2022)	Randomized controlled trials	Forty-four males with obesity	27 years	Exercise Intervention 1. For 12 weeks, each resistance training group engaged in 50 minutes of supervised activity three days a week. 2. After 12 weeks of training and at baseline, measurements were collected.	Following an exercise regimen, adiponectin levels rise dramatically.
(Marta Mallardo et al., 2023)[30]	Randomized controlled trials	Thirteen obese male subjects	18-50 years	Exercise Intervention 1. For 24 weeks in a row, the exercise is walking, running, or a mix of the two, three times a week, while enjoying their regular lives.	Following an exercise regimen, adiponectin levels rise dramatically.
(Mudjanarko et al., 2023)	Randomized controlled trials	Twenty-two participants with T2DM	46-50 years	Exercise Intervention 1. Three times a week for four weeks, the experimental group trained on a moderately intense treadmill for thirty minutes at a steady inclination and increasing speed. 2. Each week, the control group exercised alone for 150 minutes. Prior to and following the four-week program, the clinical and laboratory parameters of the participants were evaluated.	Following an exercise regimen, adiponectin levels rise dramatically.
(Saeed Abedinzadeh et al., 2025)	Randomized controlled trials	Forty-four subjects with type 2 diabetes	52 years	Exercise Intervention 1. Three resistance training protocols were used in the training intervention program, which was carried out three days a week for twelve weeks. 2. Resistance training was incorporated into the programs to enhance strength (high intensity), volume (mid intensity), or endurance (low intensity).	Following an exercise regimen, adiponectin levels rise dramatically.
(Olan et al., 2020)	Randomized controlled trials	Sixteen obese adolescent boys	13-19 years	Exercise Intervention 1. Running on an elliptical and treadmill, together with several forms of general strength training (sit-ups, back extensions, squats, and push-ups) using only their body weight, comprises the aerobic exercise program. 2. During weeks 1 through 8, the first workout consists of two sets of 15 repetitions; during weeks 9 through 24, the workout consists of three sets of 15 repetitions. These exercises include chest pulls, leg extensions, leg curls, push-ups, reverse push-ups, chest presses, biceps presses, triceps presses, and shoulder presses. 3. The duration of both exercise treatments was six months.	Following an aerobic exercise regimen, adiponectin levels rise dramatically.
(Senkus & Crowe-white, 2022)	Randomized controlled trials	163 participants	70 years	Exercise Intervention 1. For 12 months, all individuals engage in a standardized exercise regimen that includes resistance and aerobic training, both at home and at a fitness facility. 2. Participants received specific recommendations from an exercise scientist and trainer to support their weekly aerobic and resistance training goals: two resistance training sessions and 90–150 minutes of moderate-to-intense cardiovascular exercise, respectively. 3. The main muscular groups of the upper and lower extremities were the focus of the resistance training, which was finished with resistance bands. Heart rate monitors were given to participants in order to ensure that the training sessions were conducted properly.	Following physical activity intervention, the female group's levels of adiponectin dramatically increased.

Author	Design	Participants	Participants Age	Intervention	Outcome
(Henrique et al., 2025)	Randomized controlled trials	Twelve untrained young men	20-31 years	Exercise Intervention 1. The training regimen consists of three movements, each lasting five seconds (two concentric and three eccentric), with three sets of 65% repetition maximum (1MR) and a 90-second rest in between. 2. For ten weeks, training sessions were conducted three times a week.	Following an exercise regimen, adiponectin levels rise dramatically.

human and rodent muscle, exercise dramatically increases PGC-1α overexpression[57], which, by changing the extracellular matrix's composition, including the amount of fibronectin, can cause satellite cell niche reorganization and affect the proliferative output of satellite cells[58]. Running exercise has been shown to increase PGC-1α expression in the hippocampal regions of depressed rats. PGC-1α is a target of antidepressant treatment because it promotes the growth of parvalbumin-positive interneurons[59]. Mechanistically, PGC-1α activates a variety of metabolic programs in various tissues through its ability to form heteromeric complexes with many nuclear hormone receptors, such as PPARγ[60] and estrogen-related receptors (ERRs)[61].

It is interesting to note that the AMPK/SIRT1 axis, which is involved in mitochondrial signaling and promotes exercise-induced tissue regeneration, controls PGC-1α[62]. As a result, PGC-1α has been demonstrated to play a crucial role in the adaptive response to exercise and may be a major regulator of the relationship between exercise-induced regeneration and mitochondrial biogenesis. Additionally, SIRT1-mediated deacetylation of PGC-1α, which increases its transcriptional activity, promotes mitochondrial biogenesis. This alteration makes it possible for PGC-1α to interact with PPARγ/ERRα/NRF1 and increase TFAM expression, which in turn promotes respiratory chain assembly and mitochondrial DNA replication[63,64]. It is important to remember that AMPK, SIRT1, and PGC-1α are all part of a complicated feedback system[65]. While PGC-1α creates a positive feedback loop by regulating SIRT1 expression and activity, AMPK and SIRT1 reciprocally reinforce each other's activity[66,67]. To boost PGC-1α's stability and transcriptional activity, AMPK phosphorylates it directly in addition to indirectly through SIRT1[68].

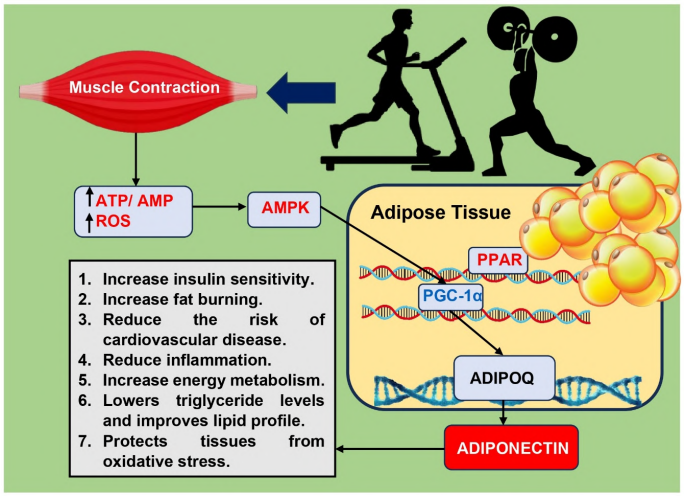

Figure 2. Molecular Mechanisms of Aerobic and Resistance Exercise Increase Adiponectin Levels

The regulation of metabolic homeostasis is significantly influenced by adiponectin[25]. Many aspects of the metabolic syndrome, including obesity, type 2 diabetes mellitus, hypertension, atherosclerosis, and coronary artery disease, have been closely linked to decreased levels of these adipokines in circulation[69]. Adiponectin expression and secretion are influenced greatly by a number of modifiable and non-modifiable factors, including age, sex, ethnicity, body mass index, nutrition, and degree of physical activity, in addition to genetic factors that contribute to individual variance in its concentration[70]. A crucial transcriptional coactivator, PGC-1α aids in the regulation of genetic programs that govern adipocyte activity, mitochondrial biogenesis, and energy metabolism. Increased PGC-1α expression in adipocytes has been demonstrated to modulate the expression profile of several important metabolic genes, including adiponectin (ADIPOQ), a well-known adipokine with anti-inflammatory and insulin-sensitizing effects, even though basal PGC-1α expression is relatively low in white adipose tissue compared to tissues such as muscle or brown adipose tissue[71]. The gene ADIPOQ causes an increase in adiponectin[12]. As a key regulator of glucose metabolism, adiponectin reduces inflammation and improves insulin sensitivity[72]. Research indicates that elevated levels of circulating adiponectin enhance insulin sensitivity[72,73], hence perhaps lowering the risk of type 2 diabetes[72].

Strength and Limitations

One benefit of this systematic review is that it eliminates the possibility of unclear cause-and-effect correlations by concentrating only on randomized controlled trials, which are the most trustworthy type of scientific data. Additionally, the human-focused samples that were gathered yielded consistent data and were not combined with samples from other categories, such as animal samples.

The dearth of knowledge regarding how exercise, especially aerobic and resistance training, can raise adiponectin levels is one of the limitations we found. In order to better understand how aerobic and resistance exercise impacts adiponectin levels and mitigates the negative impacts of metabolic illness, this study is deemed important. Resistance and aerobic exercise may help avoid metabolic disorders in general populations, including individuals with diabetes mellitus and obesity. This might, however, have to do with their undetermined effective duration and intensity. Therefore, more experimental research is required to ascertain the best time and intensity for raising human adiponectin levels.

Conclusions

According to the study's findings, both resistance and aerobic exercise help raise adiponectin levels in people, which helps control energy consumption and enhance metabolism. Molecular processes like AMPK activation and PPAR-γ modulation, which promote insulin sensitivity and fatty acid oxidation, are responsible for this rise in adiponectin. Although results may differ based on intensity, duration, and personal traits, both aerobic and resistance training showed beneficial effects. These results support exercise's importance as a non-pharmacological strategy for preventing metabolic diseases and obesity. However, the examined studies' variability and methodological differences suggest that more research with more consistent designs is required. All things considered, regular exercise has been demonstrated to be a successful tactic for boosting adiponectin and maintaining the body's energy balance.

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Conflicts of Interest

The authors declare no conflict of interest.

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