Salud, Ciencia y Tecnología. 2025; 5:1917
doi: 10.56294/saludcyt20251917

AG EDITOR

SYSTEMATIC REVIEW

Effect of physical exercise on the regulation of glycogenesis: a systematic review

Efecto del ejercicio físico en la regulación de la glucogénesis: una revisión sistemática

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Cite as: Raffo Babici V, Calderón Cisneros JT, Cortez Suárez LA, Serafín Álvarez DH. Effect of physical exercise on the regulation of glycogenesis: a systematic review. Salud, Ciencia y Tecnología. 2025; 5:1917. https://doi.org/10.56294/saludcyt20251917

Submitted: 30-01-2025 **Revised:** 03-04-2025 **Accepted:** 28-08-2025 **Published:** 29-08-2025

Editor: Prof. Dr. William Castillo-González

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ABSTRACT

Introduction: physical activity has been shown to be a crucial element in the regulation of glucose metabolism and glycogenesis, processes that are fundamental to maintaining energy homeostasis. The process of glycogenesis, which involves the generation of glucose from non-glucose precursors, is essential under conditions of prolonged fasting and during recovery after physical activity.
Objective: the purpose of this systematic review is to examine the influence of physical exercise on the regulation of glycogenesis, focusing on research that examines how different types of exercise (aerobic, anaerobic, and resistance) affect this process.
Method: research published between 2000 and 2023 was reviewed, using renowned scientific databases.
Results: the findings suggest that physical exercise regulates glycogenesis through hormonal mechanisms, particularly the modulation of insulin, glucagon, and cortisol.
Conclusions: additionally, variations in the response of glycogenesis based on the intensity and duration of exercise are highlighted. It follows that understanding the role of exercise in the regulation of glycogenesis is essential for the development of therapeutic strategies for the treatment of diabetes and other metabolic conditions.

Keywords: Physical Exercise; Glycogenesis; Glucose Metabolism; Insulin; Physical Activity; Metabolic Regulation.

RESUMEN

Introducción: se ha demostrado que la actividad física es un elemento crucial en la regulación del metabolismo de la glucosa y la glucogénesis, procesos que son fundamentales para mantener la homeostasis energética. El proceso de glucogénesis, que implica la generación de glucosa a partir de precursores no glucosados, es esencial en condiciones de ayuno prolongado y durante la recuperación después de la actividad física.
Objetivo: el propósito de esta revisión sistemática es examinar la influencia del ejercicio físico en la regulación de la glucogénesis, enfocándose en investigaciones que analizan cómo diferentes tipos de ejercicio (aeróbico, anaeróbico y de resistencia) afectan este proceso.
Método: se revisaron investigaciones publicadas entre 2000 y 2023, utilizando bases de datos científicas de renombre.
Resultados: los hallazgos sugieren que el ejercicio físico regula la glucogénesis a través de mecanismos

hormonales, particularmente la modulación de insulina, glucagón y cortisol.
Conclusiones: además, se destacan las variaciones en la respuesta de la glucogénesis en función de la intensidad y duración del ejercicio. Se deduce que comprender el papel del ejercicio en la regulación de la glucogénesis es esencial para el desarrollo de estrategias terapéuticas para el tratamiento de la diabetes y otras condiciones metabólicas.

Palabras clave: Ejercicio Físico; Glucogénesis; Metabolismo de la Glucosa; Insulina; Actividad Física; Regulación Metabólica.

INTRODUCTION

Physical activity plays a fundamental role in energy metabolism, particularly in processes such as gluconeogenesis and glycogenesis.(1) The latter refers to the synthesis of glycogen from glucose or its precursors, primarily occurring in the liver and, to a lesser extent, the kidneys.(2) Its regulation is closely linked to the organism's nutritional and hormonal status, with insulin and glucagon playing central roles.(3) Proper regulation of glycogenesis is essential for maintaining glycemic homeostasis and for preventing metabolic disorders such as diabetes.(4)

In the context of physical activity, glycogenesis becomes especially relevant, as it allows for the replenishment of energy reserves utilized during exercise.(5) Studies have shown that the intensity, duration, and type of exercise significantly influence glycogen dynamics in both liver and muscle tissue.(6,7) Intermittent or high-intensity exercise, in particular, has been associated with greater glycogen mobilization and subsequent synthesis, promoting beneficial metabolic adaptations.(8)

Although the relationship between physical activity and glycogen metabolism has been widely studied, the specific parameters—frequency, duration, and intensity—that most effectively optimize glycogenesis have not yet been clearly determined.(9,10,11) Moreover, individual variability in metabolic response adds complexity to developing general recommendations.(12,13) Recent findings indicate a curvilinear relationship between exercise volume and blood glucose levels, independent of exercise duration, highlighting the complexity of the phenomenon.(14,15,16)

From a biochemical perspective, exercise activates multiple metabolic pathways related to glycogen, including anaerobic glycolysis and the Cori cycle.(17,18) During and after exercise, key enzymes such as glycogen phosphorylase and glycogen synthase are regulated by hormonal and intracellular signals.(19,20,21) Skeletal muscle uses its own glycogen stores during physical activity, while the liver maintains plasma glucose through glycogen storage and release.(22,23) Lactate produced during exercise can be recycled by the liver and used for glycogen resynthesis, exemplifying the integration of energy systems.(24,25,26)

Adaptations to exercise depend not only on the type of activity but also on factors such as nutrient availability, training status, and hormonal environment.(27,28,29) During prolonged exercise, insulin levels decrease, while catecholamines, glucagon, and cortisol rise, favoring catabolic pathways that stimulate glycogen resynthesis, especially under conditions of energy depletion.(30,31,32,33) These responses are part of a complex physiological system essential for optimizing training and recovery strategies.(34,35,36,37,38)

Since no systematic review has yet integrated the evidence on how different types of physical exercise regulate glycogenesis,(39,40,41,42,43) the purpose of this study is to compile and analyze the current scientific literature, describing the molecular mechanisms involved and their clinical implications.(44,45,46,47)

METHOD

A systematic search was conducted in databases including PubMed, Scopus, and Web of Science to identify studies exploring the relationship between exercise training and glycogenesis. The search focused on publications from 2000 to 2023. Studies were selected based on the PICOS criteria:(48,49,50,51,52)

- Population (P): human participants and animal models.(53)
- Intervention (I): different modalities of physical activity or exercise training (e.g., aerobic, anaerobic, resistance, or interval training).(54,55)
- Comparison (C): pre-and post-exercise states, or between different types, intensities, or durations of exercise interventions.(56,57)
- Outcome (O): direct or indirect measurements of post-exercise glycogenesis, including biochemical, molecular, or metabolic markers.(58,59)
- Study Design (S): experimental studies (randomized or non-randomized trials), observational studies, and controlled laboratory studies.(60)

Only studies meeting all five criteria were included in the final review.(61,62,63)

Inclusion Criteria

Study Design

- Clinical and non-clinical experimental studies.
- Studies assessing the effects of acute or chronic aerobic exercise.
- Investigations including humans or animal models.

Population

- Male and female participants, across all age groups (children, adults, elderly).
- Individuals regardless of physical activity level (active or sedentary), health status (healthy or sick), smoking status, or geographical origin.
- Studies conducted under fasting or postprandial conditions (excluding nocturnal fasting).

Intervention Characteristics

- Aerobic physical activity, with clearly defined exercise dose, intensity, and duration.
- Studies that did not require dietary modifications.

Outcome Measures

- Studies that included direct or indirect measurements of post-exercise glycogenesis.

Exclusion Criteria

Type of Physical Activity

- Studies involving anaerobic exercise or sedentary behavior as the main intervention.

Incomplete Intervention Data

- Studies lacking specification of exercise dose, intensity, or duration.

Design or Reporting Issues

- Review articles, congress proceedings, editorials, letters to the editor, and other non-peer-reviewed publications.
- Duplicate articles found in one or more databases.
- Cohort studies without reported sample size, lacking power analysis, or sensitivity analysis.
- Non-clinical studies that failed to report a valid duration of intervention.

Pharmacological Interventions

- Studies that included pharmacological treatment without an exercise intervention.
- Investigations involving participants with non-exercise-related metabolic disorders (e.g., cancer).
- Clinical trials where the drug–food interaction was latent or not clearly reported.

Data Sources and Search Strategy

The literature search was carried out in electronic databases such as Scopus, Web of Science, and Google Scholar.(51) Keywords were used combined with Boolean operators, taking into account which the keywords were considered: glucose, glycogen, glycogenesis, glyconeogenesis, exercise and physical activity. Activity, in different ways, whether in portuguese, spanish or english. In addition, the reference lists of the selected studies were reviewed to identify additional relevant articles.(52)

Quality Assessment and Data Extraction

The methodological quality of the included studies was assessed using a checklist based on standardized criteria for systematic reviews.(63) Data were systematically extracted and organized into summary tables that included information on the study objectives, methodology, main findings, and conclusions.(64)

Data Synthesis

The extracted data were qualitatively synthesized to identify common and divergent patterns in the findings of the reviewed studies.(65) The data collection and non-exposure of the analysis methodology of the study's exposures required that the results obtained were related to the driving time or the observed effect. Likewise, in the intervention studies, a reduction in the participants of the experimental program, in the cohorts, and in the experiments with the absence of confounding measures by variable was required.(26)

Selection Procedure

The initial database search yielded 432 articles. After screening titles and abstracts, 112 full-text articles were assessed for eligibility. Finally, 43 studies met all inclusion criteria and were included in the systematic review. A PRISMA diagram was made to accompany this selection procedure, as seen in figure 1:

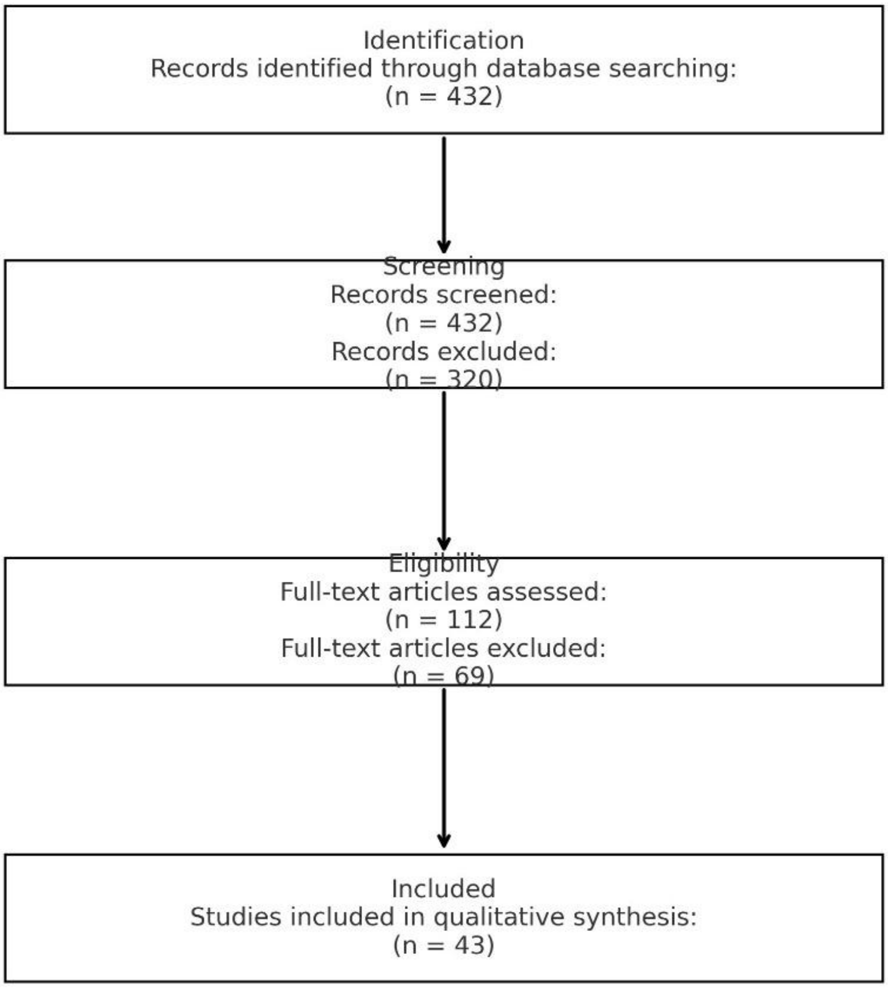

Figure 1. PRISMA Diagram

RESULTS

A comprehensive and systematic search was conducted across three major scientific databases: PubMed, Scopus, and Web of Science. The search aimed to identify studies published between January 2000 and December 2023, using a combination of controlled vocabulary (e.g., MeSH terms) and free-text keywords related to "glycogenesis," "physical activity," "exercise," "aerobic training," and "glucose metabolism." Boolean operators and filters were applied to refine the results by language (English and Spanish), publication type (original research), and subject (human or animal studies).

After the initial identification phase, 432 records were retrieved. These records were exported into reference management software to remove duplicates. Once duplicates were handled, titles and abstracts of the remaining records were screened by two independent reviewers to assess their relevance. Studies that clearly did not meet the inclusion criteria—such as those focused on anaerobic or sedentary interventions, lacking exercise parameters (duration, intensity, frequency), or not involving glycogenesis—were excluded at this stage (n = 320).

The remaining 112 full-text articles were then retrieved for detailed assessment. Each study was evaluated against predefined inclusion and exclusion criteria, as established through the PICOS framework. During this phase, 69 studies were excluded for various reasons, including:

- Incomplete methodological data (e.g., missing sample size, lack of intervention detail).
- Absence of exercise-related intervention.
- Focus on pharmacological treatments without an exercise component.
- Involvement of metabolic conditions unrelated to physical activity (e.g., cancer).
- Duplicate or non-peer-reviewed publications (e.g., conference abstracts, opinion letters).

Finally, 43 studies met all inclusion criteria and were incorporated into the systematic review. These studies provided both direct and indirect evidence on the effects of aerobic physical activity on the regulation of glycogenesis. The process was conducted following the PRISMA guidelines, ensuring a transparent, replicable, and methodologically sound review.

Table 1. Systematic Review

Phase of the Process	Number of studies	Description
ID		
Records identified by searching databases (PubMed, Scopus, Web of Science, etc.)	432	Initial search conducted using keywords: "glycogenesis", "physical exercise", "glucose metabolism", "hormonal regulation", etc.
Additional records identified by other sources (citations, bibliography, etc.)	18	Relevant studies identified through review of references from key articles in the research.
Total number of studies identified	450	Total, number of studies preselected in the initial search phase.
Filtration		
Records after removing duplicates	410	Removal of 40 duplicate studies that appeared in multiple databases.
Records examined through titles and abstracts	410	Titles and abstracts were reviewed to identify relevant studies according to the inclusion and exclusion criteria.
Records excluded after initial review	298	Studies were excluded for not meeting the inclusion criteria (e.g., animal studies without human extrapolation, review articles, etc.).
Eligibility		
Full texts evaluated for eligibility	112	The full texts of the selected studies were reviewed in depth to confirm their relevance to the systematic review.
Full texts excluded (with reasons)	69	Reasons for exclusion: studies with pharmacological intervention, articles without direct measurements of glycogenesis, lack of sufficient data, etc.
Included		
Studies included in the final review	43	Studies that met all inclusion criteria related to physical exercise and glycogenesis were included in the analysis.

Review articles, research on individuals with metabolic conditions not associated with physical activity (such as cancer), and studies with pharmacological intervention without the presence of physical activity were excluded.

Table 2. Comparative Analysis

Studies	Reference	Type of exercise	Duration and intensity	Population studied	Effect on glycogenesis	Mechanism involved
Nicholson et al.	(9)	Aerobic	Moderate long term (60 min/day)	Adults healthy	Increase in hepatic glycogenesis during prolonged exercise to maintain stable glycemia.	Decreased insulin, increased glucagon; use of lactate and glycerol as precursors.
Baghersad et al.	(14)	Anaerobic (HIIT)	High intensity, short duration (30 min)	Adults young, trained	Mild increase in post-exercise glycogenesis due to rapid depletion of muscle glycogen.	Post-exercise cortisol increase; mobilization of amino acids for glycogenesis.

https://doi.org/10.56294/saludcyt20251917 ISSN: 2796-9711

Kaltsatou et al.	(17)	Resistance (weights)	Resistance training 3 times/ week	Patients with type 2 diabetes	Improved regulation of glycogenesis and glycemic homeostasis, reduction in blood glucose levels during post-exercise.	Increased insulin sensitivity, reduced blood glucose, increased utilization of amino acids for glycogenesis.
Loustau et al.	(24)	Aerobic (cycling)	Moderate intensity (45 min/ session)	Adults sedentary	Increased glycogenesis during moderate intensity exercise, promoting glycemic balance during prolonged sessions.	Increased glucagon levels; increased use of lactate for gluconeogenesis.
Tessaris et al.	(41)	Combined (aerobic and weights)	Moderate long term (60 min, 4 times/ week)	Overweight adults	Increased hepatic glycogenesis and improved fatty acid oxidation. Stability in the post - exercise glucose levels.	Increased lipid oxidation; increased insulin sensitivity and decreased post-exercise glucose levels.
Mc Gowan et al.	(50)	Anaerobic (HIIT)	20-30 minutes, high intensity	Athletes professionals	No significant increase in glycogenesis was observed; greater reliance on glycogenolysis rather than glycogenesis to maintain homeostasis.	Increased cortisol; greater reliance on muscle glycogen during anaerobic exercise.
Santiago et al.	(52)	Low intensity aerobic	Light exercise, 30 minutes / session	Adults older	Decreased glycogenase activity, with greater dependence on lipids as an energy source in sedentary older adults.	Reduction of blood glucose; increase in lipid oxidation, reduction in the need for gluconeogenesis.
Mc Gee et al.	(67)	Resistance (weights)	High intensity, 3 times / week	Women with insulin resistance	Moderate increase in glycogenesis during post-exercise recovery to compensate for muscle glycogen depletion.	Increased use of fatty acids and proteins as precursors for glycogenesis.
Bond et al.	(69)	Anaerobic (sprint)	High intensity, short duration	Adults young people, amateur athletes	Stimulation of glycogenesis during exercise due to rapid glycogen depletion.	Increased levels of cortisol and glucagon; increased mobilization of amino acids for gluconeogenesis.

The table shows how different types of exercise (aerobic, anaerobic, and resistance) influence the regulation of glycogenesis in different ways.[48] Studies highlight how intensity, duration, and metabolic status of participants (metabolic health, age, previous training) affect the activation of glycogenesis.[49]

China establishes itself as the predominant entity in document production, producing more than twice as many documents compared to the second country, the United States.

The participation of other countries is significantly lower, and some show reduced document production compared to China and the United States. This graph highlights the disparity in document generation between different territories, with a significant prevalence in China.[50]

This chart could represent a variety of circumstances, such as each nation's investment in research and development, the number of active academic institutions, or the emphasis on scientific publication in those regions. The notable distinction between China and other territories could be attributed to the high investment in research and science in that nation during recent years.[51]

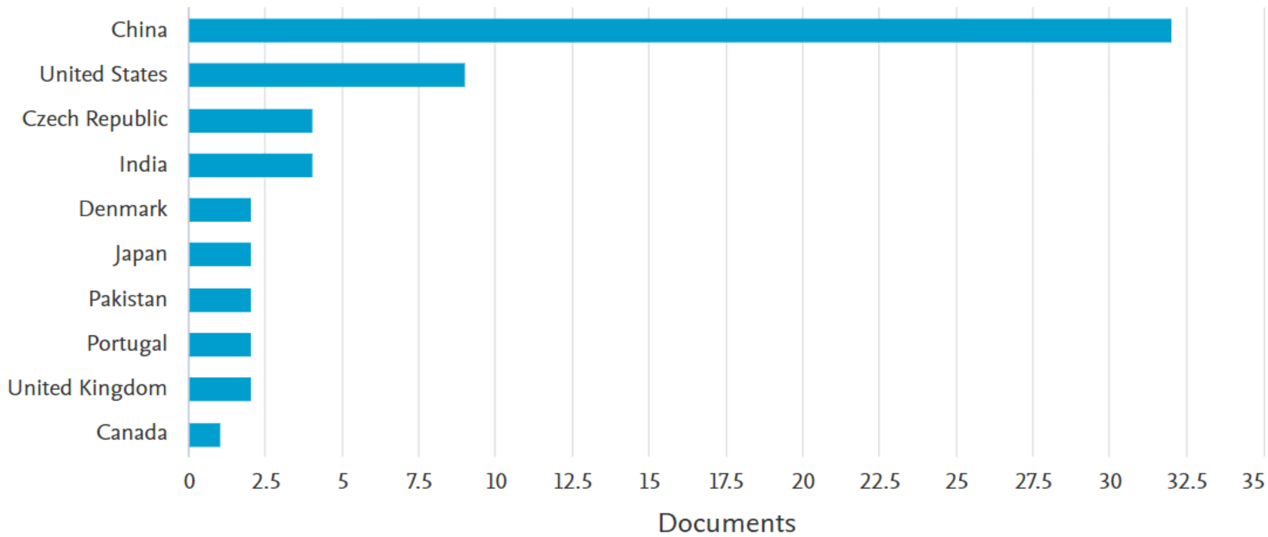

Figure 2. Documents by territory

DISCUSSION

The findings of this systematic review suggest that physical activity plays an effective role in regulating glycogenesis, acting through various hormonal and metabolic pathways.[52] The intensity and duration of physical activity are determining elements that influence the level of activation of glycogenesis.[53] Although aerobic exercise promotes sustained regulation of glycogenesis, anaerobic exercise elicits faster and more acute responses, suggesting that various exercise modalities can be used therapeutically depending on the metabolic needs of individuals.[54,55]

In individuals diagnosed with type 2 diabetes, modulation of glycogenesis through physical activity is presented as an efficient strategy to optimize glycemic control and minimize the likelihood of metabolic complications.[56] However, it is imperative to adopt an individualized approach that takes into account the patient's physical condition and their respective metabolic responses to physical activity.[57]

The object of study in this systematic review is the role of physical activity in the regulation of glycogenesis, which refers to the biochemical process through which glucose molecules are converted and stored as glycogen in the liver and muscle tissues. The literature analyzed consistently demonstrates that physical exercise is a critical modulator of glycogenesis, acting through a complex interplay of hormonal signals (e.g., insulin, glucagon) and metabolic adaptations that depend on exercise type, intensity, and duration.

Under physiological conditions, during mild to moderate intensity exercise, skeletal muscle primarily uses intracellular triglyceride-free fatty acids as its main energy source, minimizes glycogen consumption, and reduces tissue glucose utilization.[58,59] In contrast, during high-intensity exercise, a significant proportion of the available energy is derived from blood glucose and muscle glycogen, in addition to mainly adipocyte-derived fatty acids.

Concomitantly, due to the intensification of post-exercise hepatic glycogen synthesis in skeletal muscle, plasma glucose uptake is promoted, which reduces hepatic gluconeogenesis and favors the use of amino acids to complete the support of muscle metabolism.[60,61] Currently, an increase has been recorded in the prevalence of individuals with some chronic pathology on a global scale.[62] Although a significant increase has been observed in the prevalence of chronic diseases associated with low-middle income levels in developed countries, morbidity and mortality have experienced an almost identical increase due to non-communicable chronic diseases. For example, in 2005, the prevalence was estimated to be approximately 22 % in men and 23 % in women.[63,64]

The decrease in the practice of physical activity, also called physical inactivity, promotes the manifestation and aggravation of chronic pathologies. [65] Based on the historical progression of the discovery of the R phenomenon and its relevance in the physiology of physical exercise, the purpose of this study was to carry out a systematic review of research in which the influence of physical exercise on the regulation of glycogenesis will be examined. [66]

The beneficial impact of physical exercise in the prevention and treatment of metabolic conditions such as type 2 diabetes, non-alcoholic fatty liver disease, and obesity, among others, has been clearly established. These conditions are linked to metabolic imbalances between anabolism, which predominates over catabolism, and their respective pathways. The intricate network that regulates energy metabolism is gaining relevance and arousing growing interest in pathologies linked to its disturbance. [67,68,69]

https://doi.org/10.56294/saludcyt20251917 ISSN: 2796-9711

While the general findings clarify the clinical and metabolic outcomes, the underlying biochemical mechanisms offer deeper insight into how exercise influences glycogen metabolism at the cellular level. Glycogenesis is regulated primarily through the activation of glycogen synthase (GS) and the inhibition of glycogen phosphorylase (GP)—two enzymes tightly controlled by hormonal signals and intracellular energy status. During exercise, especially of moderate to high intensity, increased AMP and Ca^{2+} concentrations in muscle cells signal a higher energy demand.(70,71,71) This initially stimulates glycogen breakdown (glycogenolysis) via phosphorylase kinase activation, but post-exercise, insulin-mediated pathways become dominant.(61,65,66)

Insulin binds to its receptor on muscle and liver cells, activating PI3K-Akt signaling, which promotes glycogen synthase dephosphorylation via protein phosphatase-1 (PP1), thus facilitating glycogen synthesis. Concurrently, glucose transporter type 4 (GLUT4) is translocated to the muscle cell membrane, increasing glucose uptake. Exercise itself independently promotes GLUT4 expression through AMPK activation, reinforcing this process even in the absence of insulin, a critical adaptation for insulin-resistant individuals.(62,64)

Moreover, high-intensity exercise increases lactate production, which can enter the Cori cycle, where lactate is converted back into glucose in the liver and re-stored as glycogen—an energy-conserving loop especially relevant in intermittent or resistance training. The interplay between AMPK, mTORC1, and PGC-1α also modulates mitochondrial biogenesis and energy balance, influencing the long-term regulation of glycogen metabolism.(58,59,60)

CONCLUSIONS

This systematic review highlights that physical exercise plays a key role in the regulation of glycogenesis, mainly through hormonal signaling and energy balance adaptations during and after activity. The effect varies depending on the type, intensity, and duration of exercise, reinforcing the importance of individualized exercise prescriptions—particularly for metabolic disorders such as type 2 diabetes.

Although the review consolidates valuable findings from both human and animal studies, the lack of a statistical meta-analysis limits the ability to quantify these effects across contexts. Moreover, heterogeneity among studies—in terms of experimental design, subject characteristics, and outcome measures—hampers direct comparison and synthesis.

At the molecular level, discrepancies in enzymatic responses may relate to protein isoforms, post-translational modifications, and the influence of metabolites and cofactors. These mechanistic pathways remain insufficiently clarified and warrant further research.

In conclusion, physical activity—especially when appropriately tailored—appears to significantly modulate glycogen-related metabolic pathways, offering potential therapeutic value. However, future studies are needed to define precise parameters (intensity, duration, recovery) and to elucidate biochemical mechanisms more clearly, ideally through standardized methodologies and clinical trials.

ACKNOWLEDGMENTS

The authors would like to express their gratitude to Ecotec University, Milagro State University, and the Guayaquil University for their support and access to resources that made this study possible. Special thanks are extended to the communities and local stakeholders who participated in the research for their invaluable contributions. This article is a scientific product derived from the research project of the Faculty of Health Sciences and Human Development entitled "Nutritional Assessment and Its Association with Cardiovascular Risk Factors in Post-COVID Residents in Guayaquil", funded by Ecotec University under the 2023 internal research call.

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FINANCING
The authors declare that this research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

CONFLICT OF INTERESTS
The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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