Increased adenosine monophosphate–activated protein kinase activity in mice with physical exercise intervention: a systematic review

Aumento de la actividad de la proteína quinasa activada por monofosfato de adenosina en ratones con intervención de ejercicio físico: una revisión sistemática

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Abstract

Introduction: Physical exercise has been identified as one of the external factors that can activate AMPK, but the mechanism and effects are still unclear and need to be further explored.
Objective: The aim of this study was to examine how physical exercise increases AMPK phosphorylation from a physiological perspective.
Methodology: In this systematic review investigation, we searched literature databases such as MEDLINE-Pubmed, Web of Science, Scopus, and Science Direct. Papers published in the last five years that discussed AMPK, aerobic exercise, and high-intensity interval training met the inclusion criteria. A total of 103 published papers were found by utilizing the Web of Science, Pubmed, and Science Direct databases. For this systematic review, ten papers that met the inclusion criteria were selected and examined. In this study, the standard operating procedure was evaluated using the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA).
Results: Exercise has been shown to increase AMPK phosphorylation.
Discussion: High-intensity exercise has a higher potential to increase AMPK than aerobic exercise through increased AMP/ATP ratio, increased metabolic stress, and mitochondrial adaptation. AMPK activation has also been associated with increased fatty acid oxidation and glucose uptake by muscle.
Conclusions: It can be concluded that physical exercise has been shown to consistently increase AMPK activation and has a positive impact on metabolic regulation. Thus, physical exercise is an effective intervention in improving metabolic function. Physical exercise has been shown to increase AMPK phosphorylation, thereby increasing GLUT4 translocation and increasing glucose uptake.

Keywords

AMPK; Physical Exercise; HIIT; GLUT4.

Resumen

Introducción: El ejercicio físico se ha identificado como uno de los factores externos que pueden activar la AMPK, pero el mecanismo y los efectos aún no están claros y requieren mayor investigación.
Objetivo: El objetivo de este estudio fue examinar cómo el ejercicio físico aumenta la fosforilación de AMPK desde una perspectiva fisiológica.
Metodología: En esta revisión sistemática, se realizaron búsquedas en bases de datos bibliográficas como MEDLINE-Pubmed, Web of Science, Scopus y Science Direct. Los artículos publicados en los últimos cinco años que abordaban la AMPK, el ejercicio aeróbico y el entrenamiento interválico de alta intensidad cumplieron con los criterios de inclusión. Se encontraron 103 artículos publicados mediante el uso de las bases de datos Web of Science, Pubmed y Science Direct. Para esta revisión sistemática, se seleccionaron y analizaron diez artículos que cumplían con los criterios de inclusión. En este estudio, se evaluó el procedimiento operativo estándar (PRISMA) utilizando los elementos de informe preferidos para revisiones sistemáticas y metaanálisis.
Resultados: Se ha demostrado que el ejercicio aumenta la fosforilación de AMPK.
Discusión: El ejercicio de alta intensidad tiene mayor potencial para aumentar la AMPK que el ejercicio aeróbico debido al aumento de la relación AMP/ATP, el aumento del estrés metabólico y la adaptación mitocondrial. La activación de AMPK también se ha asociado con una mayor oxidación de ácidos grasos y la captación de glucosa por el músculo.
Conclusiones: Se puede concluir que el ejercicio físico ha demostrado aumentar de forma consistente la activación de AMPK y tiene un impacto positivo en la regulación metabólica. Por lo tanto, el ejercicio físico es una intervención eficaz para mejorar la función metabólica. Se ha demostrado que el ejercicio físico aumenta la fosforilación de AMPK, lo que aumenta la translocación de GLUT4 y la captación de glucosa.

Palabras clave

AMPK; Ejercicio físico; HIIT; GLUT4.

Introduction

One of the diseases that has become a global concern in both developed and developing countries is non-communicable diseases, which are one of the biggest health challenges in the world (Dahal et al., 2021). Chronic diseases can also develop from non-communicable disorders, if there are no good prevention efforts. There are many factors that trigger the body to be affected by non-communicable diseases, including genetic factors, physiological factors, unhealthy lifestyle behavior factors, and environmental factors (Budreviciute et al., 2020). Metabolic syndrome is an often-associated part of non-communicable diseases and has become a serious and important public health problem, including cardiovascular system diseases, and diabetes mellitus that can worsen human health (Deng et al., 2024).

Modern lifestyle changes that are identical to sedentary lifestyles, lack of movement, and eating a lot, are one of the serious causes of the increase in metabolic syndrome disease patients. A group of metabolic disorders known as the metabolic syndrome contribute to an increase in diseases such type 2 diabetes mellitus, cardiovascular disease, and neurological issues (Di Pietro, Izzo and Carrizzo, 2023). Obesity, elevated blood pressure, hypertriglyceridemia, elevated insulin resistance, and dyslipidemia of high-density lipoproteins (HDL) are further abnormalities in metabolic syndrome that, in the absence of prevention efforts, have a detrimental effect on human health (Agyemang-Yeboah et al., 2019).

It should be mentioned that according to recent reports, physical inactivity is now the fourth most common cause of death globally. In recent decades, physical activity is believed by several clinical and experimental studies to be the most important part of daily life in improving health and longevity (Bauman et al., 2021). The 2020 World Health Organization (WHO) guidelines recommend that people perform 150–300 minutes of moderate-intensity physical activity, 75–150 minutes of vigorous-intensity physical activity, or an equivalent combination of moderate-intensity and vigorous-intensity aerobic physical activity each week (Bull et al., 2020).

Right now, exercise and physical activity are the best non-pharmacological efforts to enhance the state of human health (Martín-Rodríguez et al., 2024). This physical activity includes body movements generated by the skeletal muscles that raise the amount of energy used (Chen et al., 2022). The increase in muscle contraction triggered by exercise provides exercise-induced adaptation of signal modulators. All cells, tissues, and organs, including skeletal muscle, the heart, the liver, adipose tissue, and the brain, have high levels of AMP-activated protein kinase (AMPK), is one of the signal modulators of exercise adaptation (Spaulding and Yan, 2022a).

During exercise, skeletal muscle activates a number of signaling pathways. One important signal transduction route that promotes enhanced mitochondrial biogenesis and oxidative metabolism is AMP-activated protein kinase (AMPK) (Zuo et al., 2023). Prior reviews have positively addressed AMPK's function in cellular adaptability and metabolism (Lundsgaard, Fritzen and Kiens, 2020). A reduction in ATP concentration and a rise in the AMP/ATP ratio brought on by physical activity are examples of cellular stressors that often activate AMPK, an intracellular energy change receptor (Zuo et al., 2023).

As far as we are aware, exercise interventions have been shown to improve general health and prevent muscle function decline or atrophy. Exercise is also involved in muscle remodeling and thus can promote hypertrophy. In addition, exercise also has a positive effect on increasing biogenesis in mitochondria. However, the various methods and types of physical activity that can benefit the human body as well as the underlying cellular mechanisms through increased AMPK phosphorylation are still not fully understood and are debated among researchers. This systematic review will therefore go over how physical activity's process of increasing AMPK phosphorylation, as well as how the underlying stages in increasing biogenesis in mitochondria.

Method

Study Design

This type of systematic review research uses searches through journal databases such as Science Direct, Web of Science, MEDLINE-Pubmed, and Scopus. Because they aggregate papers with a strong scientific basis and influence, these platforms are considered the greatest in the world.

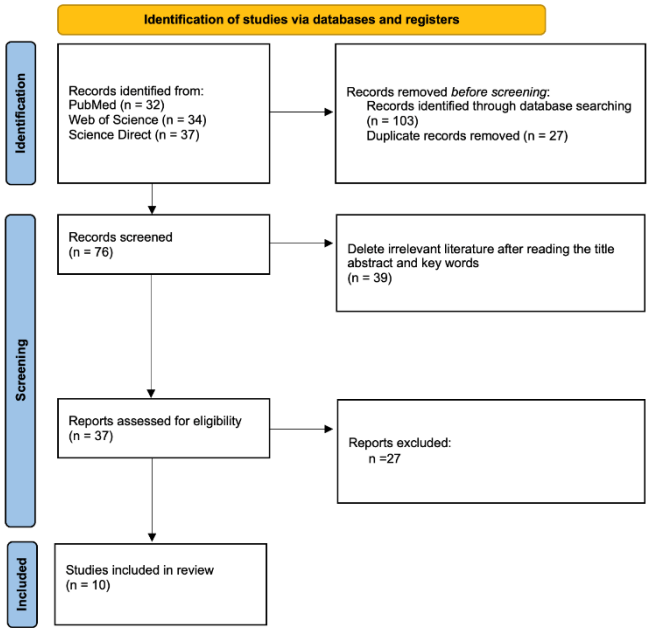
Eligibility criteria

The inclusion criteria for this study were created by reviewing publications published within the last five years that addressed AMPK, aerobic exercise, and high-intensity interval training. Furthermore, journals that did not adhere to scientific validity criteria or those were not listed in reliable search indices like Scopus, Web of Science, Pubmed, or Science Direct were not included in our analysis.

Procedure

Verified and checked abstracts, complete texts, and paper titles were added to the Mendeley database. Using the databases Science Direct, Pubmed, and Web of Science, 103 publications were located in the first phase. 76 articles that complied with the standards for the title and abstract were then chosen for the second round of screening. In the third stage, requests for additional processing were made for a total of 37 items. We now arranged the items according to how appropriate they were overall. After careful examination and observation, ten papers that met the inclusion criteria were selected for study. Following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) assessment was the operational criterion in this study.

Figure 1. PRISMA flowchart of the article selection process



Results

Table 1. Summary of the design and intervention of the studies

Author	Design	Participants	Participants Age	Intervention	Outcome
(Kim, Kim and Seong, 2022)	Randomized Controlled Trial	10 male mice	8 weeks old	Aerobic Exercise 1. Aerobic physical exercise performed daily for 4 weeks of intervention. 2. Aerobic exercise was performed on the experimental animal treadmill. 3. An activity wheel counter was used to record the daily running distance.	1. There was AMPK activity has significantly increased in the physical exercise intervention group for 4 weeks. 2. Measuring Tools: Western Blot.
(Lin *et al.*, 2020)	Randomized Controlled Trial	24 male rats	3 month old	Aerobic Exercise 1. Swimming training done 5x a week for 12 weeks. 2. Swimming in a container	1. Compared to the group that did not receive any physical exercise intervention, the senescence + exercise-induced group's AMPK levels were

				with water that is 32°C for freestyle. 3. The duration of each exercise was increased gradually from 20 minutes in week one and week two to 30 minutes in week three and 60 minutes in week four and so on.	noticeably greater. 2. Measuring Tools: Western Blot.
(Kartinah *et al.*, 2024)	Randomized Controlled Trial	24 male rats	8 weeks old	Continuous Training 1. Physical exercise was performed on a treadmill 6x a week for 6 weeks of treatment. 2. Rats ran on the treadmill starting with a duration of 20 minutes and increased by 2 minutes at regular intervals until reaching 60 minutes per training session. 3. The running speed stabilized at 27 m/min each training session. High Intensity Interval Training 1. Physical exercise was performed on a treadmill 6x a week for 6 weeks of treatment. 2. Interval training rats on the treadmill started with 2 intervals, 40 m/min, 3 minutes. 3. Until finally 20 intervals, 54 m/min, 30 seconds.	1. The group with diabetes mellitus + continuous training intervention significantly increased AMPK phosphorylation than the group with diabetes mellitus alone. 2. The group with diabetes mellitus + high-intensity interval training intervention significantly increased the phosphorylation of AMPK than the group with diabetes mellitus alone. 3. The results proved that HIIT intervention had a higher increase in AMPK phosphorylation than the continuous training group. 4. Measurement Tools: Measurement of AMPK phosphorylation in muscle using ELISA kits (Rat FGF21 BZ-08182631-EB Bioenzy, Indonesia and phosphorylated-AMPK BZ-08186340-EB-Bioenzy, Indonesia).
(Khalafi *et al.*, 2020)	Randomized Controlled Trial	40 male rats	7 weeks old	Moderate Intensity Continuous Training 1. Mice were run on a treadmill and adapted for 5 minutes at a speed of 6 m/min. 2. Until weariness, the pace was then progressively increased by 2 m/min every two minutes. 3. Physical exercise is done 5x a week. 4. Exercise intensity reaches 65% - 70%. 5. Exercise was carried out for 12 weeks of treatment. High Intensity Interval Training 1. Ten 4-minute high-intensity running sessions that achieve 85% to 90% of maximum speed are part of the HIIT protocol. 2. An active 2-minute rest interval at 50% of top speed. 3. A 2-minute active rest interval at half of one's top speed. 4. Exercise was performed for 12 weeks.	1. There was an increase in AMPK expression in the group with MICT and HIIT interventions significantly than the control group. 2. However HIIT had higher AMPK expression than the MICT group. 3. Measuring Tools: Western Blot.
(Martinez-Huenchullan *et al.*, 2019)	Randomized Controlled Trial	72 male rats	10 weeks old	Moderate Intensity Endurance Exercise 1. Physical exercise was performed using a treadmill at 70% of the maximum speed. 2. Exercise was performed for 40 minutes. 3. Exercise was performed 3x a week for 10 weeks. High Intensity Interval Training 1. Physical exercise was performed on a treadmill consisting of 8 laps (2.5 minutes each). 2. Exercise intensity was 90% of maximal speed. 3. Exercise was performed 3x a week for 10 weeks of intervention.	1. There was a significantly highest increase in AMPK phosphorylation in the group with HIIT intervention. 2. Measuring Tools: Western Blot.
(Li *et al.*, 2021)	Randomized Controlled Trial	21 male rats	8 weeks old	Moderate Intensity Swimming Training Protocol 1. Rats underwent a 1-week swimming adaptation starting with 10 minutes on the first day and gradually increasing to 60 minutes. 2. Forced swimming core	1. There was a significant increase in AMPK phosphorylation in the high fat diet (HFD) + swimming physical exercise intervention group than the high fat diet (HFD) group. 2. Measuring Tools: Western Blot.

				training for 60 minutes. 3. Exercise was performed for 5 days per week for 16 weeks of treatment.	
(Cheng *et al.*, 2022)	Randomized Controlled Trial	24 male mice	8 weeks old	Moderate Intensity Swimming Training Protocol 1. Rats swim freely in the Morris Water Maze swimming pool. 2. One week of exercise adaptation process was carried out starting with a duration of 10 minutes and increasing by 10 minutes to 60 minutes. 3. Exercise intervention was conducted for 8 weeks of treatment.	1. There was a significant increase in AMPK phosphorylation in the high fat diet (HFD) + swimming physical exercise intervention group than the high fat diet (HFD) group. 2. Measuring Tools: Western Blot.
(Zhang *et al.*, 2023)	Randomized Controlled Trial	36 mice	10 weeks old	Aerobic Exercise 1. A mouse running on a treadmill. 2. Running training was performed for 90 minutes per day, 6 days per week, for 6 weeks of intervention. 3. The treadmill speed was 15 cm per second. 4. The speed of the rats reached 80% of VO2Max.	1. There was a significant increase in AMPK phosphorylation in the group with physical exercise intervention. 2. Measuring Tools: Western Blot.
(Wang *et al.*, 2022)	Randomized Controlled Trial	48 mice	8 – 9 weeks old	Aerobic Exercise 1. Rats swam for 15 minutes with a 5-minute break in each training session.	1. There was a significant increase in AMPK phosphorylation in the group with physical exercise intervention. 2. Measuring Tools: Immunoblotting.
(Lin *et al.*, 2024)	Randomized Controlled Trial	23 male mice	4 weeks old	Aerobic Exercise 1. Mice perform physical exercise including running on a treadmill. 2. Physical exercise on the treadmill is carried out with a duration of 30 minutes per session at a speed of 12 m / min. 3. Exercise is done 5x a week with a total duration per week of 150 minutes. 4. The total intervention was conducted for 12 weeks.	1. There was a significant increase in AMPK phosphorylation in the group with physical exercise intervention. 2. Measuring Tools: Western Blot.

Description Physical Exercise Increases AMPK Phosphorylation Included in Review

Based on a systematic review, it has been shown that physical exercise can increase the AMPK (adenosine monophosphate-activated protein kinase) phosphorylation. The type of exercise, duration, and intensity of exercise affect the phosphorylation level of AMPK. The interventions were moderate to vigorous in intensity, both of which had beneficial effects in increasing AMPK phosphorylation after physical exercise.

Considering the ten publications that were examined, all of them show an increase AMPK in mice after physical exercise intervention (Kim et al., 2022; Lin et al., 2020; Kartinah et al., 2024; Khalafi et al., 2020; Martinez-Huenchullan et al., 2019; H. Li et al., 2021; Cheng et al., 2022; Zhang et al., 2023; Wang et al., 2022; Jun et al., 2024). Furthermore, the information revealed that the provision of varied physical exercise interventions ranging from the type of exercise, duration, and length of intervention had different effects on increasing AMPK phosphorylation. The physical exercise interventions used were also diverse, consisting of high-intensity interval training and aerobic exercise, all of which showed a positive increase in AMPK phosphorylation after the intervention.

This study focused on looking at and analyzing physical exercise in increasing AMPK phosphorylation in mice. Articles that did not address this were included in the exclusion criteria, which were not included in this systematic analysis. Articles that discussed how moderate or vigorous intensity physical exercise can increase AMPK were included in the inclusion criteria to be discussed further. So it can be concluded on the basis of the examined papers show a significant increase in AMPK phosphorylation after physical exercise.

Effect of Exercise Intensity on Increasing AMPK Phosphorylation

The research results presented by Kim et al., 2022 showed that rats given a moderate-intensity aerobic physical exercise intervention for 4 weeks proved to increase the activation of AMPK after the provision of physical exercise intervention. Aerobic exercise stimulates signal transduction with several stages occurring through the activation of AMPK. It has been determined that AMPK is a sensor for energy stress within cells in adipose tissue, liver, and skeletal muscle (D, Jamie I. vaner Vaart et al., 2021). The findings of a further investigation also showed that aerobic physical exercise with swimming intervention in rats 5x a week for 12 weeks was shown to significantly increase AMPK phosphorylation (Lin *et al.*, 2020).

The results of different workout routines and durations vary on the increase in AMPK phosphorylation after physical exercise intervention. Comparison of physical exercise interventions between continuous training and high intensity interval training both have an effect on increasing AMPK phosphorylation after physical exercise intervention. However, high intensity interval training has a higher effect on AMPK phosphorylation than continuous training (Kartinah *et al.*, 2024). This data also provides insight that HIIT training is better at increasing AMPK phosphorylation, however more research is required to determine the underlying mechanism.

Another study also confirmed that the comparison between moderate intensity continuous training and high intensity interval training for 12 weeks in rats had an effect on increasing AMPK phosphorylation in both types of training, but training with high intensity interval training provided higher data on increasing AMPK phosphorylation than moderate intensity continuous training (Khalafi *et al.*, 2020). Research results Martinez-Huenchullan et al., 2019 that high intensity interval training in rats performed 3x a week for 10 weeks of intervention was shown to significantly increase AMPK phosphorylation. This emphasizes that high intensity interval training has a higher effect on increasing AMPK.

Discussion

This systematic review's objective is to ascertain how physical exercise affects AMPK phosphorylation. A comprehensive explanation is needed to provide understanding and evidence of how exercise and its signal transduction stages lead to an increase in AMPK. Cells must connect to maintain a steady internal state, growth and proliferation are linked to the availability and consumption of resources like as glucose, lipids, and amino acids (Smiles et al., 2024). A serine/threonine kinase called A sensor of intracellular energy stress has been identified as AMPK (Hardie, 2020). Alpha (isoform α1 or α2), beta (isoform β1 or β2), and gamma (isoform γ1, γ2, or γ3) subunits make up the heterotrimer known as AMPK. The β subunit acts as a scaffold to join the α and γ subunits, whereas the α subunit houses the catalytic domain. In order to cause a conformational shift in the α subunit that promotes AMPK activation through phosphorylation by upstream kinases, the γ subunit monitors the relative amounts of AMP or ADP to ATP. Only the three cystationin-β-synthase (CBS) domains on the γ subunit—CBS1, CBS3, and CBS4—bind AMP, ADP, and ATP (Spaulding & Yan, 2022).

Noteworthy is the fact that the mechanism underlying physical exercise in increasing AMPK is still a matter of debate among researchers. Further exploration is needed to increase knowledge related to how the mechanisms underlying physical exercise on AMPK increase. During physical exercise, the body activates the sympathetic nervous system which will increase the performance of skeletal muscles (Chiang et al., 2024). These skeletal muscles contract and work maximally to saturate oxygen to the cells. A well-designed control system guarantees the quick supply of ATP and maintenance of ATP content in muscle cells because the metabolic rate can increase by more than 100 times from rest to exercise (Hargreaves and Spriet, 2020). The increased need for ATP makes the body automatically fulfill the need for ATP production. Energy production during physical exercise occurs by the mechanism of oxidative phosphorylation in the mitochondria (Vargas-Mendoza et al., 2021).

In the process, physical exercise also triggers an increase in reactive oxygen species (ROS) (Wibawa, Arifin and Herawati, 2021). Other data also shows the same thing that when we do physical exercise, the body will increase ROS (reactive oxygen species) as a form of physiological response (Shamsnia et al., 2023). Physical exercise can effectively increase glucose uptake in skeletal muscle (Wang et al.,

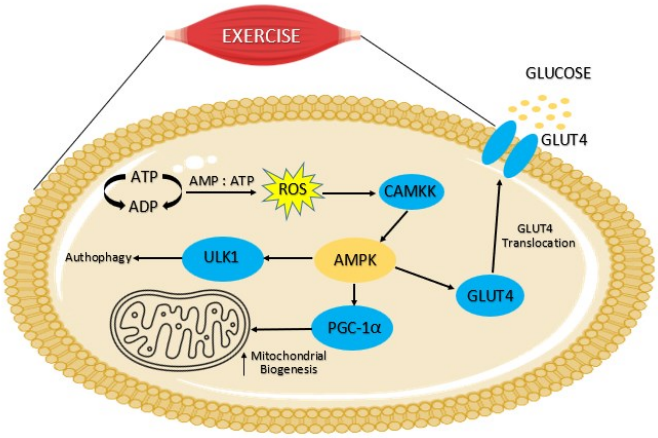
2023). After the muscle contracts and ROS increases, calcium ion (Ca2+) release channels in the sarcoplasmic reticulum open in response to the activation of muscle contraction through propagating the action potential throughout the T tubules. Passively, Ca2+ ions penetrate the cytoplasm, thereby increasing cytosolic Ca2+ between ten- to twenty-fold (Gejl et al., 2020). Increased intracellular Ca2+ concentration triggers activation of calcium/calmodulin-dependent protein kinase kinase (CaMKK) (Tokumitsu and Sakagami, 2022).

Physical exercise also triggers an increase in CaMKK as a physiological response to exercise. CaMKK activation will increase AMPK activity which is also very important and necessary for physical fitness, and research results show that exercise can increase these benefits by improving muscle function through AMPK regulation (Chen et al., 2023). This increase in AMPK will provide a physiological response by triggering several subsequent mechanisms. Peroxisome proliferator-activated receptor γ coactivator 1α (PGC-1α) also increases after being activated by AMPK (Guo and Lu, 2024). PGC-1α may be a key mediator of AMPK-induced gene expression, as evidenced by the intriguing overlap between genes controlled by PGC-1α and those regulated by AMPK in transcription. Furthermore, PGC-1α is characterized as a master regulator of mitochondrial biogenesis and function, which improves mitochondrial metabolism and generates energy to fulfill the body's demands (Shelbayeh et al., 2023).

In order to influence metabolic regulation and cellular adaptation processes, activated AMPK phosphorylates downstream effectors, which either activates or inhibits them. Then, an increase in phosphorylated AMPK will increase the production of glucose transporter 4 GLUT4, which is a protein that plays a role in glucose transportation (Kartinah et al., 2024). Glucose transporters type 4 (GLUT4) facilitate glucose entry into skeletal muscle cells by providing a glucose concentration gradient from the cytoplasm (the inside of the muscle) to the interstitial space (the region outside the muscle cell), increasing the muscle's absorption of glucose (Richter, 2021).

The control of protein, lipid, and glucose metabolism as well as cellular adaptation mechanisms like autophagy and mitochondrial remodeling have all been linked to AMPK activation (Spaulding & Yan, 2022). In particular, AMPK is a downstream signaling molecule that is necessary for controlling glucose and lipid metabolism as well as preserving mitochondrial homeostasis (Lin et al., 2024). Part of what causes exercise-induced PGC-1α expression is AMPK phosphorylation (activation) (Xiao et al., 2024). By activating AMPK and alleviating many chronic disease states through mitochondrial remodeling in skeletal muscle, exercise is the most effective strategy to enhance health (Spaulding & Yan, 2022).

Figure 2. The Mechanisms of Physical Exercise in Increasing AMPK Phosphorylation



The results of research conducted by Li et al., 2021 showed that mice modeled with high fat diet and given aerobic exercise intervention for 16 weeks proved to have an effect on significantly increasing AMPK phosphorylation. Other and similar data in rats induced high fat diet and given aerobic physical exercise intervention with intervention duration for 8 weeks demonstrated that there was a notable rise in AMPK phosphorylation (Cheng et al., 2022). It can be concluded that animals that are high in fat and

given physical exercise interventions have a good effect on metabolic mechanisms by triggering an increase in the signal transduction pathway of AMPK. Other data on 10-week-old rats given aerobic physical exercise intervention using a treadmill performed 90 minutes per day, 6x a week for 6 weeks of intervention had a significant effect on increasing AMPK phosphorylation (Zhang et al., 2023). Another significant increase in AMPK phosphorylation in rats given the aerobic physical exercise intervention was also shown by Wang et al., 2022.

Strenght and Limitations

The advantage of this systematic review is that it only looks at randomized controlled trials, which are the most reliable type of scientific evidence because there is no possibility of ambiguous causal relationships. In addition, the samples taken are focused on rats so that all samples can show homogeneous data and are not mixed with other animal categories and human categories. The topic related to regular physical activity and its effects on increasing AMPK phosphorylation after physical exercise is very relevant with a detailed discussion of its role so that it can recommend physical exercise as the best effort in increasing the mechanism of mitochondrial biogenesis through AMPK activation. An in-depth explanation of the mechanisms and stages of how physical exercise affects other signal transduction is important to show as a development of knowledge and understanding of the complex mechanisms in cells. The limitation that we encountered is the lack of discussion and discussion related to how physical exercise in increasing AMPK phosphorylation and its mechanism has not gotten much attention, although regular physical exercise can affect how well people live their lives so as to improve the mechanism of mitochondrial biogenesis to affect the absorption of glucose levels as an effort to meet energy needs in the body. Therefore, this review is considered important to do so as to add insight and repertoire of knowledge related to how the mechanism underlying this physical exercise in increasing AMPK phosphorylation.

Conclusions

Based on the articles we have reviewed it can be said that physical activity has been shown to increase AMPK phosphorylation after exercise. This increase in AMPK triggers other physiological responses such as increased GLUT4 translocation. The impact of this increase in GLUT4 translocation will increase the absorption of blood glucose levels during exercise directly. In addition, AMPK also increases mitochondrial biogenesis, thus contributing to increased energy formation that functions to meet the body's needs.

Conflicts of Interest

The authors declare no conflict of interest.

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