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ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии
საქართველოს სამედიცინო სიახლენი

GEORGIAN MEDICAL NEWS

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GMN: Georgian Medical News is peer-reviewed, published monthly journal committed to promoting the science and art of medicine and the betterment of public health, published by the GMN Editorial Board since 1994. GMN carries original scientific articles on medicine, biology and pharmacy, which are of experimental, theoretical and practical character; publishes original research, reviews, commentaries, editorials, essays, medical news, and correspondence in English and Russian.

GMN is indexed in MEDLINE, SCOPUS, PubMed and VINITI Russian Academy of Sciences. The full text content is available through EBSCO databases.

GMN: Медицинские новости Грузии - ежемесячный рецензируемый научный журнал, издаётся Редакционной коллегией с 1994 года на русском и английском языках в целях поддержки медицинской науки и улучшения здравоохранения. В журнале публикуются оригинальные научные статьи в области медицины, биологии и фармации, статьи обзорного характера, научные сообщения, новости медицины и здравоохранения. Журнал индексируется в MEDLINE, отражён в базе данных SCOPUS, PubMed и ВИНТИ РАН. Полнотекстовые статьи журнала доступны через БД EBSCO.

GMN: Georgian Medical News – საქართველოს სამედიცინო სიახლენი – არის ყოველთვიური სამეცნიერო სამედიცინო რეცენზირებადი ჟურნალი, გამოიცემა 1994 წლიდან, წარმოადგენს სარედაქციო კოლეგიისა და აშშ-ის მეცნიერების, განათლების, ინდუსტრიის, ხელოვნებისა და ბუნებისმეტყველების საერთაშორისო აკადემიის ერთობლივ გამოცემას. GMN-ში რუსულ და ინგლისურ ენებზე ქვეყნდება ექსპერიმენტული, თეორიული და პრაქტიკული ხასიათის ორიგინალური სამეცნიერო სტატიები მედიცინის, ბიოლოგიისა და ფარმაციის სფეროში, მიმოხილვითი ხასიათის სტატიები.

ჟურნალი ინდექსირებულია MEDLINE-ის საერთაშორისო სისტემაში, ასახულია SCOPUS-ის, PubMed-ის და ВИНТИ РАН-ის მონაცემთა ბაზებში. სტატიების სრული ტექსტი ხელმისაწვდომია EBSCO-ს მონაცემთა ბაზებშიდან.

WEBSITE

www.geomednews.com

К СВЕДЕНИЮ АВТОРОВ!

При направлении статьи в редакцию необходимо соблюдать следующие правила:

1. Статья должна быть представлена в двух экземплярах, на русском или английском языках, напечатанная через **полтора интервала на одной стороне стандартного листа с шириной левого поля в три сантиметра**. Используемый компьютерный шрифт для текста на русском и английском языках - **Times New Roman (Кириллица)**, для текста на грузинском языке следует использовать **AcadNusx**. Размер шрифта - **12**. К рукописи, напечатанной на компьютере, должен быть приложен CD со статьей.

2. Размер статьи должен быть не менее десяти и не более двадцати страниц машинописи, включая указатель литературы и резюме на английском, русском и грузинском языках.

3. В статье должны быть освещены актуальность данного материала, методы и результаты исследования и их обсуждение.

При представлении в печать научных экспериментальных работ авторы должны указывать вид и количество экспериментальных животных, применявшиеся методы обезболивания и усыпления (в ходе острых опытов).

4. К статье должны быть приложены краткое (на полстраницы) резюме на английском, русском и грузинском языках (включающее следующие разделы: цель исследования, материал и методы, результаты и заключение) и список ключевых слов (key words).

5. Таблицы необходимо представлять в печатной форме. Фотокопии не принимаются. **Все цифровые, итоговые и процентные данные в таблицах должны соответствовать таковым в тексте статьи**. Таблицы и графики должны быть озаглавлены.

6. Фотографии должны быть контрастными, фотокопии с рентгенограмм - в позитивном изображении. Рисунки, чертежи и диаграммы следует озаглавить, пронумеровать и вставить в соответствующее место текста **в tiff формате**.

В подписях к микрофотографиям следует указывать степень увеличения через окуляр или объектив и метод окраски или импрегнации срезов.

7. Фамилии отечественных авторов приводятся в оригинальной транскрипции.

8. При оформлении и направлении статей в журнал МНГ просим авторов соблюдать правила, изложенные в «Единых требованиях к рукописям, представляемым в биомедицинские журналы», принятых Международным комитетом редакторов медицинских журналов - <http://www.spinesurgery.ru/files/publish.pdf> и http://www.nlm.nih.gov/bsd/uniform_requirements.html. В конце каждой оригинальной статьи приводится библиографический список. В список литературы включаются все материалы, на которые имеются ссылки в тексте. Список составляется в алфавитном порядке и нумеруется. Литературный источник приводится на языке оригинала. В списке литературы сначала приводятся работы, написанные знаками грузинского алфавита, затем кириллицей и латиницей. Ссылки на цитируемые работы в тексте статьи даются в квадратных скобках в виде номера, соответствующего номеру данной работы в списке литературы. Большинство цитированных источников должны быть за последние 5-7 лет.

9. Для получения права на публикацию статья должна иметь от руководителя работы или учреждения визу и сопроводительное отношение, написанные или напечатанные на бланке и заверенные подписью и печатью.

10. В конце статьи должны быть подписи всех авторов, полностью приведены их фамилии, имена и отчества, указаны служебный и домашний номера телефонов и адреса или иные координаты. Количество авторов (соавторов) не должно превышать пяти человек.

11. Редакция оставляет за собой право сокращать и исправлять статьи. Корректур авторам не высылаются, вся работа и сверка проводится по авторскому оригиналу.

12. Недопустимо направление в редакцию работ, представленных к печати в иных издательствах или опубликованных в других изданиях.

При нарушении указанных правил статьи не рассматриваются.

REQUIREMENTS

Please note, materials submitted to the Editorial Office Staff are supposed to meet the following requirements:

1. Articles must be provided with a double copy, in English or Russian languages and typed or computer-printed on a single side of standard typing paper, with the left margin of 3 centimeters width, and 1.5 spacing between the lines, typeface - **Times New Roman (Cyrillic)**, print size - 12 (referring to Georgian and Russian materials). With computer-printed texts please enclose a CD carrying the same file titled with Latin symbols.

2. Size of the article, including index and resume in English, Russian and Georgian languages must be at least 10 pages and not exceed the limit of 20 pages of typed or computer-printed text.

3. Submitted material must include a coverage of a topical subject, research methods, results, and review.

Authors of the scientific-research works must indicate the number of experimental biological species drawn in, list the employed methods of anesthetization and soporific means used during acute tests.

4. Articles must have a short (half page) abstract in English, Russian and Georgian (including the following sections: aim of study, material and methods, results and conclusions) and a list of key words.

5. Tables must be presented in an original typed or computer-printed form, instead of a photocopied version. **Numbers, totals, percentile data on the tables must coincide with those in the texts of the articles.** Tables and graphs must be headed.

6. Photographs are required to be contrasted and must be submitted with doubles. Please number each photograph with a pencil on its back, indicate author's name, title of the article (short version), and mark out its top and bottom parts. Drawings must be accurate, drafts and diagrams drawn in Indian ink (or black ink). Photocopies of the X-ray photographs must be presented in a positive image in **tiff format**.

Accurately numbered subtitles for each illustration must be listed on a separate sheet of paper. In the subtitles for the microphotographs please indicate the ocular and objective lens magnification power, method of coloring or impregnation of the microscopic sections (preparations).

7. Please indicate last names, first and middle initials of the native authors, present names and initials of the foreign authors in the transcription of the original language, enclose in parenthesis corresponding number under which the author is listed in the reference materials.

8. Please follow guidance offered to authors by The International Committee of Medical Journal Editors guidance in its Uniform Requirements for Manuscripts Submitted to Biomedical Journals publication available online at: http://www.nlm.nih.gov/bsd/uniform_requirements.html
http://www.icmje.org/urm_full.pdf

In GMN style for each work cited in the text, a bibliographic reference is given, and this is located at the end of the article under the title "References". All references cited in the text must be listed. The list of references should be arranged alphabetically and then numbered. References are numbered in the text [numbers in square brackets] and in the reference list and numbers are repeated throughout the text as needed. The bibliographic description is given in the language of publication (citations in Georgian script are followed by Cyrillic and Latin).

9. To obtain the rights of publication articles must be accompanied by a visa from the project instructor or the establishment, where the work has been performed, and a reference letter, both written or typed on a special signed form, certified by a stamp or a seal.

10. Articles must be signed by all of the authors at the end, and they must be provided with a list of full names, office and home phone numbers and addresses or other non-office locations where the authors could be reached. The number of the authors (co-authors) must not exceed the limit of 5 people.

11. Editorial Staff reserves the rights to cut down in size and correct the articles. Proof-sheets are not sent out to the authors. The entire editorial and collation work is performed according to the author's original text.

12. Sending in the works that have already been assigned to the press by other Editorial Staffs or have been printed by other publishers is not permissible.

**Articles that Fail to Meet the Aforementioned
Requirements are not Assigned to be Reviewed.**

ავტორთა საყურადღებო!

რედაქციაში სტატიის წარმოდგენისას საჭიროა დავიცვათ შემდეგი წესები:

1. სტატია უნდა წარმოადგინოთ 2 ცალად, რუსულ ან ინგლისურ ენებზე, დაბეჭდილი სტანდარტული ფურცლის 1 გვერდზე, 3 სმ სიგანის მარცხენა ველისა და სტრიქონებს შორის 1,5 ინტერვალის დაცვით. გამოყენებული კომპიუტერული შრიფტი რუსულ და ინგლისურენოვან ტექსტებში - **Times New Roman (Кириллица)**, ხოლო ქართულენოვან ტექსტში საჭიროა გამოვიყენოთ **AcadNusx**. შრიფტის ზომა – 12. სტატიას თან უნდა ახლდეს CD სტატიით.

2. სტატიის მოცულობა არ უნდა შეადგენდეს 10 გვერდზე ნაკლებს და 20 გვერდზე მეტს ლიტერატურის სიის და რეზიუმეების (ინგლისურ, რუსულ და ქართულ ენებზე) ჩათვლით.

3. სტატიაში საჭიროა გაშუქდეს: საკითხის აქტუალობა; კვლევის მიზანი; საკვლევი მასალა და გამოყენებული მეთოდები; მიღებული შედეგები და მათი განსჯა. ექსპერიმენტული ხასიათის სტატიების წარმოდგენისას ავტორებმა უნდა მიუთითონ საექსპერიმენტო ცხოველების სახეობა და რაოდენობა; გაუტკივარებისა და დაძინების მეთოდები (მწვავე ცდების პირობებში).

4. სტატიას თან უნდა ახლდეს რეზიუმე ინგლისურ, რუსულ და ქართულ ენებზე არანაკლებ ნახევარი გვერდის მოცულობისა (სათაურის, ავტორების, დაწესებულების მითითებით და უნდა შეიცავდეს შემდეგ განყოფილებებს: მიზანი, მასალა და მეთოდები, შედეგები და დასკვნები; ტექსტუალური ნაწილი არ უნდა იყოს 15 სტრიქონზე ნაკლები) და საკვანძო სიტყვების ჩამონათვალი (key words).

5. ცხრილები საჭიროა წარმოადგინოთ ნაბეჭდი სახით. ყველა ციფრული, შემავსებელი და პროცენტული მონაცემები უნდა შეესაბამებოდეს ტექსტში მოყვანილს.

6. ფოტოსურათები უნდა იყოს კონტრასტული; სურათები, ნახაზები, დიაგრამები - დასათაურებული, დანომრილი და სათანადო ადგილას ჩასმული. რენტგენოგრაფიის ფოტოსურათები წარმოადგინეთ პოზიტიური გამოსახულებით **tiff** ფორმატში. მიკროფოტოსურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შედეგების ან იმპრეგნაციის მეთოდი და აღნიშნოთ სურათის ზედა და ქვედა ნაწილები.

7. სამამულო ავტორების გვარები სტატიაში აღინიშნება ინიციალების თანდართვით, უცხოურისა – უცხოური ტრანსკრიპციით.

8. სტატიას თან უნდა ახლდეს ავტორის მიერ გამოყენებული სამამულო და უცხოური შრომების ბიბლიოგრაფიული სია (ბოლო 5-8 წლის სიღრმით). ანბანური წყობით წარმოდგენილ ბიბლიოგრაფიულ სიაში მიუთითეთ ჯერ სამამულო, შემდეგ უცხოელი ავტორები (გვარი, ინიციალები, სტატიის სათაური, ჟურნალის დასახელება, გამოცემის ადგილი, წელი, ჟურნალის №, პირველი და ბოლო გვერდები). მონოგრაფიის შემთხვევაში მიუთითეთ გამოცემის წელი, ადგილი და გვერდების საერთო რაოდენობა. ტექსტში კვადრატულ ფხიხლებში უნდა მიუთითოთ ავტორის შესაბამისი N ლიტერატურის სიის მიხედვით. მიზანშეწონილია, რომ ციტირებული წყაროების უმეტესი ნაწილი იყოს 5-6 წლის სიღრმის.

9. სტატიას თან უნდა ახლდეს: ა) დაწესებულების ან სამეცნიერო ხელმძღვანელის წარდგინება, დამოწმებული ხელმოწერითა და ბეჭდით; ბ) დარგის სპეციალისტის დამოწმებული რეცენზია, რომელშიც მითითებული იქნება საკითხის აქტუალობა, მასალის საკმაობა, მეთოდის სანდოობა, შედეგების სამეცნიერო-პრაქტიკული მნიშვნელობა.

10. სტატიის ბოლოს საჭიროა ყველა ავტორის ხელმოწერა, რომელთა რაოდენობა არ უნდა აღემატებოდეს 5-ს.

11. რედაქცია იტოვებს უფლებას შეასწოროს სტატია. ტექსტზე მუშაობა და შეჯერება ხდება საავტორო ორიგინალის მიხედვით.

12. დაუშვებელია რედაქციაში ისეთი სტატიის წარდგენა, რომელიც დასაბეჭდად წარდგენილი იყო სხვა რედაქციაში ან გამოქვეყნებული იყო სხვა გამოცემებში.

აღნიშნული წესების დარღვევის შემთხვევაში სტატიები არ განიხილება.

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AEROBIC AND RESISTANCE TRAINING SHOW DIVERGENT ASSOCIATIONS WITH INSULIN SENSITIVITY AND SHORT-TERM GLYCEMIC EXPOSURE IN PREDIABETES: A CROSS-SECTIONAL STUDY

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Abstract.

Background: Prediabetes is a reversible condition where lifestyle changes can prevent type 2 diabetes. While physical activity is crucial for management, traditional glycemic markers like HbA1c may not adequately reflect short-term exercise effects. Serum fructosamine, indicating average blood sugar levels over 1-3 weeks, is a more responsive biomarker but is under-researched in prediabetic populations, especially in the Gulf region.

Aim: To evaluate the association between regular physical activity and short-term glycemic control, indexed by serum fructosamine, in adults with prediabetes, and to examine whether exercise modality (aerobic, resistance, or combined) differentially influences short-term and long-term glycemic indices, body composition, and insulin sensitivity.

Methods: A cross-sectional analytical study at Thumbay Hospital and Thumbay Laboratories in Ajman, UAE, was conducted from November 2025 to April 2026, involving 78 adults aged 18–65 with prediabetes. Participants were stratified by physical activity levels using the IPAQ-SF. Various measurements, including anthropometric indices, fasting plasma glucose, HbA1c, fasting insulin, HOMA-IR, and serum fructosamine, were taken under standardized conditions. The Hemoglobin Glycation Index (HGI) was calculated based on the difference between observed and predicted HbA1c levels. Statistical analyses included Mann–Whitney U and Kruskal–Wallis tests for between-group comparisons and Spearman's coefficient for correlations, with a significance level set at $p < 0.05$.

Results: The study described a predominantly male (73.1%) and middle-aged cohort, with 82.1% classified as overweight or obese. Participants were 44.9% active and 55.1% sedentary, with no significant differences in fasting glucose, insulin, HOMA-IR, HbA1c, fructosamine, or BMI between the two groups. Within the active subgroup, aerobic exercisers ($n = 13$) had the lowest median BMI (25.7 kg/m^2) and fasting insulin but the highest fructosamine levels. However, these differences diminished in multivariable regression models, indicating that

male sex and BMI were key determinants of fasting insulin and HOMA-IR. Sedentary status was linked to higher HbA1c, while age correlated with higher fructosamine.

Conclusions: Self-reported physical activity did not affect glycemic outcomes in prediabetics, probably owing to misclassification and limited exercise benefits. Despite the short sample size, exploratory analysis suggested that aerobic exercise may improve insulin sensitivity and body composition while resistance training may improve glycemic exposure. Combining exercise may be beneficial, requiring additional study. In equivocal HbA1c findings, short-term glycemic indicators are important, but the absence of serum albumin measurement and albumin adjustment for fructosamine in exercise group comparisons limits interpretations.

Key words. Prediabetes, physical activity, exercise modality, fructosamine, HbA1c, HOMA-IR, hemoglobin glycation index, short-term glycemic control, United Arab Emirates.

Introduction.

The American Diabetes Association (ADA) defines prediabetes as a metabolic condition between normal blood sugar and type 2 diabetes. A fasting plasma glucose level of 100–125 mg/dL, a 2-hour post-load glucose level of 140–199 mg/dL after a 75 g oral glucose tolerance test (OGTT), or 5.7–6.4% HbA1c are indicators [1]. Global prediabetes rates are rising. The 11th Diabetes Atlas of the International Diabetes Federation estimates 589 million persons aged 20–79 have diabetes worldwide. Expect 853 million by 2050 [2]. According to new regional estimates, impaired glucose tolerance (IGT) and impaired fasting glucose (IFG) are still frequent worldwide [3]. One in three US individuals has prediabetes, and 10% develop T2DM without therapy [1].

Public health, health care, and the economy are harmed by this pandemic. Before diabetes symptoms show, prediabetes increases the risk of heart disease, chronic kidney disease, and mortality from any cause [1]. Both IFG and IGT may cause prediabetes. Therefore, each person's pathophysiology is unique. Example: IFG has higher hepatic insulin resistance, whereas IGT has more peripheral skeletal muscle insulin

resistance. Variety influences preventative measure selection and evaluation [3]. Lifestyle adjustments like organized exercise may alter the crucial prediabetes window.

Progressive insulin resistance and diminishing pancreatic β -cell activity lead to the development from normoglycemia to prediabetes and T2DM. In early prediabetes, compensatory hyperinsulinemia keeps blood glucose levels normal. Glucotoxicity, lipotoxicity, mitochondrial dysfunction, and persistent low-grade inflammation may reduce the β -cell's ability to release hormones, leading to postprandial and fasting hyperglycemia [1]. Long-term high glucose levels increase oxidative damage and inflammation. This cycle accelerates blood vessel destruction of all sizes [4].

One of the most essential molecules that removes glucose is GLUT-4. GLUT-4 can't migrate to the plasma membrane as quickly in skeletal muscle that doesn't react to insulin, making it tougher for insulin to assist cells absorb glucose. Myocytes struggle with glycogen production, mitochondrial oxidative phosphorylation, and lipid metabolism [5]. To understand how physical exercise decreases blood sugar, understand these methods. Exercise activates GLUT-4 translocation via non-insulin mechanisms.

Key randomized controlled studies show that glycemic goals reduce the incidence of diabetes sequelae such as retinopathy, nephropathy, neuropathy, and atherosclerotic cardiovascular disease [4]. This emphasizes the need of early and frequent blood sugar checks for prediabetics, particularly when considering lifestyle adjustments.

Exercise helps manage blood sugar and avoid type 2 diabetes. It affects insulin sensitivity, glucose metabolism, body composition, and heart health. Exercise helps blood sugar in several ways. It can increase skeletal muscle glucose uptake by moving GLUT-4 without insulin, increase mitochondrial energy production, reduce visceral fat, improve endothelial function, and increase pancreatic β -cell responsiveness [5,6].

Both persons with normal and high blood sugar levels may reduce their blood sugar with aerobic exercise (AE), resistance training (RT), and AE+RT. A complete network meta-analysis by Zhang et al. (2024) comprised several randomized controlled trials. Moderate-intensity aerobic exercise and low-to-moderate load resistance training improved glucose control, weight management, and cardiovascular risk factors best for prediabetics. Moderate-intensity aerobic and low-to-moderate resistance training were next best [7]. A 2025 network meta-analysis found that coupled AE+RT resulted in the greatest fasting blood glucose decline (standardized mean difference: -0.44), regardless of dosage. It also showed that 800 MET-minutes/week was best for reducing FBG in this group across all modalities [8].

Despite growing data, we still have substantial gaps in our knowledge. First, the ideal exercise prescription for prediabetes is still unknown, particularly when considering FBG and post-load glucose outcomes [5, 8]. Second, most prediabetes exercise intervention studies evaluate HbA1c and FBG to assess outcomes. Few studies have employed biomarkers to measure short-term glycemic responses in the initial weeks after exercise [5]. Third, more individuals are understanding that exercise affects blood sugar levels differently. Lewis et al.

(2025) reported that aerobic exercise may not improve HbA1c in a large proportion of patients. This emphasizes the need for sensitive early biomarkers [9]. These limitations suggest this research should use serum fructosamine as a short-term glycemic endpoint.

Glycated hemoglobin (HbA1c), which displays the average blood glucose level over the past 8 to 12 weeks (erythrocyte lifetime), is the major approach to monitor glycemic management in clinical practice and research. HbA1c is useful for long-term blood sugar trends, however its biological features prevent it from detecting short-term metabolic alterations caused by lifestyle modifications like exercise [4].

In addition to its time constraint, HbA1c is less dependable in persons with medical disorders that alter hemoglobin structure or red blood cell death. Regardless of the true mean glycemia, iron deficiency anemia, vitamin B12 or folate deficiency, hemolytic anemias, hemoglobinopathies (like sickle cell trait and other hemoglobin variants), chronic kidney disease, pregnancy, and drug treatments like erythropoietin can make HbA1c values appear higher or lower [4,10]. Iron deficiency anemia and hemoglobinopathies are frequent in Gulf and Middle Eastern healthcare, making this restriction crucial.

Short-term glycemic evaluation using serum fructosamine is now clinically important. Fructosamine is a ketoamine formed by non-enzymatic glycation of serum proteins, predominantly albumin. A reversible Schiff base production precedes an irreversible Amadori rearrangement. Fructosamine indicates mean glycemic exposure over 1–3 weeks, which matches albumin's 20-day turnover half-life [9]. Fructosamine is sensitive to blood sugar fluctuations before HbA1c increases due to its narrower reflection window. This makes it an ideal biomarker for early metabolic alterations from exercise or lifestyle modifications [10].

More data supports fructosamine as a therapeutically relevant glycemic indicator. The ARIC research found that fructosamine and glycated albumin alone caused new retinopathy and heart disease. They may predict future occurrences, not simply average glucose levels [10]. In a cross-sectional hospital-based study, John et al. (2023) found that albumin-corrected fructosamine had diagnostic efficacy similar to HbA1c but with lower reagent costs, improved automation on standard analyzers, and independence from red blood cell characteristics [11]. For laboratories with limited resources or many samples to handle, fructosamine assays are cheaper and simpler than HbA1c testing [10,11].

Fructosamine levels may be impacted by renal syndrome, protein-losing enteropathies, hepatic cirrhosis, and significant thyroid dysfunction, which modify blood protein concentration. High bilirubin and vitamin C levels may also hinder [10]. Thus, participants with such problems will be excluded from the trial, and blood albumin levels will be taken for all participants to contextualize fructosamine data.

In T2DM patients, supervised aerobic exercise reduced fructosamine levels by 14.2% after three weeks, proving its utility in the exercise-glycemia link. This shift was not yet detected by HbA1c, suggesting fructosamine may be an early, responsive indication of exercise-induced glycemic improvement [12].

While physical exercise is widely recommended as a cornerstone of diabetes prevention, evidence concerning its short-term effects on glycemic biomarkers particularly serum fructosamine in prediabetic individuals remains scarce. The prevailing reliance on HbA1c as the principal glycemic endpoint in both research and clinical settings may obscure early, biologically meaningful changes in glycemic control that arise from lifestyle modifications such as exercise. Given that HbA1c reflects an 8–12-week glycemic average and is susceptible to interference from hematological conditions common in Gulf and Middle Eastern populations, a more temporally sensitive biomarker is warranted for capturing the acute metabolic response to physical activity.

Against this background, the central question guiding this study was whether regular physical activity correlates with enhanced short-term glycemic control, as reflected by serum fructosamine levels, in adults with prediabetes. By examining fructosamine alongside fasting blood glucose, this study addresses a meaningful evidence gap and offers clinicians and researchers a more responsive framework for evaluating the early efficacy of exercise-based interventions. The findings are expected to support the integration of short-term glycemic biomarkers into structured diabetes prevention programs.

The primary objective of this study was to evaluate the association between regular physical activity and short-term glycemic control, as indicated by serum fructosamine levels, in adults with prediabetes. Secondary objectives were to compare short-term and long-term glycemic control markers represented by serum fructosamine and HbA1c, respectively alongside fasting blood glucose, fasting insulin, and HOMA-IR, between physically active and sedentary prediabetic individuals; to determine whether physical activity differentially influences short-term versus long-term glycemic indices; to examine the correlation between BMI and these glycemic indicators within the study cohort; and to investigate the differential association of exercise modality aerobic, resistance, or combined with short-term glycemic control.

Materials and Methods.

A cross-sectional analytical study was conducted to assess the association between physical activity and short-term glycemic control in adults with prediabetes.

The study was conducted at Thumbay Hospital and Thumbay Laboratories, Ajman, United Arab Emirates, over a six-month period between November 2025 to April 2026. The target population comprised adults aged 18 to 65 years with a confirmed diagnosis of prediabetes, encompassing both physically active and sedentary individuals. Participants were eligible if they had a confirmed diagnosis of prediabetes according to the American Diabetes Association (ADA) criteria — defined as fasting plasma glucose of 100–125 mg/dL, 2-hour post-load glucose of 140–199 mg/dL following a 75 g oral glucose tolerance test (OGTT), or glycated hemoglobin (HbA1c) of 5.7–6.4% and provided written informed consent. Participants were excluded if they had a confirmed diagnosis of type 1 or type 2 diabetes mellitus, chronic liver or renal disease, active pregnancy or lactation, or current use of pharmacological agents known to alter glucose metabolism, including metformin,

insulin, systemic corticosteroids, or other anti-hyperglycemic agents. Additionally, individuals with any acute or chronic condition deemed by the investigator to potentially confound glucose metabolism or impair physical activity assessment were excluded from participation.

The minimum required sample size was calculated using the correlation coefficient formula appropriate for cross-sectional studies, assuming a moderate anticipated correlation between physical activity and serum fructosamine ($r = 0.3$), a two-tailed significance level of $\alpha = 0.05$, and a statistical power of 80% ($\beta = 0.20$), according to the formula: $n = [(Z_{1-\alpha/2} + Z_{1-\beta}) / (0.5 \times \ln[(1+r)/(1-r)])]^2 + 3$. Substituting $Z_{1-\alpha/2} = 1.96$ and $Z_{1-\beta} = 0.84$ yielded a minimum sample of 85 participants. A 10% attrition allowance was applied to account for incomplete data or non-response, bringing the final recruitment target to 95 participants.

Sociodemographic data including age, sex, occupation, smoking status, and self-reported dietary habits were collected via a structured interviewer-administered questionnaire. Body weight (kg) and standing height (cm) were measured using calibrated scales and a stadiometer, and body mass index (BMI) was calculated as weight divided by height squared (kg/m^2).

Physical activity was assessed using the validated International Physical Activity Questionnaire – Short Form (IPAQ-SF). Exercise modality aerobic, resistance, or combined was additionally recorded from participant self-report to enable subgroup analysis in line with the secondary objectives of the study.

Following a minimum eight-hour overnight fast, venous blood samples were obtained from each participant. Two tubes were collected per participant: one sodium fluoride (NaF) plasma tube for fasting blood glucose determination and one serum separator tube (SST) for fructosamine analysis. NaF tubes were gently inverted 8–10 times immediately after collection to inhibit glycolysis and ensure pre-analytical stability, while SST tubes were allowed to clot at room temperature for 30 minutes prior to centrifugation. Both tube types were centrifuged at 3,000 rpm for 10 minutes, and the resulting plasma and serum aliquots were processed within two hours of collection.

Fasting plasma glucose (FPG) was measured on the Beckman Coulter AU/DxC 700 automated chemistry analyzer using the hexokinase enzymatic ultraviolet (UV) method. In this two-step coupled reaction, hexokinase catalyzes the phosphorylation of glucose to glucose-6-phosphate (G-6-P) in the presence of adenosine triphosphate (ATP) and magnesium ions (Mg^{2+}), after which G-6-P is oxidized by glucose-6-phosphate dehydrogenase (G-6-PDH) to 6-phosphogluconate, concomitantly reducing NAD^+ to NADH. The resultant increase in absorbance at 340 nm is directly proportional to the glucose concentration in the sample, and results are reported in milligrams per deciliter (mg/dL).

Glycated hemoglobin (HbA1c; Ref. No. B00389) was measured using turbidimetric immunoinhibition on the Beckman Coulter AU/DxC 700 platform. Fasting serum insulin (Ref. No. 33410) was quantified via a chemiluminescent immunoassay on a Beckman Coulter Dxl 800 analyzer, and insulin resistance was assessed using the Homeostatic Model Assessment for Insulin Resistance (HOMA-IR), calculated as: $\text{fasting insulin } (\mu\text{U/mL}) \times \text{fasting glucose } (\text{mg/dL}) / 405$.

Serum fructosamine was measured on the Roche Cobas c automated analyzer using the nitroblue tetrazolium (NBT) colorimetric kinetic reduction method (Roche Diagnostics reagent kit, Cat. No. 08105677190). In an alkaline environment, ketoamine groups present on glycated serum proteins predominantly albumin reduce NBT to a purple formazan compound, and the rate of color development is measured kinetically at 530–550 nm, being directly proportional to the fructosamine concentration. Two-point calibration was performed using the Precimat Fructosamine Calibrator (Cat. No. 11098993122), and internal quality control was verified against Precinorm (normal, Cat. No. 11098985122) and Precipath (pathological, Cat. No. 11174118122) fructosamine controls at the start of each analytical run. Results are reported in micromoles per liter ($\mu\text{mol/L}$). Fructosamine reflects mean glycemic exposure over the preceding 1–3 weeks, consistent with the approximately 20-day half-life of serum albumin, thereby serving as a short-term glycemic memory index that is analytically and temporally distinct from HbA1c.

The study was conducted in full accordance with the principles of the Declaration of Helsinki (2013 revision), and ethical approval was obtained from the Institutional Ethics Committee of Gulf Medical University and Thumbay Hospital prior to participant recruitment. All participants provided written informed consent before enrollment. Participant confidentiality was maintained throughout the study; all data were anonymized, stored in password-protected electronic files, and accessible exclusively to the principal investigator and supervisor, with no personally identifiable information retained within the analytical database.

Statistical analyses were performed using IBM SPSS Statistics, version 30.0 (IBM Corp., Armonk, NY, USA). Continuous variables were median with interquartile range (IQR) depending on distributional properties. The Shapiro–Wilk test was applied to assess the normality of continuous outcomes. Mann–Whitney U or Kruskal–Wallis test was applied for non-normally distributed data. Pearson's or Spearman's correlation coefficients were calculated to examine bivariate associations between BMI, and glycemic markers. Statistical significance was set at $p < 0.05$ (two-tailed) for all tests.

To address potential confounding by sex, age, and body mass index in the modality-stratified comparisons, multivariable linear regression models were fitted for each metabolic outcome (fasting glucose, fasting insulin, HOMA-IR, HbA1c, and fructosamine). Exercise modality was entered as a categorical predictor with aerobic training as the reference category, alongside activity status (active as reference, sedentary as comparator), sex (female as reference), age (continuous, per year), and BMI (continuous, per kg/m^2). Adjusted regression coefficients (β) with 95% confidence intervals and two-tailed p-values were reported. Statistical significance was set at $p < 0.05$.

Results.

Study Population and Descriptive Characteristics of Prediabetes patients:

The study cohort comprised 78 prediabetic patients with a mean age distribution heavily weighted toward middle-aged adults, with nearly 90% of participants falling below age 60 years

(46.2% in the 45–59 age range and 43.6% below age 45), while only 10.3% were over 60 years old. The sample demonstrated a pronounced male predominance, with males comprising 73.1% of the cohort compared to 26.9% female participants, which may limit generalizability to female prediabetic populations. Anthropometric characteristics revealed substantial metabolic burden, as 82.1% of the population was classified as overweight or obese, with overweight being the most prevalent category (42.3%), followed by obesity class I (24.4%), obesity class III (10.3%), and obesity class II (5.1%), while only 17.9% maintained normal body weight and no participants were underweight. Physical activity patterns indicated that the sedentary lifestyle was more prevalent than active engagement, with 55.1% classified as sedentary compared to 44.9% as active. Among those engaging in structured exercise ($n=35$), the exercise modalities were distributed across resistance training (17.9%, $n=14$), aerobic exercise (16.7%, $n=13$), and combined modalities (10.3%, $n=8$), suggesting heterogeneous exercise preferences within the active subgroup. Overall, this cohort represents a typical prediabetic population characterized by middle-aged individuals with pronounced overweight/obesity, male predominance, and mixed physical activity engagement, providing a representative sample for investigating metabolic and exercise-related biomarker associations in this at-risk population (Table 1).

Metabolic Characteristics of Prediabetes patients by Physical Activity Status:

Baseline metabolic biomarker profiles were remarkably similar between active and sedentary prediabetic patients,

Table 1. Demographic and anthropometric characteristics of the DM2 Study Population ($n = 78$).

Category	Level	N	%
Age group (WHO)	45–59	36	46.2
	<45	34	43.6
	>60	8	10.3
Gender	Female	21	26.9
	Male	57	73.1
BMI	Normal	14	17.9
	Underweight	0	0.0
	Obesity Class I	19	24.4
	Obesity Class II	4	5.1
	Obesity Class III	8	10.3
	Overweight	33	42.3
Physical Activity Status	Active	35	44.9
	Sedentary	43	55.1
Exercise Subgroup	Aerobic	13	16.7
	Combined	8	10.3
	Resistance	14	17.9

DM2: Type 2 Diabetes Mellitus; BMI: Body Mass Index; WHO: World Health Organization. Age groups are classified according to WHO criteria. BMI categories are defined as: Underweight ($<18.5 \text{ kg/m}^2$), Normal ($18.5\text{--}24.9 \text{ kg/m}^2$), Overweight ($25.0\text{--}29.9 \text{ kg/m}^2$), Obesity Class I ($30.0\text{--}34.9 \text{ kg/m}^2$), Obesity Class II ($35.0\text{--}39.9 \text{ kg/m}^2$), and Obesity Class III ($\geq 40.0 \text{ kg/m}^2$). No participants were classified as underweight. Physical activity status was determined based on self-reported participation in structured exercise. The Exercise Subgroup breakdown applies only to the Active group ($n = 35$). Values are expressed as frequencies (n) and percentages (%).

Table 2. Comparison of Metabolic Parameters Between Active and Sedentary Groups.

Parameters	Active (Median (IQR)) (N:35)	Sedentary (Median (IQR)) (N:43)	p-value
Weight (kg)	84.0 (74.9–93.0)	85.7 (73.0–105.15)	0.419
BMI (kg/m ²)	28.7 (26.2–29.85)	30.4 (26.2–34.75)	0.195
Fasting Glucose (mg/dl)	104.1 (97.0–111.0)	102.0 (95.0–112.1)	0.472
Fasting Insulin (uU/mL)	10.53 (7.22–15.43)	13.1 (8.0–17.14)	0.488
HOMA IR	2.9 (1.9–4.1)	3.5 (2.05–4.5)	0.523
A1C (%)	6.08 (5.94–6.22)	6.09 (5.96–6.31)	0.298
Fructosamine umol/L	269.0 (250.5–301.5)	269.5 (253.75–296.75)	0.971

Biochemical parameters comparing active vs. sedentary participants (Mann–Whitney U test) Values are presented as median (interquartile range). P values are from Mann–Whitney U tests (Active vs Sedentary).

Table 3. Metabolic and Biomarker Parameters by Exercise Modality.

Parameter	Aerobic (Median (IQR)) (N:13)	Resistance (Median (IQR)) (N:14)	Combined (Median (IQR)) (N:8)	p-value
Weight (kg)	74.0 (67.2–76.0)	89.0 (80.98–95.18)	93.65 (84.48–107.35)	0.002
BMI (kg/m ²)	25.7 (23.2–28.7)	29.59 (28.88–31.72)	30.35 (27.0–33.2)	0.005
Fasting Glucose (mg/dl)	103.0 (93.0–107.0)	109.5 (103.28–115.0)	102.15 (96.5–104.22)	0.08
Fasting Insulin (uU/mL)	7.02 (3.4–9.05)	12.22 (10.53–25.43)	11.41 (7.41–14.87)	0.016
HOMA IR	1.9 (0.9–2.1)	3.2 (2.9–7.4)	2.75 (1.9–3.5)	0.011
A1C (%)	6.08 (5.98–6.17)	6.1 (5.97–6.35)	5.85 (5.7–6.07)	0.309
Fructosamine umol/L	300.0 (273.0–308.0)	258.5 (234.5–271.25)	255.5 (248.5–280.75)	0.016
Gender	Male N: 8 (61.5%)	Male N:13 (92.9%)	Male N: 8 (100%)	p = 0.04
	Female N: 5 (38.5%)	Female N:1 (7.1%)	Female 0 (0.0%)	

Table3 presents a comprehensive comparison of metabolic and biochemical parameters across three distinct exercise modality groups in physically active prediabetes patients (n=35): aerobic training (n=13), resistance training (n=14), and combined modality training (n=8). Data are presented as medians with interquartile ranges (IQR) to account for non-normal distributions. Overall group differences were assessed using the Kruskal–Wallis test. Pvalue less than 0.05 is considered significant

Fisher's exact test was used for the sex × modality comparison because three of six expected cell counts were <5; the chi-square assumption was therefore not met.

Table 4. Hemoglobin Glycation Index by Physical Activity Status (Prediabetes).

Group	n	Median HGI	IQR (Q1 to Q3)	IQR Width	P.value
Active	35	-0.05	-0.22 to 0.12	0.34	p = 0.115
Sedentary	43	0.03	-0.09 to 0.21	0.31	

Median HGI and interquartile range (Q1 to Q3). P.value from Mann-Whitney U test (Active vs Sedentary).

Table 5. HGI by Exercise Modality.

Group	n	Median HGI	IQR (Q1 to Q3)	IQR Width	P.value
Aerobic	13	-0.03	-0.08 to 0.07	0.15	p = 0.45
Combined	8	-0.22	-0.41 to -0.0	0.41	
Resistance	14	-0.06	-0.21 to 0.24	0.45	

Median HGI and interquartile range (Q1 to Q3). P.value from Kruskal-Wallis test across exercise modalities (Aerobic, Resistance, Combined).

Table 6. Hemoglobin Glycation Index and Correlations with Biomarkers in Prediabetes.

Biomarker	r	p	Direction	Significance	r	p	Direction	Significance
	Active (N: 35)				Sedentary (N: 43)			
Fasting Glucose (mg/dl)	0.076	0.664	positive	non-significant	-0.026	0.87	negative	non-significant
Fasting Insulin (uU/mL)	-0.076	0.663	negative	non-significant	0.215	0.167	positive	non-significant
HOMA IR	-0.084	0.632	negative	non-significant	0.215	0.167	positive	non-significant
Fructosamine	0.183	0.291	positive	non-significant	-0.207	0.188	negative	non-significant

r: Pearson/Spearman correlation coefficient; p: p-value; HOMA-IR: Homeostatic Model Assessment of Insulin Resistance; N: number of participants. Positive direction indicates a direct relationship between the biomarker and the outcome variable; negative direction indicates an inverse relationship. Statistical significance was set at $p < 0.05$. All correlations were non-significant in both the Active (n = 35) and Sedentary (n = 43) subgroups. Fasting glucose is expressed in mg/dL; fasting insulin in μ U/mL.

Table 7. Multivariate linear regression analyses evaluating the association between exercise type and metabolic outcomes, adjusted for age, sex, and BMI. Aerobic exercise and female sex were used as reference categories. Values represent β coefficients (95% confidence intervals) with corresponding *p*-values.

Predictor	Fasting Glucose (mg/dL)	Fasting Insulin (μ U/mL)	HOMA-IR	HbA1c (%)	Fructosamine (μ mol/L)
Resistance	+9.76 (-4.56, 24.08) <i>p</i> =0.172	-0.50 (-7.49, 6.49) <i>p</i> =0.884	+0.18 (-1.78, 2.14) <i>p</i> =0.850	+0.30 (-0.03, 0.63) <i>p</i> =0.069	-14.58 (-43.70, 14.54) <i>p</i> =0.311
Combined	-1.45 (-16.74, 13.83) <i>p</i> =0.846	-4.32 (-11.78, 3.15) <i>p</i> =0.244	-1.10 (-3.19, 0.99) <i>p</i> =0.289	+0.03 (-0.32, 0.38) <i>p</i> =0.857	-9.76 (-40.85, 21.34) <i>p</i> =0.523
Sedentary (No exercise)	+8.82 (-7.25, 24.90) <i>p</i> =0.268	+0.42 (-7.43, 8.27) <i>p</i> =0.912	+0.33 (-1.87, 2.52) <i>p</i> =0.761	+0.58 (0.22, 0.95) <i>p</i> =0.003	+3.26 (-29.44, 35.96) <i>p</i> =0.839
Sex (Male)	-0.74 (-13.39, 11.92) <i>p</i> =0.905	+7.68 (1.50, 13.85) <i>p</i> =0.017	+1.86 (0.13, 3.59) <i>p</i> =0.036	+0.02 (-0.27, 0.31) <i>p</i> =0.891	+0.74 (-24.99, 26.48) <i>p</i> =0.953
Age (per year)	-0.09 (-0.63, 0.45) <i>p</i> =0.733	+0.08 (-0.19, 0.34) <i>p</i> =0.549	+0.01 (-0.07, 0.08) <i>p</i> =0.835	+0.004 (-0.008, 0.017) <i>p</i> =0.471	+1.74 (0.64, 2.84) <i>p</i> =0.003
BMI (kg/m^2)	-0.67 (-1.77, 0.43) <i>p</i> =0.219	+0.91 (0.37, 1.45) <i>p</i> =0.002	+0.20 (0.05, 0.35) <i>p</i> =0.011	-0.01 (-0.04, 0.01) <i>p</i> =0.281	+0.51 (-1.73, 2.75) <i>p</i> =0.643

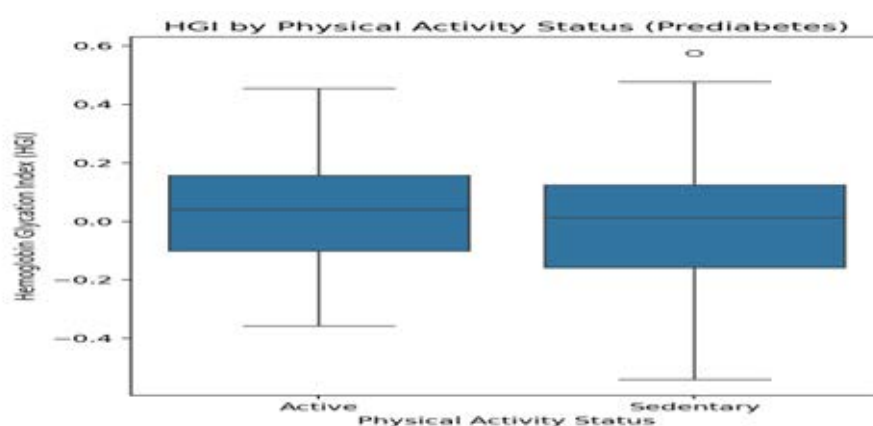


Figure 1. Hemoglobin Glycation Index Distribution by Physical Activity Status. The graph employs box-and-whisker to illustrate the median, interquartile range, and overall distribution of HGI values.

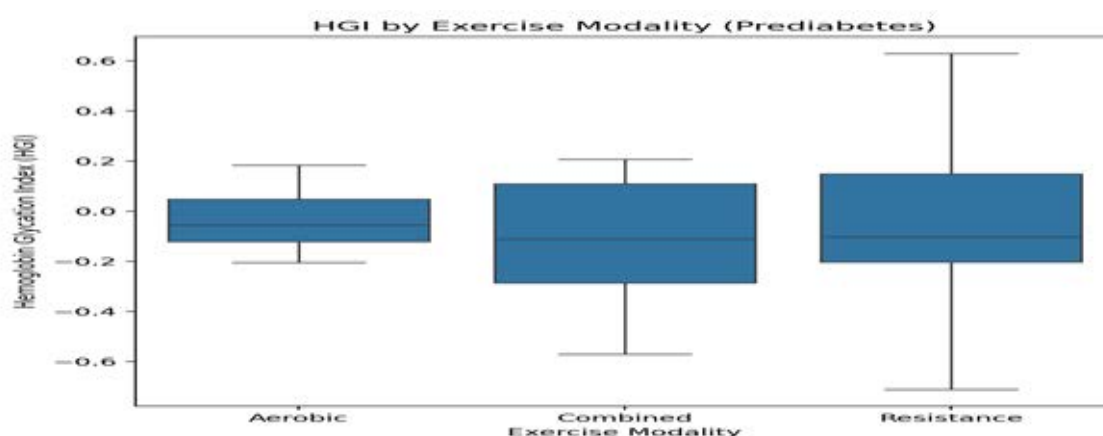


Figure 2. HGI by Exercise Modality. Displays box and whisker plot of Hemoglobin Glycation Index by different exercise modalities.

with no statistically significant differences observed across the comprehensive panel of assessed parameters (all $p>0.05$). Weight and BMI distributions were comparable between groups, with active individuals showing median BMI of 28.7 kg/m^2 (IQR: 26.2–29.85) versus 30.4 kg/m^2 (IQR: 26.2–34.75) in the sedentary group, though neither reached statistical significance. Glycemic control markers including fasting glucose (104.1 vs 102.0 mg/dl), A1C (6.08% vs 6.09%), and Fructosamine (269.0 vs 269.5 $\mu\text{mol}/\text{L}$) demonstrated virtually superimposable medians

between groups, suggesting that baseline glucose homeostasis was equitably impaired across both cohorts (Table 2).

Metabolic and Biomarker Parameters by Exercise Modality in Prediabetes:

Among active participants, three exercise categories were compared: aerobic ($n=13$), combined ($n=8$), and resistance ($n=14$) training. Using the Kruskal–Wallis test, significant differences were identified in several parameters. Weight was

significantly lower in the aerobic group (74.0 kg, IQR 67.2–76.0) compared to resistance (89.0 kg, IQR 80.98–95.18) and combined (93.65 kg, IQR 84.48–107.35) groups ($p=0.002$). Correspondingly, BMI differed significantly across groups ($p=0.005$), with aerobic participants showing a median BMI of 25.7 kg/m² (IQR 23.2–28.7) versus resistance participants with 29.59 kg/m² (IQR 28.88–31.72).

Glycemic and metabolic indices showed exercise-specific patterns. Fasting insulin was significantly lower in the aerobic group (7.02 μ U/mL, IQR 3.4–9.05) compared to resistance (12.22 μ U/mL, IQR 10.53–25.43, $p=0.016$). Correspondingly, HOMA-IR was lowest in aerobic participants (1.9, IQR 0.9–2.1) and highest in resistance participants (3.2, IQR 2.9–7.4, $p=0.011$). Fructosamine levels were significantly higher in the aerobic group (300.0 μ mol/L, IQR 273.0–308.0) compared to both combined (255.5 μ mol/L, IQR 248.5–280.75) and resistance groups (258.5 μ mol/L, IQR 234.5–271.25, $p=0.015$) (Table 3).

Hemoglobin Glycation Index (HGI) by Physical Activity Status:

HGI stratified by physical activity status revealed minimal differences between groups without achieving statistical significance ($p=0.115$). The active population ($n=35$) demonstrated a median HGI of -0.05 with an interquartile range from -0.22 to 0.12 (IQR width=0.34). The sedentary population ($n=43$) exhibited a median HGI of 0.03 with an interquartile range from -0.09 to 0.21 (IQR width=0.31). The absolute difference in median HGI between groups was 0.08 units, with the active group showing slightly lower values compared to the sedentary group. The interquartile ranges were comparable between groups, with the active group demonstrating a marginally wider distribution (0.34 units) relative to the sedentary group (0.31 units). Both groups displayed overlapping interquartile ranges, with the sedentary group's IQR entirely contained within a shift of the active group's distribution toward higher values (Table 4 and Figure 1).

Hemoglobin Glycation Index by Exercise Modality:

HGI stratified by exercise modality demonstrated variable distributions across training types without achieving statistical significance ($p=0.45$). The aerobic exercise group ($n=13$) exhibited a median HGI of -0.03 with an interquartile range from -0.08 to 0.07 (IQR width=0.15), representing the most tightly distributed values. The combined exercise group ($n=8$) demonstrated the lowest median HGI of -0.22 with an interquartile range from -0.41 to -0.0 (IQR width=0.41), indicating a wider spread and more negative values. The resistance training group ($n=14$) showed a median HGI of -0.06 with an interquartile range from -0.21 to 0.24 (IQR width=0.45), displaying the widest interquartile range among the three exercise modalities. The absolute difference in median HGI between the highest (aerobic, -0.03) and lowest (combined, -0.22) values was 0.19 units. The combined exercise group exhibited the most leftward-shifted distribution toward negative HGI values, while the aerobic group showed values clustered near zero. Interquartile range widths varied considerably across modalities, with aerobic demonstrating substantially narrower dispersion (0.15 units) compared to both combined (0.41 units)

and resistance (0.45 units) training groups (Table 5 and Figure 2).

Hemoglobin Glycation Index and Correlations with Biomarkers:

Correlations between biomarkers and HGI stratified by physical activity status revealed were non-significant (Table 6).

Multivariable regression analysis adjusting for sex, age, and BMI:

To formally evaluate whether the modality-specific patterns described above persisted after adjustment for sex, age, and BMI and to interrogate the sex imbalance identified across modality subgroups multivariable linear regression models were fitted for each metabolic outcome (Table X). With aerobic training as the reference modality and active status as the reference for activity, neither resistance training nor combined training was independently associated with statistically significant differences in fasting glucose, fasting insulin, HOMA-IR, HbA1c, or fructosamine after adjustment. The previously observed univariate fructosamine difference between modalities was attenuated and lost statistical significance in the adjusted model (resistance vs aerobic: $\beta = -14.58 \mu\text{mol/L}$, 95% CI -43.70 to 14.54, $p = 0.311$; combined vs aerobic: $\beta = -9.76 \mu\text{mol/L}$, 95% CI -40.85 to 21.34, $p = 0.523$), as did the differences in fasting insulin and HOMA-IR. Sedentary status remained independently associated with higher HbA1c ($\beta = +0.58\%$, 95% CI 0.22 to 0.95, $p = 0.003$) but not with any other glycemic or insulin-sensitivity outcome. Male sex was independently associated with higher fasting insulin ($\beta = +7.68 \mu\text{U/mL}$, 95% CI 1.50 to 13.85, $p = 0.017$) and higher HOMA-IR ($\beta = +1.86$, 95% CI 0.13 to 3.59, $p = 0.036$), confirming sex as a substantive determinant of insulin-sensitivity readouts in this cohort. BMI was independently associated with higher fasting insulin ($\beta = +0.91 \mu\text{U/mL per kg/m}^2$, 95% CI 0.37 to 1.45, $p = 0.002$) and HOMA-IR ($\beta = +0.20 \text{ per kg/m}^2$, 95% CI 0.05 to 0.35, $p = 0.011$). Age was independently associated with higher fructosamine concentrations ($\beta = +1.74 \mu\text{mol/L per year}$, 95% CI 0.64 to 2.84, $p = 0.003$).

Discussion.

This cross-sectional study set out to determine whether habitual physical activity was associated with short-term glycemic control, indexed by serum fructosamine, in 78 prediabetic adults attending Thumbay Hospital and Thumbay Laboratories in Ajman. Three findings structure the interpretation. First, the overall comparison between active ($n = 35$) and sedentary ($n = 43$) participants revealed no statistically significant differences in fructosamine, HbA1c, fasting glucose, fasting insulin, HOMA-IR, BMI, or the hemoglobin glycation index (HGI). Second, when the active group was stratified by exercise modality, aerobic training was associated with significantly lower body weight, BMI, fasting insulin, and HOMA-IR than resistance or combined training, yet paradoxically with the highest serum fructosamine concentrations. Third, HbA1c and HGI distributions were statistically indistinguishable across both activity status and exercise modality. Taken together, these findings refine rather than refute the role of exercise in prediabetes management. The modality-stratified comparisons

should, however, be interpreted as exploratory and hypothesis-generating: subgroup sample sizes were small (aerobic $n = 13$, resistance $n = 14$, combined $n = 8$), and nonparametric tests at this scale are particularly vulnerable to influential observations and inflated Type I error. The patterns described below are accordingly framed as candidate signals warranting confirmatory testing in adequately powered prospective designs, not as established modality-specific effects.

Reconciling the null active-versus-sedentary signal with the interventional literature:

The absence of significant glycemic separation between active and sedentary groups is, at first reading, discordant with the weight of randomized evidence documenting exercise-induced glycemic improvement in prediabetes [5,7,8]. Three methodological considerations reconcile the present result with contemporary findings. First, meta-analytic evidence is drawn predominantly from structured, supervised programmes of ≥ 12 weeks at defined intensities, whereas the current study classified habitual activity using the IPAQ-SF a self-report tool with documented validity correlations of only 0.09–0.39 against accelerometry and a tendency to overestimate moderate-to-vigorous activity and misclassify respondents on either side of the activity threshold [13,14]. In populations characterized by high baseline sedentariness and climatic or cultural constraints on outdoor activity, such as those of the United Arab Emirates and wider Gulf region, self-report misclassification is expected to attenuate true between-group differences [15].

Second, the metabolic phenotype of the "active" category did not meaningfully diverge from the sedentary category: median BMI (28.7 vs 30.4 kg/m²) and HOMA-IR (2.9 vs 3.5) were similar, suggesting the active classification was insufficient to confer a distinct cardiometabolic state. This is consistent with the 2025 umbrella review by Chung and colleagues of 48 meta-analyses, which affirmed that exercise-induced glycemic effects, though robust across modalities, are of small magnitude in prediabetes and require sustained dose–response thresholds of roughly 800 MET-min/week for fasting glucose and 750–880 MET-min/week for HbA1c to be clearly detectable [8,16]. Third, the INTENSITY trial of Hrubeniuk et al. reported that after 16 weeks of supervised aerobic training, only 4.1% of prediabetic and type 2 diabetic participants were classifiable as responders on HbA1c criteria, while 87.7% remained "unclear" [17]. Individual heterogeneity in glycemic adaptation is thus the norm, and a moderately sized cross-sectional cohort cannot reasonably be expected to resolve an aggregate exercise signal that a supervised trial struggles to demonstrate at the group level.

The aerobic–fructosamine paradox:

The most striking observation was that aerobic exercisers displayed the most favourable body composition and insulin-sensitivity indices the lowest median BMI (25.7 kg/m²), fasting insulin (7.02 μ U/mL), and HOMA-IR (1.9) yet the highest median fructosamine (300 μ mol/L), compared with 258.5 μ mol/L in resistance and 255.5 μ mol/L in combined groups. Several mutually non-exclusive explanations merit consideration.

Fructosamine reflects non-enzymatic glycation of circulating

proteins, predominantly albumin, and is therefore sensitive not only to mean glycemia but also to serum albumin concentration and protein turnover [10,11]. Aerobic training, particularly when sustained at higher weekly volumes, alters protein kinetics and can transiently modify albumin distribution through plasma-volume expansion [18]. Because the present study reported uncorrected fructosamine, part of the between-modality difference may reflect protein-compartment dynamics rather than glycation per se. Routine albumin correction in future analyses is strongly advised.

A second, more physiologically interesting possibility relates to the temporal window captured by fructosamine. Fructosamine integrates glycemic exposure over the preceding 1–3 weeks and includes postprandial excursions, whereas HOMA-IR is a static fasting index [19]. Aerobic exercise, especially in the fasted state or when followed closely by carbohydrate intake, can produce transient catecholamine-driven hepatic glucose output and amplified glycemic responses to subsequent feeding during the early recovery period [20]. Resistance training, by contrast, chronically expands the insulin-sensitive glucose storage compartment through skeletal muscle hypertrophy and enhanced glycogen synthase activity, thereby blunting cumulative postprandial glycemic exposure [21,22]. The observed pattern better fasting insulin sensitivity in aerobic exercisers, but lower cumulative glycation in resistance trainers is therefore biologically plausible and reflects the distinct physiological signatures of each modality. Aerobic training preferentially improves fasting-state insulin action, while resistance training preferentially attenuates the integrated postprandial glycemic burden that fructosamine is sensitive to.

A third consideration is reverse causation, which a cross-sectional design cannot exclude. Adults who adopt aerobic training may have done so in response to perceived or clinically measured glycemic deterioration, producing a confounding-by-indication signal that biases the aerobic group toward higher fructosamine. A fourth, and arguably the most important, consideration is residual confounding by dietary intake, which was not assessed in this study. Fructosamine integrates glycemic exposure over the preceding 1–3 weeks and is therefore highly sensitive to recent carbohydrate load, dietary glycemic index, total energy intake, and meal-timing patterns variables that can shift mean glycation independently of habitual physical activity. Exercise modality and dietary behaviour are seldom independent in free-living populations: individuals engaged in regular aerobic training frequently increase carbohydrate intake to support endurance fueling and post-exercise glycogen replenishment, whereas those favouring resistance training more commonly adopt higher-protein, comparatively lower-carbohydrate dietary patterns aimed at supporting lean-mass accretion. If such modality-linked dietary differences were present in the current cohort, they could plausibly generate higher cumulative postprandial glycemic exposure in the aerobic subgroup despite their more favourable fasting insulin sensitivity, thereby producing precisely the dissociation observed between HOMA-IR and fructosamine. Because dietary intake was neither quantified by food diary nor estimated through a validated food-frequency instrument, this potential confounder cannot be adjusted for analytically, and its contribution to the aerobic–

fructosamine paradox cannot be excluded. This limitation should be regarded as a primary alternative explanation for the observed pattern, and routine dietary assessment minimally a 3-day food record or short FFQ capturing carbohydrate quantity and quality should be considered mandatory in any prospective replication of these analyses. Finally, the aerobic subgroup was small ($n = 13$), and although the Kruskal–Wallis test reached nominal significance ($p = 0.016$), the estimate is vulnerable to a handful of influential observations. Given the small subgroup size, the susceptibility of the Kruskal–Wallis test to influential observations at this scale, and the elevated Type I error risk associated with multiple subgroup comparisons, the aerobic–fructosamine signal must be treated as a hypothesis to be tested in adequately powered prospective cohorts rather than as an established biological phenomenon.

The distribution of sex across exercise modalities was significantly non-random (Fisher's exact $p = 0.04$), with female participants concentrated in the aerobic group (5/13, 38.5%), markedly underrepresented in the resistance group (1/14, 7.1%), and entirely absent from the combined group (0/8). This pattern is consistent with well-documented sex-related preferences in self-selected exercise modality, whereby women are more likely to engage in aerobic activity and men in resistance training. Because sex differences in body composition, lean mass, hormonal milieu, and protein turnover all plausibly modify both insulin sensitivity and the dynamics of the circulating albumin pool that determines fructosamine concentrations, the observed modality-specific differences in BMI, fasting insulin, HOMA-IR, and fructosamine cannot be cleanly disentangled from the underlying sex imbalance between groups. The aerobic group's combination of lower BMI and fasting insulin together with paradoxically higher fructosamine could therefore reflect, at least in part, the higher proportion of female participants in that subgroup rather than a modality-specific metabolic signature. Sex-stratified analyses were not feasible at the present sample size, and the modality-specific findings should accordingly be interpreted as confounded by sex distribution; future work must ensure balanced recruitment across sex strata and pre-specify sex-stratified or sex-adjusted modality comparisons.

Modality-dependent effects on body composition and insulin action:

The finding that aerobic training was associated with substantially lower body weight, BMI, fasting insulin, and HOMA-IR than either resistance or combined training is internally consistent with the mechanistic literature. Aerobic training improves whole-body insulin sensitivity through increased skeletal muscle capillarization, up-regulation of GLUT4 expression, and mitochondrial biogenesis driven by the AMPK–PGC-1 α pathway, and it is particularly effective at mobilizing visceral adipose tissue [21–23]. Resistance training induces smaller reductions in fat mass but produces its glycemic benefits principally through skeletal muscle hypertrophy, enhanced post-receptor insulin signalling (IRS-1/PI3K/Akt), and expansion of intramuscular glycogen storage [22]. The 2024 network meta-analysis by Zhang et al. concluded that combined moderate-intensity aerobic and low-to-moderate-load resistance training produced the greatest composite improvements in

HbA1c, BMI, and cardiovascular risk factors in prediabetes [7], while the updated 2025 Bayesian synthesis by Zhang et al. identified aerobic exercise as superior for lowering HbA1c and 2-hour post-meal glucose, and combined training for fasting glucose reduction at an optimum of ~ 800 MET-min/week [8]. The cross-sectional patterns observed here are compatible with, but do not constitute evidence for, these mechanistic distinctions. Given the small modality-specific sample sizes and the susceptibility of nonparametric subgroup tests to outlier influence, the present results should be read as exploratory observations that generate hypotheses for confirmatory trials, rather than as demonstrations that aerobic and resistance training act through distinct metabolic axes in this population.

Two adjusted findings nonetheless merit comment. First, sedentary status remained independently associated with higher HbA1c after full adjustment ($\beta = +0.58\%$, $p = 0.003$), a signal that the unadjusted pairwise comparison did not detect and that is consistent with the long-term integrated nature of the HbA1c readout. Second, age was independently associated with higher fructosamine concentrations ($\beta = +1.74$ $\mu\text{mol/L}$ per year, $p = 0.003$), reinforcing the importance of age adjustment in any subsequent fructosamine-based investigation in this population. These two adjusted associations are the most defensible findings of the present analysis and warrant prioritization in confirmatory work.

HbA1c inertia and the rationale for short-term biomarkers:

Despite modality-dependent divergence in body composition, insulin sensitivity, and fructosamine, HbA1c was indistinguishable across all groups (medians 5.85–6.10%; $p = 0.309$). This reinforces the principal rationale for incorporating short-term glycemic biomarkers into lifestyle-intervention research: HbA1c integrates glycemic exposure across the full erythrocyte lifespan (~ 120 days) and is therefore biologically incapable of resolving short-term behavioral effects. The Gulf region compounds this limitation, since iron-deficiency anemia and inherited haemoglobinopathies both prevalent in the local population distort HbA1c independently of glycemia [10,19]. Rossi et al. earlier demonstrated that an 8-week supervised aerobic programmes produced detectable fructosamine reductions in type 2 diabetes patients without concurrent HbA1c change [12], and the present finding of fructosamine divergence by modality in the absence of HbA1c change extends that logic to prediabetes. When the research question concerns the acute or sub-acute metabolic response to a lifestyle behaviour, fructosamine optimally with albumin correction offers greater temporal resolution than HbA1c.

Hemoglobin glycation index: interpretation of the null signal:

The HGI, defined as the residual between observed and FPG-predicted HbA1c, quantifies inter-individual variation in the propensity to glycate Haemoglobin at a given glucose exposure, and has been linked to insulin resistance, hepatic steatosis, cardiovascular events, and all-cause mortality in both prediabetic and diabetic cohorts [24–26]. In the present study, HGI did not differ by activity status ($p = 0.115$) or exercise modality ($p = 0.45$), and no significant correlations were observed with fasting glucose, insulin, HOMA-IR, or fructosamine in either subgroup. This null signal is consistent with the reference-population

work of Hempe and colleagues, who observed that HGI is not independently associated with plasma insulin, HOMA-IR, or 2-hour OGTT glucose when assessed in demographically diverse NHANES participants [27]. Within a clinically homogeneous prediabetic sample featuring narrow HbA1c and FPG ranges, HGI appears unlikely to unmask exercise-related differences that the primary glycemic biomarkers do not already reveal, and its utility is probably greater in wider glycemic strata or in specifically interrogating cardiovascular risk phenotypes.

Regional context.

The Gulf Cooperation Council countries carry among the highest age-adjusted rates of diabetes and prediabetes worldwide, with combined prediabetes-and-diabetes prevalence in the UAE adult population estimated at approximately 30% [15,28]. Physical inactivity contributes substantially: only around 40% of men and 27% of women in the Gulf meet WHO recommendations of ≥ 150 minutes of moderate-to-vigorous activity per week [15]. Set within this epidemiological landscape, the present study offers a locally relevant phenotypic snapshot of active and sedentary prediabetic adults attending a tertiary center, and contributes regionally sparse evidence on the behaviour of short-term glycemic biomarkers.

Strengths.

The study employed a rigorously standardized pre-analytical protocol, with immediate sodium-fluoride glycolysis inhibition, two-hour processing windows, platform-specific validated assays, and two-point calibration with bi-level quality control for fructosamine measurement. It measured short-term (fructosamine), long-term (HbA1c), and fasting (FPG, insulin, HOMA-IR) glycemic indices concurrently, enabling multi-axis metabolic characterisation rather than reliance on a single biomarker. Stratification by self-reported exercise modality allowed an exploratory modality-specific analysis, and inclusion of HGI added a complementary glycation-phenotype dimension.

Limitations.

Several limitations warrant acknowledgement. Most fundamentally, the cross-sectional design is intrinsically misaligned with the temporal property that motivates the use of fructosamine as a biomarker. Fructosamine derives its analytical value from sensitivity to short-term shifts in glycemic exposure over the preceding 1–3 weeks, a property that can only be fully exploited within a longitudinal framework incorporating repeated pre- and post-intervention sampling. The present design captures a single static reflection of recent cumulative glycemia and cannot, by construction, demonstrate the responsiveness of fructosamine to behaviour change; the modality-specific differences observed here therefore represent inter-individual covariation in habitual glycemic exposure rather than evidence of biomarker responsiveness per se. A prospective, repeated-measures design with baseline, mid-intervention, and post-intervention fructosamine sampling such as that employed by Moura et al. in type 2 diabetes [12] is required to establish Fructosamine's utility as a proximal endpoint for exercise-based interventions in prediabetes, and this should be regarded as the principal methodological priority for follow-up work. The cross-sectional architecture also

precludes causal inference more broadly; associations observed here should be interpreted as covariation. Physical activity was measured by self-report using the IPAQ-SF without objective corroboration, with the attendant risks of recall and social-desirability bias [13,14]. The sample was male-predominant (73.1%) and middle-aged, limiting generalizability to women and older adults, and exercise-modality subgroups were small, particularly the combined group ($n = 8$). The combination of small subgroup sizes and nonparametric subgroup testing materially elevates the risk of Type I error and renders the modality-stratified comparisons vulnerable to influential observations; these analyses should accordingly be regarded as exploratory and hypothesis-generating rather than as definitive evidence of modality-specific effects, and no inferential weight is placed on them in the absence of independent replication. Fructosamine was not corrected for serum albumin, and dietary intake a major short-term determinant of glycemic exposure was neither quantified nor controlled. Finally, participants were recruited by convenience from a single tertiary center in Ajman, raising the possibility of selection bias.

Although multivariable regression adjusting for sex, age, and BMI substantially attenuated the univariate modality-specific differences and provides a more conservative characterisation of the data, the adjusted analyses remain limited by the small overall sample, the small sex-imbalanced modality subgroups, and the absence of albumin and dietary covariates; the adjusted estimates should accordingly be regarded as informative but not definitive.

Clinical implications and future research directions.

The modality-specific patterns documented here, while hypothesis-generating, align with current international guidance advocating combined aerobic and resistance training in prediabetes [7], because each modality appears to operate on distinct metabolic axes: aerobic training on body composition and fasting insulin sensitivity, and resistance training on cumulative glycemic exposure as reflected by fructosamine. Future work should: (i) adopt prospective randomized designs with objective activity monitoring (accelerometry or heart-rate telemetry) and quantified dietary intake; (ii) incorporate albumin-corrected fructosamine and, where feasible, glycated albumin to disentangle protein-pool from glycation effects; (iii) integrate continuous glucose monitoring to capture postprandial excursions that fasting indices and HbA1c cannot resolve; and (iv) ensure balanced recruitment across sex and age strata. In the Gulf and wider Eastern Mediterranean region, where HbA1c interpretation is confounded by widespread haemoglobinopathies and iron deficiency [10,15], routine incorporation of short-term glycemic markers into prediabetes evaluation deserves serious operational consideration. The modality-specific patterns documented here are hypothesis-generating rather than confirmatory, and their clinical translation must be framed accordingly. Nonetheless, the divergent metabolic signatures align with current international guidance advocating combined aerobic and resistance training in prediabetes [7], because each modality appears to operate on distinct metabolic axes: aerobic training was associated with a more favourable body-composition and fasting-insulin-sensitivity profile, consistent

with its established effects on visceral adiposity and AMPK-driven mitochondrial adaptation, while resistance training was associated with lower cumulative glycemic exposure as indexed by fructosamine, plausibly reflecting expansion of the insulin-sensitive glycogen storage compartment. The two modalities therefore appear complementary rather than redundant, strengthening the physiological case for prescribing them in combination.

Several methodological priorities follow from the limitations of the present work. Most fundamentally, the field requires prospective designs capable of exploiting the temporal property that motivates fructosamine in the first place; pre–post or randomized repeated-measures protocols with serial fructosamine sampling at baseline, three weeks, and post-intervention would formally test biomarker responsiveness, a question cross-sectional architecture cannot resolve. Subsequent work should also move beyond self-reported activity to accelerometry or heart-rate telemetry, routinely correct fructosamine for serum albumin and ideally complement it with glycated albumin, integrate continuous glucose monitoring to capture postprandial excursions invisible to fasting indices and HbA1c, and ensure balanced recruitment across sex and age strata.

Finally, the regional context warrants explicit operational reflection. In the Gulf and wider Eastern Mediterranean region, where iron deficiency and inherited haemoglobinopathies are sufficiently prevalent to compromise HbA1c interpretation in a non-trivial proportion of clinical encounters [10,15], the case for short-term glycemic markers extends beyond research utility into routine practice. Albumin-corrected fructosamine, deployed alongside rather than in place of HbA1c, would offer a temporally complementary readout that is analytically robust to the most common local haematological confounders, and operationalizing this dual-biomarker approach within structured diabetes prevention programmes represents the most immediately actionable clinical implication of this work.

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Conflict of interest.

The authors declare that there is no conflict of interest.

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