

Journal of Diabetes Education

To Dispel Darkness Of Diabetes

DIET MANAGEMENT ►



◀ EXERCISE

MEDICATION ►



**An Official Publication of
Association of Diabetes Educators
(India)**

JOURNAL OF DIABETES EDUCATION

To Dispel Darkness of Diabetes

Vol. 13

Number 2

April-June, 2025

EDITOR-IN-CHIEF

Hemraj Chandalia

EXECUTIVE EDITOR

Sonal Chandalia

EDITORIAL ASSISTANT

Blasee R Fernaandes

EDITORIAL COMMITTEE

Kavita Gupta

Niti Desai

Salome Benjamin

Shobha Udipi

Shaival Chandalia

ASSOCIATION OF DIABETES EDUCATORS

PRESIDENT

Shobha Udipi, Mumbai

IMMEDIATE PAST PRESIDENT

Hemraj Chandalia, Mumbai

VICE PRESIDENT

Sonal Chandalia, Mumbai

Niti Desai, Mumbai

SECRETARY

Shubhda Bhanot, Delhi

TREASURER

Shaival Chandalia, Mumbai

EXECUTIVE MEMBERS

Kavita Gupta, Nagpur

Meenakshi Bajaj, Chennai

Priyangee Lahiry, Kolkatta

Savita Singh, Delhi

Sheryl Salis, Mumbai

CONTENTS

1. **Exploring the Gaps: Misconceptions in Glycemic Monitoring Technologies – SMBG, HbA1c and CGMS** 02
Vijay Negalur
2. **Individualizing Exercise Prescription in People with Diabetes** 07
Srilaxmi Aishwarya Chavali, Tvisha Parikh
3. **Nutritional Strategies to Reduce Post-Prandial Hyperglycemia** 15
Priyangee Lahiry
4. **Questions and Answers** 21
5. **Recipes** 23
6. **How Knowledgeable Are You?** 25
7. **Myths and Facts**..... 26

EXPLORING THE GAPS: MISCONCEPTIONS IN GLYCEMIC MONITORING TECHNOLOGIES – SMBG, HbA1c AND CGMS

Vijay Negalur*

INTRODUCTION

Glycemic monitoring is essential for advanced diabetes management, directly shaping patient outcomes and minimizing complications. Evolving technologies in glycemic assessment play a critical role in precision diabetes care. This monitoring can be performed through various methods, including self-monitoring blood glucose (SMBG) and continuous glucose monitoring system (CGMS), each offering unique benefits (1). Effective glycemic control is paramount in diabetes management, significantly influencing the prevention of complications, providing real-time feedback and enhancing the understanding of glycemic variability (2). Rigorous glycemic control has been shown to reduce the risk of microvascular complications such as retinopathy, nephropathy and neuropathy. Landmark studies, including the Diabetes Control and Complications Trial (DCCT) and the United Kingdom Prospective Diabetes Study (UKPDS), demonstrated that improved glycemic control can decrease the incidence of these complications by 25% to 75%, underscoring the importance of maintaining optimal blood glucose levels (3). The use of CGMS provides real-time feedback on glucose levels, allowing for immediate adjustments in treatment. This capability is crucial for preventing hypoglycemic events and maintaining time-in-range (TIR). Research indicates that patients using CGMS can achieve a 10-20% increase in TIR compared to those relying solely on traditional monitoring methods (4). Monitoring glycemic variability is essential for tailoring diabetes management strategies. Studies have shown that high glycemic variability is associated with an increased

risk of complications, emphasizing the need for consistent monitoring to identify patterns and make informed treatment decisions. By understanding these fluctuations, healthcare providers can better manage diabetes and improve patient outcomes (2).

For glycemic monitoring, the primary tools include SMBG, glycated hemoglobin (HbA1c) and CGMS (1). The HbA1c testing is a key indicator of long-term glucose control, with frequent assessments (every 3 months) ensuring that glycemic targets are met. Maintaining an HbA1c below 7% significantly reduces microvascular complications by 25–30% (1). Self-monitoring of blood glucose, for instance, provides discrete blood glucose readings but may yield inaccurate results due to user errors such as improper meter usage or inadequate hand hygiene. It was found that 19% of patients experienced inaccuracy rates exceeding 15% in SMBG readings (5). The CGMS enhances diabetes management by providing real-time insights, enabling immediate adjustments to glucose fluctuations and reducing hypoglycemia risk (<70 mg/dL). Patients achieving 70% TIR often maintain an HbA1c of around 7%. Regular monitoring empowers patients, improving self-management and reducing severe hypoglycemia by up to 85%, ultimately leading to better long-term health outcomes (6). Individuals with Type 1 diabetes mellitus (T1DM) using CGMS experienced a reduction in HbA1c levels by 0.4% over 24 weeks, reflecting better glycemic management, which underscores the importance of incorporating CGMS into routine diabetes care to enhance patient safety and treatment efficacy (7).

* Physician Diabetologist and Metabolic Physician, Dr Negalur's Diabetes and Thyroid Specialities Centre, Thane, India; Honorary Mentor, Post Graduate Course in Diabetes, D.Y. Patil University, Navi Mumbai, India.
Email ID: negalurvijay@gmail.com

Glucose monitoring technologies are vital in diabetes management, yet they face several challenges and misconceptions. For instance, establishing glucose meter accuracy is challenging due to differences between whole blood samples used by meters and serum-based standards, as well as the instability of glucose in whole blood. Additionally, HbA1c may not always provide a precise reflection of individual glycemic control, as a given HbA1c level can correspond to a wide range of glucose concentrations and glucose variability (8). Continuous glucose monitoring systems provide real-time data but often lack the accuracy required to detect hypoglycemia reliably a frequent complication of diabetes treatment. Furthermore, CGMS systems measure glucose levels in interstitial fluid rather than directly in the blood, resulting in a 5 to 20-minute lag between actual blood glucose levels and those detected by the CGMS (9).

Overcoming these limitations is pivotal for improving the accuracy and reliability of glucose monitoring technologies. This review article provides a critical analysis of the inherent fallacies in commonly used glycemic monitoring methods, including SMBG, HbA1c and CGMS, with an emphasis on their clinical implications.

FALLACIES OF HbA1c

Over-Reliance on HbA1c and Conditions Impacting HbA1c Levels

Relying exclusively on HbA1c as the primary measure of diabetes control can be problematic because various factors can influence HbA1c levels, potentially leading to inaccurate blood glucose assessments. So, it is important to adopt a more comprehensive approach that considers additional factors alongside HbA1c for accurate diabetes management (10).

Specific Conditions That Affect HbA1c Accuracy

A falsely low HbA1c level can be caused by various factors such as high altitude, pregnancy, blood loss, blood transfusions, erythropoietin

treatment, iron supplements, hemolytic anemia, chronic kidney disease, liver cirrhosis, alcohol use, sickle cell anemia and spherocytosis (11,12). On the other hand, a falsely elevated HbA1c level can be caused by low iron levels in the blood, which may result from conditions such as iron deficiency anemia, anemia due to infection, thalassemia, B12 deficiency or anemia caused by tumors. Other factors include hypertriglyceridemia and organ transplantation and increased glycation in certain ethnic groups (13).

Drug Interference and Supporting Data

Certain widely used medications can alter HbA1c levels, making them appear higher or lower than the actual blood glucose levels. Vitamin C supplementation may either raise or lower HbA1c levels, depending on the testing method. Additionally, drugs like immunosuppressants and protease inhibitors can occasionally cause a falsely elevated HbA1c value (10).

Pooled data from some of the recent studies have revealed a wide variation in average glucose levels for a specific HbA1c value. For instance, an HbA1c of 8.0% (64 mmol/mol) corresponds to mean glucose concentrations ranging from 155 to 218 mg/dL, which overlaps significantly with the ranges for HbA1c of 7.0% at 128 to 190 mg/dL and HbA1c of 9.0% at 182 to 249 mg/dL. As a result, an HbA1c of 8.0% could reflect either good, fair or poor glycemic control, depending on the corresponding mean glucose levels, which can range from 128 to 249 mg/dL (8). The ADAG (A1c-Derived Average Glucose) study discovered that measures of glucose variability, as well as average and postprandial glycemia, are strongly correlated within each category. However, variability indexes show only a weak correlation with other categories, suggesting they provide distinct information. Fasting blood glucose is not a reliable indicator of overall glycemic control. Additionally, preprandial glucose levels have a greater influence on HbA1c than postprandial glucose levels (14).

Alternatives to HbA1c

When HbA1c fails to provide an accurate representation of glycemic control, it is preferable to use an alternative index. Possible alternatives include fructosamine, glycated albumin, 1,5-anhydroglucitol (1,5-AG) and CGMS (15).

FALLACIES OF SELF-MONITORING OF BLOOD GLUCOSE

Self-monitoring of blood glucose has been scrutinized for its effectiveness, particularly in non-insulin-treated Type 2 diabetes mellitus (T2DM) patients, but its effectiveness can be influenced by various factors, often leading to fallacies in results. Inaccurate readings are a major issue, which may arise from external factors such as temperature or humidity affecting the glucose meters or from improper sampling techniques such as using insufficient blood volumes or poor strip placement (16). These inaccuracies can lead to incorrect insulin dosing, risking episodes of hypoglycemia or hyperglycemia, which negatively impact long-term health outcomes, including cardiovascular and neurological complications (17). Another significant challenge is patient adherence. Many patients struggle with consistent SMBG due to the pain, inconvenience or anxiety caused by frequent fingerstick tests. Inadequate education about SMBG use is also a contributing factor, as patients and healthcare teams may not fully understand the nuances of proper meter calibration or interpreting results. This gap in knowledge can result in inappropriate adjustments to therapy, exacerbating the disease (5). To mitigate these issues, choosing reliable and validated glucose meters is crucial. Meters must meet ISO standards, ensuring accuracy and reproducibility across a range of testing conditions. Recent systematic reviews and meta-analyses reveal that while SMBG can lead to a modest reduction in HbA1c levels (approximately 0.30% to 0.35%), its clinical utility remains questionable due to inconsistent study designs and participant responses (18,19). Moreover, SMBG is often perceived as burdensome, leading to inconsistent adherence among users. Studies

show that many patients experience technical inaccuracies with their devices, which can further undermine the reliability of SMBG results (19,20). Additionally, reliance on HbA1c alone to assess glycemic control can be misleading, as it does not capture the individual variability in glucose levels effectively. These factors collectively challenge the efficacy and practicality of SMBG in diabetes management (8,18).

FALLACIES OF CONTINUOUS GLUCOSE MONITORING SYSTEM

Continuous glucose monitoring devices allow people to check their glucose levels in real-time throughout the day. The CGMS helps users take control of their diabetes management daily by providing them with real-time glucose information to guide their decisions (21). However, like any technology, CGMS devices have their limitations. The constant flow of glucose data can feel overwhelming, making it hard for users to spot trends. Frequent false alarms can cause alarm fatigue, where users start ignoring alerts and might miss important changes in glucose levels (22).

Hypoglycemia occurs when blood glucose levels drop below 70 mg/dL (23), but some individuals may not experience any typical symptoms. This condition, known as hypoglycemia unawareness, is more common in people with long-term diabetes or frequent hypoglycemic episodes. Without warning signs, individuals are at a higher risk of severe hypoglycemia (24). Regular glucose monitoring and working closely with healthcare providers are important to manage and prevent such episodes.

A discrepancy between sensor glucose values and capillary glucose readings occurs because a sensor measures glucose levels in the interstitial fluid (fluid surrounding cells) which is slightly delayed compared to the blood glucose measured by a finger prick. This means the sensor reading will often be slightly lower when blood sugar is rising and slightly higher when it's falling rapidly; this difference is considered normal and expected when using CGMS (25).

The accuracy of CGMS can be affected by various factors. Proper sensor placement and insertion are crucial to ensure reliable readings. Body movements, skin issues like sweating, even pressure from clothing or sleeping positions can interfere with the device. Hydration levels, blood flow to the tissues and temperature changes also play a role, as they impact the glucose levels in the interstitial fluid where the sensor works. Activities like exercise, certain medications and recent meals can further influence the readings. Finally, the condition of the sensor itself, including proper calibration and care, is essential for accurate results. Understanding these factors helps in getting the best out of CGMS and making their data more dependable (26,27).

Inaccuracies in CGMS devices (specifically the CGMS by Medtronic) led to 35% of cases with the CGMS graphs being interpreted inconsistently by clinicians. This lack of consistency could result in 17% of clinical recommendations being incorrect, potentially impacting patient care (28). Calibration and regular validation are essential for ensuring the accuracy and reliability of CGMS. Calibration aligns the CGMS readings with actual blood glucose levels, helping to reduce errors and improve the precision of glucose tracking.

PRACTICAL CONSIDERATION AND RECOMMENDATION

Importance of Individualized Glycemic Monitoring Strategies

Glycemic management should be tailored to each individual rather than adopting a 'one-size-fits-all' approach. Achieving personalized glycemic goals is essential to prevent both microvascular and macrovascular complications of diabetes, necessitating proactive efforts to overcome therapeutic inertia. When setting glycemic targets, numerous factors need to be considered. While the ADA provides general recommendations suitable for many individuals, it underscores the significance of customizing goals based on individual characteristics. A patient-centred approach that incorporates shared decision-making can better address unique needs

and preferences while accounting for factors that influence the risks and benefits of therapy. This personalized strategy not only enhances patient engagement but also fosters greater self-efficacy in managing diabetes (1).

COMBINING SMBG, HbA1c AND CGMS FOR COMPREHENSIVE DIABETES MANAGEMENT

A holistic approach to diabetes management involves integrating SMBG, HbA1c and CGMS. The HbA1c reflects long-term glycemic control but fails to capture daily fluctuations or hypoglycemia. The SMBG provides real-time insights into glucose trends but is episodic and patient-dependent. The CGMS offers continuous, dynamic data, highlighting glycemic variability and trends over time. Combining these tools provides a comprehensive understanding of glycemic control, enabling tailored treatment adjustments. This integration particularly benefits high-risk patients or during therapy changes, optimizes outcomes (7,23).

Effective diabetes management requires addressing misconceptions among patients and healthcare teams through education. Misunderstandings, such as over-reliance on HbA1c or fear of hypoglycemia, can hinder optimal care. Patient education should focus on the proper use of monitoring tools, the significance of glycemic variability and the impact of lifestyle on glucose levels. Training on advancements such as CGMS and shared decision-making is essential for healthcare teams to ensure evidence-based practices. Bridging these knowledge gaps improves adherence, patient engagement and therapeutic outcomes, fostering collaboration between patients and providers (29).

CONCLUSION

A balanced approach to glycemic monitoring is crucial for effective diabetes management. While HbA1c, SMBG and CGMS each have limitations, integrating these tools with patient education and physician awareness of their appropriate use can enhance accuracy and

treatment decisions, ultimately improving health outcomes.

REFERENCES

1. American Diabetes Association Professional Practice Committee. 6. Glycemic Goals and Hypoglycemia: Standards of Care in Diabetes—2025. *Diabetes Care*. 2025;48:S128-45.
2. Bin Rakhis SA, AlDuwayhis NM, Aleid N et al. Glycemic Control for Type 2 Diabetes Mellitus Patients: A Systematic Review. *Cureus*. 2022;14:e26180.
3. Standards of Medical Care in Diabetes—2016 Abridged for Primary Care Providers. *Clin Diabetes Publ Am Diabetes Assoc*. 2016;34:3-21.
4. Beck RW, Riddlesworth T, Ruedy K et al. Effect of Continuous Glucose Monitoring on Glycemic Control in Adults with Type 1 Diabetes Using Insulin Injections: The DIAMOND Randomized Clinical Trial. *JAMA*. 2017;317:371-8.
5. Klonoff DC. Benefits and Limitations of Self-Monitoring of Blood Glucose. *J Diabetes Sci Technol Online*. 2007;1:130-2.
6. Juvenile Diabetes Research Foundation Continuous Glucose Monitoring Study Group. Sustained Benefit of Continuous Glucose Monitoring on A1C, Glucose Profiles and Hypoglycemia in Adults with Type 1 Diabetes. *Diabetes Care*. 2009;32:2047-9.
7. Battelino T, Danne T, Bergenstal RM et al. Clinical Targets for Continuous Glucose Monitoring Data Interpretation: Recommendations From the International Consensus on Time in Range. *Diabetes Care*. 2019;42:1593-603.
8. Beck RW, Connor CG, Mullen DM et al. The Fallacy of Average: How Using HbA1c Alone to Assess Glycemic Control Can Be Misleading. *Diabetes Care*. 2017;40:994-9.
9. Oriot P, Klipper dit kurz N, Ponchon M et al. Benefits and limitations of hypo/hyperglycemic alarms associated with continuous glucose monitoring in individuals with diabetes. *Diabetes Epidemiol Manag*. 2023;9:100125.
10. Heinemann L, Freckmann G. Quality of HbA1c Measurement in the Practice: The German Perspective. *J Diabetes Sci Technol*. 2015;9:687-95.
11. Guo W, Zhou Q, Jia Y et al. Increased Levels of Glycated Hemoglobin A1c and Iron Deficiency Anemia: A Review. *Med Sci Monit*. 2019;25:8371-8.
12. Pilla R, Palleti SK, Rayala R et al. Glycated Hemoglobin (HbA1c) Variations in Nondiabetics with Nutritional Anemia. *Cureus*. 2020;12:e11479.
13. Shepard JG, Airee A, Dake AW et al. Limitations of A1c Interpretation. *South Med J*. 2015;108:724-9.
14. Borg R, Kuenen JC, Carstensen B et al. Associations Between Features of Glucose Exposure and A1C. *Diabetes*. 2010;59:1585-90.
15. Radin MS. Pitfalls in Hemoglobin A1c Measurement: When Results may be Misleading. *J Gen Intern Med*. 2014;29:388-94.
16. Franciosi M, Pellegrini F, De Berardis G et al. The Impact of Blood Glucose Self-Monitoring on Metabolic Control and Quality of Life in Type 2 Diabetic Patients: An urgent need for better educational strategies. *Diabetes Care*. 2001;24:1870-7.
17. Davis WA, Bruce DG, Davis TME. Is self-monitoring of blood glucose appropriate for all type 2 diabetic patients? The Fremantle Diabetes Study. *Diabetes Care*. 2006;29:1764-70.
18. Zou Y, Zhao S, Li G et al. Efficacy and Frequency of Self-monitoring of Blood Glucose in Non-insulin-Treated T2D Patients: a Systematic Review and Meta-analysis. *J Gen Intern Med*. 2023;38:755-64.
19. Holmes-Truscott E, Baptista S, Ling M et al. The Impact of Structured Self-Monitoring of Blood Glucose on Clinical, Behavioral and Psychosocial Outcomes Among Adults with Non-Insulin-Treated Type 2 Diabetes: A Systematic Review and Meta-Analysis. *Front Clin Diabetes Healthc*. 2023; 4: 1177030.
20. Schnell O, Alawi H, Battelino T et al. Self-Monitoring of Blood Glucose in Type 2 Diabetes: Recent Studies. *J Diabetes Sci Technol*. 2013;7:478-88.
21. Rodbard D. Continuous Glucose Monitoring: A Review of Successes, Challenges and Opportunities. *Diabetes Technol Ther*. 2016;18:S3-13.
22. Stone JY, Bailey TS. Benefits and Limitations of Continuous Glucose Monitoring In Type 1 Diabetes. *Expert Rev Endocrinol Metab*. 2020;15:41-9.
23. American Diabetes Association Professional Practice Committee. 7. Diabetes Technology: Standards of Care in Diabetes—2025. *Diabetes Care*. 2024;48:S146-66.
24. Hölzen L, Schultes B, Meyhöfer SM et al. Hypoglycemia Unawareness—A Review on Pathophysiology and Clinical Implications. *Biomedicines*. 2024;12:391.
25. Siegmund T, Heinemann L, Kolassa R et al. Discrepancies Between Blood Glucose and Interstitial Glucose—Technological Artifacts or Physiology: Implications for Selection of the Appropriate Therapeutic Target. *J Diabetes Sci Technol*. 2017;11:766-72.
26. Ahmed HB, Serener A. Effects of External Factors in CGMS Sensor Glucose Concentration Prediction. *Procedia Comput Sci*. 2016;102:623-9.
27. Bellido V, Freckman G, Pérez A et al. Accuracy and Potential Interferences of Continuous Glucose Monitoring Sensors in the Hospital. *Endocr Pract Off J Am Coll Endocrinol Am Assoc Clin Endocrinol*. 2023;29:919-27.
28. Metzger M, Leibowitz G, Wainstein J et al. Reproducibility of Glucose Measurements Using the Glucose Sensor. *Diabetes Care*. 2002;25:1185-91.
29. Powers MA, Bardsley J, Cypress M et al. Diabetes Self-Management Education and Support in Type 2 Diabetes: A Joint Position Statement of the American Diabetes Association, the American Association of Diabetes Educators and the Academy of Nutrition and Dietetics. *J Acad Nutr Diet*. 2015;115:1323-34.

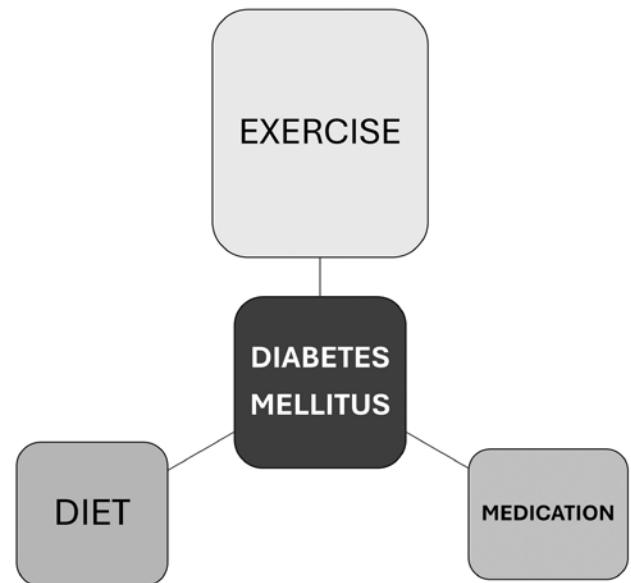
INDIVIDUALIZING EXERCISE PRESCRIPTION IN PEOPLE WITH DIABETES

Srilaxmi Aishwarya Chavali*, Tvisha Parikh**

INTRODUCTION

Diabetes mellitus (DM) is a chronic metabolic condition that affects 460 million people globally and is estimated to affect 700 million by 2045 (1). Diabetes was initially considered a disease of the affluent “Western” countries, but it has now spread globally, affecting epidemic proportions in countries such as India. India ranks second in the global diabetes epidemic, with an estimated 101 million people suffering from Type 2 diabetes mellitus (T2DM) and 136 million from prediabetes (1,2). By 2045, 134 million Indians are expected to be affected by diabetes. As of 2020, 11.4% of Indian adults were affected with diabetes and 15.3% were affected with prediabetes, accounting for one in six global cases and its vast diaspora amplifies its impact on global health, economy and society (2,3). Longstanding DM directly affects the cardiovascular (two-to-three-fold increased risk of heart attacks and stroke), renal (primarily kidney failure) and immune systems of the body (2,4). Reduced physical activity and diminished cardiovascular and musculoskeletal fitness, both modifiable risk factors, contribute to poor glycemic control and the onset of T2DM (5). Traditional DM management has predominantly relied on pharmacological interventions and dietary modifications, often neglecting the critical role of exercise (4,6). Exercise is one of the cornerstones for the most beneficial non-pharmacological treatment of DM as it increases insulin sensitivity, lowers blood sugar levels, reduces body fat, improves cardiovascular function, improves blood pressure and has psychological benefits such as reducing depression, anxiety and improving sleep quality (7).

Figure 1
The Three Cornerstones of Diabetes Management



Despite the well-established benefits of exercise, physical activity remains underutilized as a primary intervention. Evidence from the Diabetes Prevention Programme suggests that exercise, diet and behavioural modification may be superior to medication in individuals with impaired glucose tolerance to delay or prevent the development of T2DM (8). The rising prevalence of sedentary lifestyles, exacerbated by urbanization and technological advancements, further compounds physical inactivity. This shift highlights the urgent need for targeted interventions to mitigate its negative impact. Recognizing its significance, the U.S. Food and Drug Administration is considering approving exercise therapy as a prescribed treatment for diabetes (9). For exercise to be effective,

* Sports and Exercise Medicine Physician, Sir HN Reliance Foundation Hospital, Mumbai, India.
Email ID: chavaliaishwarya95@gmail.com

** Sports and Exercise Medicine Physician, Sir HN Reliance Foundation Hospital, Mumbai, India.
Email ID: drtvisha@gmail.com

it must be adhered to and a tailored exercise prescription is crucial. Uncertainty about the optimal timing, frequency, intensity, duration of exercise, psychological constraints including time limitations, sedentary lifestyles and mood instability often hinder exercise initiation and continuation. Sometimes, insufficient knowledge among the care team regarding current guidelines in diabetes and the limited facilities for such care further contribute to poor adherence. Leading organizations such as the American College of Sports Medicine (ACSM) and the American Diabetes Association (ADA) emphasize on exercise frequency, intensity and modality to improve glycemic control (1,9). The ultimate goal of exercise intervention for diabetes management is to implement individualized exercise prescription that integrate patient education, optimize glycemic control and reduce the risk of cardiovascular diseases.

EFFECT OF EXERCISE ON METABOLISM AND REGULATION OF BLOOD SUGAR

The major way that exercise has a positive effect on DM is through its action on insulin action (9). Exercise significantly influences insulin action in muscle and liver, both acutely and over the long term. Aerobic exercise acutely enhances muscle glucose uptake by up to fivefold through insulin-independent pathways. Post-exercise, glucose uptake remains elevated via insulin-independent mechanisms for about two hours and insulin-dependent mechanisms for up to 48 hours, depending on exercise type, duration and intensity (7,11). This enhancement aids muscle glycogen replenishment (12,13). Short, high-intensity activities (~20 minutes) can improve insulin sensitivity for 24 hours, while even low-intensity aerobic exercise lasting 60 minutes improves insulin action in insulin-resistant individuals for at least a day (10,14,17).

Chronic training increases muscle capillarity, oxidative capacity, lipid metabolism and insulin signalling proteins, but these adaptations reverse with inactivity (16,17). Aerobic and resistance exercises improve insulin sensitivity in skeletal

muscle, adipose tissue and liver, even without weight loss (17,18). Aerobic training boosts insulin action in prediabetes and T2DM patients, with improvements proportional to exercise volume. Even low-volume training (400 kcal/week) shows benefits, with the most significant gains observed in those with higher baseline insulin resistance (10). Resistance training similarly improves insulin action and high-intensity interval training (HIIT) may surpass continuous aerobic exercise in effectiveness for individuals with diabetes (19). Combining aerobic and resistance training yields the most substantial benefits (19).

EXERCISE PRESCRIPTION

There are four crucial aspects of any exercise program: the type of exercise, the frequency, duration and intensity of exercise.

Type of Exercise: The ADA and the ACSM highlights that a combination of aerobic and resistance exercise modalities may be more effective than one alone (10,19). Flexibility and balance training are also recommended for older adults with T2DM. Additionally, Diabetes Canada emphasizes that supervised exercise programs yield better outcomes for T2DM patients (20,21). Traditionally, aerobic exercise has been studied more than resistance training. However, with 80% of the T2DM population being overweight or obese (22) with associated comorbidities and mobility problems, resistance training may be more doable than aerobic training as the latter has higher demands of volume and intensity (23). Growing evidence shows the benefits of resistance training and HIIT in T2DM. (19,24,25).

Frequency, Duration and Intensity of Exercise:

Aerobic activities such as walking, cycling, jogging or swimming should be performed for a minimum of 150-300 minutes per week at moderate intensity (3-7 days/week) or 75-150 minutes of high (vigorous) intensity to improve glycemic control, support weight management and reduce cardiovascular risk (19). Moderate-to high-intensity exercise (4-5 days/week) appears to reduce abdominal, mainly visceral fat in adults

with T2DM and may also reduce the metabolic risk (19). Resistance training, such as lifting weights or exercises with resistance bands, should be performed 3 days/week and can complement aerobic activities to enhance muscle strength (19,24). Flexibility and balance exercises such as yoga or tai chi, can improve joint range of motion and gait while reducing fall risk (19). People with diabetes should ensure no more than two consecutive days without activity to ensure consistent glycemic improvement (7,19).

The intensity of exercise is subjective for each person. Individuals with diabetes, especially those who are inactive, should begin with low-to-moderate intensity activities due to potential cardiovascular risks and higher likelihood of injury with high intensity. Furthermore, one must recognize their body's responses during and after exercise, typically assessed using a maximum heart rate (HRmax) %. Maximal oxygen uptake (VO₂max) is another method for more precise monitoring. The rate of perceived exertion (RPE) is a simpler alternative than these, as it does not require any device. The RPE correlates with heart rate, where a score of 15 on a scale of 6-20 corresponds to approximately 150 bpm, provided the individual is not on cardiovascular system-altering medications (19). Developing an individualized exercise plan that considers the most effective exercise type, intensity and duration as well as glycemic control, comorbidities, fitness levels and personal preferences is crucial for improving metabolic health and reducing disease risk (7). Psychological impediments such as time constraints in young individuals, monotony, boredom associated with the methodology of the instrument's usage, weather constraints, mood instability and sedentary lifestyle, which contribute to reasons towards inactivity, need to be acknowledged by the clinicians prescribing the exercise prescription.

PRE-EXERCISE HEALTH SCREENING

The risk of exercise-induced adverse events remains minimal in adults with T2DM engaging

in low-to-moderate intensity physical activity (10). However, an effective exercise program should be individualized and this should begin with a pre-exercise health screening. Exercising in people with diabetes could be associated with certain complications and hence, a pre-exercise evaluation is highly recommended. The extent of this evaluation would be individualized based on the clinical profile. Cardiac events such as acute myocardial infarction, arrhythmia and sudden cardiac death can occur during exercise in diabetics and prediabetics. Currently, exercise stress testing is primarily recommended in people with diabetes with a previous sedentary lifestyle who wish to engage in more intense exercise than brisk walking (9). The focus is efficiently targeting high-risk individuals prone to cardiovascular events (9,26). Below are the guidelines for the ECG stress testing in people with diabetes.

Table 1

The American College of Sports Medicine Guidelines on ECG Stress Testing on Individuals Matching One or More Than One of These Criteria (9)

1. Age >40 years, with or without cardiovascular disease risk factors other than diabetes
2. Age >30 years
 - Type 1 or Type 2 diabetes of >10 years in duration
 - Hypertension
 - Cigarette smoking
 - Dyslipidemia
 - Proliferative or pre-proliferative retinopathy
 - Nephropathy including microalbuminuria
3. Any of the following, regardless of age
 - Known or suspected coronary artery disease, cerebrovascular disease and/or peripheral artery disease
 - Autonomic neuropathy
 - Advanced nephropathy with renal failure

Clinical judgement is essential in determining the need for pre-exercise stress testing in individuals wishing to participate in a low-intensity exercise like walking (9). Although an exercise stress test can be conducted on a low-risk individual with coronary artery disease (CAD) who wishes to engage in less intense exercise, a chance of false positive tests can exist with low CAD risk (9). Patients with long-term complications of diabetes need thorough pre-exercise testing and regular assessments during a progressive exercise program. Individuals with proliferative diabetic retinopathy should avoid activities that markedly increase blood pressure or involve the Valsalva manoeuvre such as heavy weightlifting due to the risk of vitreous hemorrhage and retinal detachment. Instead, non-jarring activities such as walking, swimming or cycling are preferable, with mandatory ophthalmologic assessments every 1-2 months (26). Individuals with autonomic neuropathy require a comprehensive cardiovascular evaluation due to its strong association with elevated cardiovascular risk (27).

Similarly, individuals with diabetic nephropathy should undergo pre-exercise screening, as risk factors for nephropathy closely align with those for CAD. Although exercise may cause transient microalbuminuria due to increased blood pressure, no evidence suggests it accelerates nephropathy progression (7). Diabetic peripheral neuropathy predisposes individuals to foot ulcers and Charcot foot morphology. As sensory impairment in the hands and feet reduces awareness of injuries, weight-bearing exercises such as running or walking should be approached with caution. Protective footwear, routine foot inspections and engagement in low-impact activities, including swimming and stationary cycling, are strongly recommended (26,28). Despite these concerns, physically active individuals with neuropathy do not appear to have an elevated risk of ulceration (32).

According to the ADA guidelines, achieving adequate glycemic control is essential before initiating physical activity (10). The occurrence of hypoglycemia in individuals with T2DM is

relatively rare. However, it is more prevalent among poorly controlled patients, particularly in older adults with a prolonged disease history and those on multiple oral hypoglycemic agents or long-acting insulin therapy (27,28). Conversely, in individuals with T1DM of any age and those with T2DM requiring insulin therapy, hypoglycemia is the most commonly observed exercise-induced complication (10,29). Additionally, these individuals may experience acute hyperglycemia during high-intensity, short-duration exercise and in some cases, ketosis following strenuous activity.

Hypertension is a common comorbidity in T2DM, necessitating careful management. While exercise is beneficial in lowering blood pressure, individuals with poorly controlled hypertension should avoid high-intensity resistance training and exercises that incorporate the Valsalva manoeuvre (7). PVD is prevalent in those with diabetes, particularly in patients with concurrent hypertension, obesity or a history of smoking. Exercise remains a cornerstone of claudication management (30), though high-impact activities should be avoided. In severe cases where walking is limited, resistance training offers a viable alternative to promote functional capacity and cardiovascular health (30).

PRACTICAL CONSIDERATIONS FOR INDIVIDUALIZING EXERCISE PRESCRIPTION IN T2DM AND T1DM

Exercise Prescription for T2DM As Per ACSM Guidelines (19)

1. Aerobic Exercise for Glycemic Control

- *Frequency:* Most days of the week (≥ 3 days/week), avoiding >2 consecutive sedentary days
- *Intensity:* Moderate (55%-75% HRmax); high (75%-95% HRmax)
- *Time:* 150-300 minutes/week of moderate or 75 to 150 minutes of vigorous activity or equivalent combination thereof
- *Type:* Walking, cycling and swimming

2. *Resistance Exercise for Muscle Strength and Glucose Uptake*

- *Frequency:* 2-3 non-consecutive days/week
- *Intensity:* Load sufficient to induce muscle failure (1-3 sets per muscle group)
- *Interval:* Controlled concentric (2 seconds) and eccentric (2seconds) repetitions
- *Time:* 8-10 exercises targeting major muscle groups
- *Type:* Lifting weights and resistance bands

3. *Flexibility*

- *Frequency:* ≥ 2 -3 days/week
- *Intensity:* Stretch until slight discomfort (point of tightness) starts
- *Time:* 10-30 seconds/stretch, 2-4 repetition of each
- *Type:* e.g. static, dynamic, tai-chi and balance exercises

4. *High Intensity Interval Training (may be considered under guided supervision)*

- *Frequency:* 2-3 days/week
- *Intensity:* 75%-95% HRmax for work intervals; 30%-60% HRmax for active recovery
- *Interval:* 10seconds to 4 minutes high-intensity bouts, alternating with recovery
- *Time:* Progressively increase duration after 8 weeks; replace one continuous session initially

Exercise Considerations for T1DM

1. *Aerobic Exercise and Glycemic Monitoring:*

Monitor glucose levels before, during and after aerobic exercise to prevent hypoglycemia. Insulin levels remain elevated during exercise. Depending on the duration and intensity of the exercise, one can consume up to 80g carbohydrate / hour. Adjust insulin pump dosages 30-90

minutes pre-exercise to prevent hypoglycemia episodes.

2. *Resistance Exercise and Glucose Stability:*

Resistance training has a lower hypoglycemia risk than aerobic exercise but can cause transient glucose increases. A low-intensity cool-down may aid in muscle glucose clearance and lactate utilization, potentially preventing hyperglycemia.

3. *High Intensity Interval Training and Glycemic Management:*

The HIIT benefits fitness and glucose regulation but may cause delayed hypoglycemia, particularly at night. Close post-exercise glucose monitoring is essential, with carbohydrate intake adjustments as needed.

It is vital to note exercise should be avoided if fasting glucose levels exceed 250 mg/dL in the presence of elevated blood or urine ketones. To reduce the risk of hypoglycemia, it is advised to consume carbohydrate when blood glucose levels drop below 100 mg/dL. Furthermore, continuous blood glucose monitoring before, during and after physical activity is advised, particularly for those on insulin therapy. For extended exercise sessions, strategic food intake is necessary to prevent hypoglycemic episodes. These individualized considerations underscore the importance of tailored exercise prescriptions in diabetes management, ensuring both safety and optimal glycemic control.

MISCELLANEOUS EXERCISE PRESCRIPTION

Chrono exercise has gained popularity recently as it relates to an individual's chronotype and circadian biology, which eventually shows differences in underlying glucose metabolism (31). The effect of exercise at specific times of the day on the human body, showing diurnal oscillations, which include body temperature, glucose tolerance and circulating insulin, needs further research for a better understanding. In T2DM, insulin sensitivity is best in the evening, worse throughout sleep (nocturnal hypoglycemia

or hyperglycemia) and early morning with a disturbed circadian rhythm and hence evening physical activity/exercise may be beneficial in contrast to morning for better insulin sensitivity (32).

Concerning timing, the next question arises whether exercise should be done before or after meals. The ACSM reports that postprandial exercise effectively improves glucose regulation by mitigating acute glycemic spikes, irrespective of exercise intensity or modality, with sessions lasting ≥ 45 minutes yielding the most consistent benefits (18). Additionally, engaging in postprandial exercise has been shown to lower acute increases in serum triglyceride levels following high-fat meals and mitigate lipid-induced oxidative stress-related functional impairments (33). However, in individuals with T1DM, exercising in a fasted state, particularly in the morning, may increase blood glucose levels compared to postprandial afternoon exercise, which is associated with a higher risk of hypoglycemia (34). The underlying mechanism remains unclear but is hypothesized to result from an exercise-induced catecholamine response, including elevated cortisol and growth hormone levels (34). Notably, fasted-state morning exercise, particularly resistance training, appears to contribute to increased blood glucose levels (34), possibly due to elevated free fatty acid levels impairing glucose uptake and a heightened hormonal response involving cortisol and growth hormone compared to exercise performed in a fed state (34).

THE ROLE OF SEDENTARY BEHAVIOUR, ACTIVITY BREAKS, TIMING OF EXERCISE IN T2DM AND T1DM

Prolonged sedentary behaviour, including extended periods of sitting or lying while awake, significantly increases the risk of developing T2DM across all racial and ethnic groups (35). This risk is particularly concerning in the elderly, as individuals who accumulate more than 1 hour of sedentary time per day over 8 days experience a 22% higher likelihood of developing T2DM (19). In individuals affected by T2DM, this can

be counteracted by encouraging them to break long periods of sitting with short activity bouts. Light-intensity walking or resistance training for 3 minute every 30 minutes over eight hours has been shown to reduce postprandial glucose values (19,36). Also, light walking (2.2 hours/day) and standing (2.5 hours/day) every 30minutes/8 hours were shown to be more beneficial in enhancing 24-hour glucose regulation and insulin sensitivity in comparison to moderate-intensity cycling (19,37). However, further research is needed in this area.

DIABETES AND EXERCISE DURING PREGNANCY

Diabetes mellitus affects about 1 in 6 (17%) pregnancies in which pre-gestational diabetes is 14% and gestational diabetes mellitus (GDM) is 86.4% (38). Regular physical activity during pregnancy offers significant advantages for both the mother and fetus, aligning with the increased metabolic demands of gestation. Maternal benefits include enhanced cardiovascular function, controlled gestational weight gain, reduced musculoskeletal discomfort, lower incidence of muscle cramps and lower limb oedema, as well as improved mood stability. Additionally, exercise lowers the risk of GDM and gestational hypertension (7). Furthermore, physically active pregnancies are associated with shorter labour, reduced operative deliveries and decreased insulin requirements (7). Fetal benefits include lower fat mass, enhanced neurobehavioural development and improved stress resilience (7). According to the ACSM and the Centers for Disease Control and Prevention (ACSM-CDC), pregnant women with diabetes and no obstetric complications should engage in 60-150 minutes of aerobic exercise per week, with a daily upper limit of 30 minutes at moderate intensity aerobic exercise. Lightweight training can be added in healthy pregnant women (7,19). Physical activity can be progressively increased as pregnancy advances. Further research is required to determine optimal training modalities for women with prediabetes (7).

EXERCISE IN DIABETICS WITH MUSCULOSKELETAL CONDITION

Many people with diabetes suffer from musculoskeletal (MSK) conditions such as osteoarthritis (OA), foot ulcers and foot deformities. Whereas the pain and discomfort from these could prevent one from being physically active and exercising. This should not be a deterrent for an individual to exercise as exercise can be beneficial not only for their diabetes but also for many MSK conditions. The ACSM recommends at least 150 minutes of moderate or 75 minutes of vigorous aerobic activity weekly, in bouts of at least 10 minutes (39). Weight-bearing exercises, such as walking and resistance training, can benefit individuals with knee and hip OA, particularly when combined with weight loss (40). Non-weight-bearing exercises, including aquatic therapy, cycling and seated resistance exercises, are particularly suitable for individuals with severe OA or those who are overweight, as they maintain mobility and strength while being less discomforting to the patient. Range-of-motion activities should be incorporated to preserve joint flexibility and function. Patients with foot deformities should prioritize non-weight-bearing exercises to prevent excessive plantar pressures, while daily foot examinations are crucial for detecting ulcers or blisters early (10). In cases of foot ulcers or amputations, weight-bearing activities should be avoided until healing is complete, with a focus on seated or supine resistance exercises and cycling to reduce plantar stress (10). Although these are general recommendations, various factors such as disease severity, body weight, instability and exercise knowledge should be considered while planning exercises for this population. It is recommended that the patient consult a scientific professional such as a sports-medicine physician who understands the disease, its complications and also understands exercise physiology.

CONCLUSION

People with diabetes and cardiovascular or exercise-related complications should undergo

comprehensive pre-exercise screening, ensuring thorough evaluation by healthcare professionals. While the basic framework of 150-300 minutes/week of moderate-intensity aerobic exercise and 2-3 days/week of resistance exercise is recommended, multiple considerations must be made while considering individualized exercise prescription. An exercise prescription must be tailored to align with an individual's daily routine while minimizing potential risks. Personalized adaptation is essential for optimizing safety and effectiveness. Sedentary time reduction is a crucial addition to exercise prescription. Further research is warranted to reinforce the role of exercise as a fundamental component in diabetes management, highlighting its extensive benefits in improving overall health outcomes.

REFERENCES

1. Saeedi P, Petersohn I, Salpea P et al. Global and Regional Diabetes Prevalence Estimates For 2019 And Projections For 2030 And 2045: Results from the International Diabetes Federation Diabetes Atlas, 9th edition. *Diabetes Res Clin Pract.* 2019;157:107843.
2. Anjana RM, Unnikrishnan R, Deepa M et al. Metabolic Non-Communicable Disease Health Report of India: The ICMR-INDIAB National Cross-Sectional Study (ICMR-INDIAB-17). *Lancet Diabetes Endocrinol.* 2023;11:474-89.
3. International Diabetes Federation, IDF Diabetes Atlas. 9th Edition, Brussels.2019. Accessed on January 2025. <https://diabetesatlas.org/atlas/ninth-edition/>
4. Bodke H, Wagh V, Kakar G. Diabetes Mellitus and Prevalence of Other Comorbid Conditions: A Systematic Review. *Cureus.* 2023;15:e49374.
5. Hordern MD, Dunstan DW, Prins JB et al. Exercise Prescription for Patients with Type 2 Diabetes and Pre-Diabetes: A Position Statement from Exercise and Sport Science Australia. *J Sci Med Sport.* 2012;15:25-31.
6. Mendes R, Sousa N, Almeida A et al. Exercise Prescription for Patients with Type 2 Diabetes-A Synthesis of International Recommendations: Narrative Review. *Br J Sports Med.* 2016;50:1379-81.
7. Doupis J, Karras K, Avramidis K. The Role of Individualized Exercise Prescription in Type 2 Diabetes Mellitus Management. *touchREV Endocrinol.* 2021;17:2-4.
8. Knowler WC, Fowler SE, Hamman RF et al. 10-Year Follow-Up of Diabetes Incidence and Weight Loss in The Diabetes Prevention Program Outcomes Study. *Lancet.* 2009;374:2054. Corrected and republished from: *Lancet.* 2009;374:1677-86.
9. Colberg SR, Sigal RJ, Fernhall B et al. Exercise and Type 2 Diabetes: The American College of Sports Medicine and The

- American Diabetes Association: Joint Position Statement. *Diabetes Care*. 2010;33:e147-67.
10. Colberg SR, Sigal RJ, Yardley JE et al. Physical Activity/Exercise and Diabetes: A Position Statement of the American Diabetes Association. *Diabetes Care*. 2016;39:2065-79.
 11. Magkos F, Tsekouras Y, Kavouras SA et al. Improved insulin sensitivity after a single bout of exercise is curvilinearly related to exercise energy expenditure. *Clin Sci*. 2008;114:59-64.
 12. Wang X, Patterson BW, Smith GI et al. A ~60-Min Brisk Walk Increases Insulin-Stimulated Glucose Disposal but Has No Effect on Hepatic and Adipose Tissue Insulin Sensitivity in Older Women. *J Appl Physiol* (1985). 2013;114:1563-8.
 13. Wojtaszewski JF, Nielsen JN, Richter EA. Invited Review: Effect of Acute Exercise on Insulin Signaling and Action in Humans. *J Appl Physiol* (1985). 2002;93:384-92.
 14. Gillen JB, Little JP, Punthakee Z et al. Acute High-Intensity Interval Exercise Reduces the Postprandial Glucose Response and Prevalence of Hyperglycemia in Patients with Type 2 Diabetes. *Diabetes Obes Metab*. 2012;14:575-7.
 15. Manders RJ, Van Dijk JW, van Loon LJ. Low-Intensity Exercise Reduces the Prevalence of Hyperglycemia in Type 2 Diabetes. *Med Sci Sports Exerc*. 2010;42:219-25.
 16. Roberts CK, Hevener AL, Barnard RJ. Metabolic Syndrome and Insulin Resistance: Underlying Causes and Modification by Exercise Training. *Compr Physiol*. 2013;3:1-58.
 17. Olsen RH, Krogh-Madsen R, Thomsen C et al. Metabolic Responses to Reduced Daily Steps in Healthy Nonexercising Men. *JAMA*. 2008;299:1261-63.
 18. Bacchi E, Negri C, Targher G et al. Both Resistance Training and Aerobic Training Reduce Hepatic Fat Content in Type 2 Diabetic Subjects with Non-alcoholic Fatty Liver Disease (The RAED2 Randomized Trial). *Hepatology*. 2013;58:1287-95.
 19. Kanaley JA, Colberg SR, Corcoran MH et al. Exercise/Physical Activity in Individuals with Type 2 Diabetes: A Consensus Statement from the American College of Sports Medicine. *Med Sci Sports Exerc*. 2022;54:353-68.
 20. Sigal RJ, Armstrong MJ, Bacon SL et al. Physical Activity and Diabetes. *Can J Diabetes*. 2018;42 Suppl 1:S54-63.
 21. Canadian Diabetes Association Clinical Practice Guidelines Expert Committee. Canadian Diabetes Association 2008 Clinical Practice Guidelines for The Prevention and Management of Diabetes in Canada. *Can J Diabetes*. 2008;32:S1-201.
 22. Bloomgarden Z. T. American Diabetes Association Annual Meeting, 1999: Diabetes And Obesity. *Diabetes Care*. 2000;23:118-24.
 23. Linke SE, Gallo LC, Norman GJ. Attrition and Adherence Rates of Sustained Vs. Intermittent Exercise Interventions. *Ann Behav Med*. 2011;42:197-209.
 24. Irvine C, Taylor NF. Progressive Resistance Exercise Improves Glycaemic Control In People With Type 2 Diabetes Mellitus: A Systematic Review. *Aust J Physiother*. 2009;55:237-46.
 25. Al-Mhanna SB, Franklin BA, Jakicic JM et al. Impact of Resistance Training on Cardiometabolic Health-Related Indices in Patients with Type 2 Diabetes and Overweight/Obesity: A Systematic Review and Meta-Analysis of Randomised Controlled Trials. *Br J Sports Med*. January 7, 2025 [Online ahead of print]
 26. Pelliccia A, Sharma S, Gati S et al. 2020 ESC Guidelines on Sports Cardiology and Exercise in Patients with Cardiovascular Disease. *Eur Heart J*. 2021;42:548-49. Corrected and republished from: *Eur Heart J*. 2021;42:17-96.
 27. Entezari M, Hashemi D, Taheriazam A et al. AMPK Signaling in Diabetes Mellitus, Insulin Resistance and Diabetic Complications: A Pre-Clinical and Clinical Investigation. *Biomed Pharmacother*. 2022;146:112563.
 28. Streckmann F, Balke M, Cavaletti G et al. Exercise and Neuropathy: Systematic Review with Meta-Analysis. *Sports Med*. 2022;52:1043-65.
 29. Bremer JP, Jauch-Chara K, Hallschmid M et al. Hypoglycemia Unawareness in Older Compared with Middle-Aged Patients with Type 2 Diabetes. *Diabetes Care*. 2009;32:1513-17.
 30. Armen J, Smith BW. Exercise Considerations in Coronary Artery Disease, Peripheral Vascular Disease and Diabetes Mellitus. *Clin Sports Med*. 2003;22:123-33.
 31. Remchak ME, Heiston EM, Ballantyne A et al. Insulin Sensitivity and Metabolic Flexibility Parallel Plasma TCA Levels in Early Chronotype with Metabolic Syndrome. *J Clin Endocrinol Metab*. 2022;107:e3487-96.
 32. Heden TD, Kanaley JA. Syncing Exercise With Meals and Circadian Clocks. *Exerc Sport Sci Rev*. 2019;47:22-8.
 33. Katsanos CS, Moffatt RJ. Acute Effects of Premeal Versus Post Meal Exercise on Postprandial Hypertriglyceridemia. *Clin J Sport Med* 2004;14:33-9.
 34. Fitzpatrick R, Davison G, Wilson JJ et al. Exercise, Type 1 Diabetes Mellitus and Blood Glucose: The Implications of Exercise Timing. *Front Endocrinol*. 2022;13:1021800.
 35. Larsen BA, Martin L, Strong DR. Sedentary Behavior and Prevalent Diabetes in Non-Latino Whites, Non-Latino Blacks and Latinos: Findings from The National Health Interview Survey. *J Public Health*. 2015;37:634-40.
 36. Dempsey PC, Larsen RN, Sethi P et al. Benefits for Type 2 Diabetes of Interrupting Prolonged Sitting with Brief Bouts of Light Walking or Simple Resistance Activities. *Diabetes Care*. 2016;39:964-72.
 37. Duvivier BM, Schaper NC, Hesselink MK et al. Breaking Sitting with Light Activities Vs Structured Exercise: A Randomised Crossover Study Demonstrating Benefits for Glycaemic Control and Insulin Sensitivity in Type 2 Diabetes. *Diabetologia*. 2017;60:490-8.
 38. Egan AM, Dunne FP. Epidemiology of gestational and pregestational diabetes mellitus. In: Lapolla A, Metzger BE, eds. *Frontiers in Diabetes*. Basel, Switzerland: S. Karger AG, 2020.
 39. Garber CE, Blissmer B, Deschenes MR et al. American College of Sports Medicine position stand. Quantity and Quality of Exercise for Developing and Maintaining Cardiorespiratory, Musculoskeletal and Neuromotor Fitness in Apparently Healthy Adults: Guidance for Prescribing Exercise. *Med Sci Sports Exerc*. 2011;43:1334-59.
 40. Skou ST, Pedersen BK, Abbott JH et al. Physical Activity and Exercise Therapy Benefit More Than Just Symptoms and Impairments in People with Hip and Knee Osteoarthritis. *J Orthop Sports Phys Ther*. 2018;48:439-47.

NUTRITIONAL STRATEGIES TO REDUCE POST-PRANDIAL HYPERGLYCEMIA

Priyangee Lahiry*

INTRODUCTION

The post-prandial (PP) state refers to the “period following a meal” during which ingested carbohydrate is sequentially hydrolyzed into monosaccharides, mainly glucose. A small fraction (15-25%) of this glucose is taken up by the splanchnic tissues and the remaining enters directly into the systemic circulation, leading to a transient increase in blood glucose levels (1). In response to the rising blood glucose levels, insulin is released by the pancreas, facilitating glucose uptake by cells and promoting glycogen storage in the liver. Simultaneously, glucagon levels drop, reducing glucose production by the liver. Additionally, gut hormones such as GLP-1, GIP and neuroendocrine hormone amylin enhance insulin secretion, suppress glucagon and slow gastric emptying, which collectively help blood glucose return to pre-meal levels within 2-3 hours (2,3).

SIGNIFICANCE OF MAINTENANCE OF POST-PRANDIAL GLUCOSE LEVELS

The PP period represents a critical window during which blood glucose levels rise in response to food intake. The PP state lasts around 4 hours for glucose, followed by the post-absorptive state, lasting 6-8 hours, during which the breakdown of hepatic glycogen stores maintains blood glucose. The fasted state typically represents 10 hours without food intake. Interestingly, with the typical three meal (breakfast, lunch and dinner) dietary pattern, an individual remains in the PP state for 50% of the time in a day (12 hours) (4). Since a substantial portion of the day is spent in the PP state, proper and timely disposal of glucose from the bloodstream is essential because prolonged exposure to high glucose concentrations would have serious pathophysiological effects (5).

Impaired glucose tolerance (IGT) is characterized by PP hyperglycemia (PPHG), where there is an exaggerated rise in blood glucose after a meal. This is generally one of the first signs of insulin resistance and a major risk factor for Type 2 diabetes mellitus (T2DM) (6). Prevention of post-meal glycemic excursions is an important aspect of the prevention of T2DM and its management. It is well established that HbA1c - the gold standard measure for glycemic control is a combination of both fasting and PP glucose (PPG). As one approaches the HbA1c targets, PPG predominantly influences HbA1c levels. Therefore, maintaining PPG within the desired range is extremely important in achieving optimal glycemic control (7).

Apart from this, PPHG is an independent predictor of cardiovascular risk and mortality as well as all-cause mortality. A significant increase in carotid intima thickness has been observed with increased PPG levels. The PPHG is also associated with microvascular complications and it has been observed that a reduction in PPG has led to reduced incidences of nephropathy and retinopathy. Treating PPHG can improve oxidative stress, inflammation, endothelial dysfunction and thrombosis (7).

FACTORS DETERMINING POST-PRANDIAL GLUCOSE LEVELS

The blood glucose level at any given time is determined by the equilibrium between the rate at which glucose enters the bloodstream and the rate at which it is utilized and cleared from the bloodstream (3). Understanding the factors that influence the influx and disposal of blood glucose is key to developing effective strategies for glycemic management.

* Consultant Clinical Dietitian and Diabetes Educator, N.G Medicare & Calcutta Hope Infertility Clinic, Kolkata, India.
Email ID: priyangee.lahiry@gmail.com

Factors Affecting Post-Prandial Blood Glucose***Amount of Food and Composition of Meals:***

In normal individuals, almost 90% of the blood glucose pool is exogenous, i.e., derived from food. Hence, the amount of food and the composition of meals is a major determining factor (8). Evidently, this is one of the primary factors that can be controlled and modified to attenuate PP glyceemic response.

Rate of Gastric Emptying: The rate at which food particles are propelled into the duodenum across the pylorus, after being initially retained in the stomach, is another important factor that determines PPG levels. The initial rise in PPG levels, i.e., 30-60 minutes following a meal, is directly related to the speed of gastric emptying in healthy individuals. In people with IGT and T2DM, the effect is observed beyond 60 minutes (9,10). In both healthy individuals and those with T2DM, even slight differences in gastric emptying rates can significantly affect blood glucose levels after meals (9). Thus, dietary strategies aimed at slowing the rate of gastric emptying would help reduce PP glucose excursions.

Pace of Digestion and Absorption: Since carbohydrate has the most direct impact on blood glucose levels, the rate at which they are broken down and released into the bloodstream is another major factor affecting blood glucose levels post-meal. The quality of carbohydrate eaten and the composition of the meal therefore affects the pace of digestion and absorption and PP glyceemic response (3).

Factors Determining Glucose Disposal

Rate of Prandial Insulin Secretion: The first phase-insulin secretion, which begins within two minutes of food ingestion and continues for about 15-20 minutes, helps in the utilization of ingested glucose load by peripheral tissues and suppression of hepatic glucose output. This is followed by the second phase of insulin secretion until glucose levels return to normal. The first phase, insulin secretion, is an important determinant of post-meal glucose response and is

decreased in individuals with impaired glucose tolerance and T2DM (3).

Insulin Sensitivity in Peripheral Tissues:

Insulin sensitivity in peripheral tissues, such as skeletal muscle and adipose tissue, is a key determinant of PP glucose levels. When sensitivity is high, glucose is efficiently utilized by these tissues, resulting in lower PPG levels. Conversely, reduced insulin sensitivity impairs glucose uptake, leading to higher PPG levels. This process is influenced by factors such as the hepatic parasympathetic system, which enhances insulin sensitivity in the PP state (5).

Secretion and Effect of Incretin Hormones:

50-70 % of prandial insulin secretion occurs under the effect of incretin hormones GLP-1 and GIP. This phenomenon, called gastrointestinal-mediated glucose disposal (GIGD) is markedly reduced in individuals with T2DM (3).

MANAGING POST-PRANDIAL HYPERGLYCEMIA

The PP state plays a crucial role in overall metabolic health and managing PPHG is essential for preventing long-term complications associated with dysregulated blood sugar levels. Implementing targeted nutritional strategies can significantly mitigate PPHG, promoting better glyceemic control and reducing the risk of developing metabolic disorders.

Type and Amount of Dietary Carbohydrate

The speed and extent to which a carbohydrate-rich food can increase the blood glucose level following its ingestion is an important determinant of the PP glyceemic response. Based on this, foods that contain 80% or more of energy content from carbohydrate are assigned a ranking from 0-100 which is known as the glyceemic index (GI) (11). This concept was first introduced by Jenkins et al. and is defined as the “Incremental area under the blood glucose response curve of a specific portion (50g) of a test food, expressed as a percent of the response to the same amount of carbohydrate from a reference food (glucose) taken by the same

subject” (12). Thus, a meal containing high GI food, e.g., white rice or refined wheat flour products, will elicit a higher glycemic response compared to an isocaloric meal containing whole grains and vegetables. Therefore, choosing unrefined, complex carbohydrate sources over refined carbohydrate and avoiding of simple sugars is one of the basic strategies for attenuation of PPG levels.

However, not just the type, but also the total amount of carbohydrate ingested in a meal is an equally important determinant of the glycemic response to that meal. Thus, portioning carbohydrate appropriately is essential. Glycemic load takes into account the amount of available carbohydrate in a specific food portion along with the glycemic index. Also, keeping the amount of carbohydrate in each meal consistent on a day-to-day basis is important to avoid variability in glycemic responses to the meals (13).

Role of Dietary Fibre

The role of dietary fibre in blood sugar regulation is well established. The effects of dietary fibre vary based on their type and they act through multiple mechanisms. Dietary fibre delays digestion and absorption of starch by several mechanisms, such as delaying gastric emptying, forming starch-fibre complex, non-competitive inhibition of enzymes, reducing water availability for starch hydrolysis, reducing the diffusion of starch hydrolysis products and sugar to the intestinal mucosa as well as their transport through the mucosal membrane by downregulation of glucose transporters. Dietary fibre is also known to ameliorate the secretion of GLP-1 and other gut hormones. Incorporation of dietary fibre into a meal also helps in reducing its energy density and promotes satiety. Additionally, prebiotic fibre by colonic fermentation and the production of short-chain fatty acids can influence hepatic insulin sensitivity (14).

Modification of Macronutrient Composition

Of the macronutrients, carbohydrate evidently has the largest and most direct impact on blood

glucose levels. Thus, modifying the portion and type of carbohydrate in a meal is important as discussed in the previous section. Both protein and fat are known to decrease gastric emptying rate. Protein and fat are also known to stimulate the release of GIP and GLP-1 and contribute to the incretin effect (15). Thus, adding protein and fat to a meal rich in carbohydrate is an important and easily implementable strategy for reducing the PP glycemic response. This is particularly important in the context of a typical Indian diet primarily starch-based and deficient in protein. Reducing the carbohydrate energy percentage and replacing it with protein and/ or unsaturated fat can lead to a reduction in PP glycemic response. For example, decreasing the carbohydrate percentage from 55% to 40% and increasing the protein percentage from 15% to 30%, keeping the fat percentage constant (30%) led to a reduction in PPG by 40% (16). Replacing refined carbohydrate and sugars with low GI and fibre-rich foods such as whole grains, vegetables, legumes along with unsaturated fat, particularly MUFA is one of the established evidence-based strategies mentioned in most standard guidelines for diabetes management.

The Second Meal Effect

The ability of a prior meal to influence the glycemic response of the subsequent meal is known as the “second meal effect”. Consuming low GI foods such as whole grains and legumes at one meal can lower the PP glucose levels not only after that meal but also after the subsequent meal. This can be primarily explained by the “Staub Traugott” effect, in which there is potentiation of insulin release upon prior exposure to glucose (17). Thus, the first meal increases the beta cell responsiveness to the second meal (18). The reduced PP glycemia improves insulin sensitivity and action by lowering circulating free fatty acids and also by reducing cytokine production thereby reducing oxidative stress, which is known to impair insulin signaling. Also, colonic fermentation of indigestible carbohydrates leads to the production of short-chain fatty acids which can delay gastric emptying and improve insulin sensitivity (18).

However, it is important to note that extensive milling and cooking at high temperatures can not only counteract the second meal effect but can also increase glycemia after the subsequent meal. Therefore, consumption of legumes and whole grains in the minimally processed form is important (19).

Nutrient Preloading

Nutrient preloading involves ingesting a small portion of a nutrient, typically 30-60 minutes before a meal. This is emerging as an effective intervention for attenuation of PPHG, as evidenced by various laboratory experiments and clinical trials. In this context, the most commonly studied nutrients are protein and fat. A recent body of evidence has suggested that fibre and fructose ingestion before carbohydrate-rich meals can also have a significant post-meal glucose-lowering impact.

While the PPG lowering effect of preloads can be attributed to several mechanisms depending on the type of nutrient, the principal mechanism revolves around slowing of gastric emptying, stimulation of incretin and other gut hormones and consequent effect on satiety. In a randomized crossover study of T2DM subjects, gastric emptying of a mashed potato meal was measured on three occasions: when water was ingested before the meal, when oil was ingested before the same meal and when water was ingested before a mashed potato meal that contained oil. The most pronounced reduction in glycemic response was observed when oil was consumed before the meal (20). As suggested by this study, the effect of gastric emptying of fat is more prominent when consumed separately compared to when mixed with other nutrients. It has been noted that soy or whey-based preloads have a more significant impact on PP glycemia than fat (21). It is important to note that while pre-meal nutrient loading appears to be an easy and promising strategy, it involves the consumption of additional calories, which needs to be adjusted and accounted for.

Meal Sequencing

Meal sequencing is the strategic arrangement of the order in which various food groups are eaten during a meal that favourably affects the post-meal glycemic response. This typically involves starting the meal with fibre, e.g., vegetable salad, followed by protein, healthy fat and finishing the meal with carbohydrate. The effect of meal sequencing is well documented in several studies. For example, it has been noted that consumption of meat/fish or vegetables before carbohydrate intake can reduce PPG spike by 50% and delay it by 30-60 minutes (22,23). This method works by the same principle as pre-meal nutrient loading, i.e., enhancement of GLP-1 secretion from the gut, optimizing insulin and glucagon responses and reducing gastric emptying (24). This approach is now established as a simple and effective strategy for the management of PPHG and has become a part of standard guidelines for the management of diabetes. An advantage of this approach is that it does not involve additional calorie intake, unlike pre-meal nutrient loading (21).

Role of Functional Foods and Bioactive Compounds

Polyphenols found in various plant-based foods have been shown to reduce PP glycemia by reducing digestion and absorption of carbohydrate. Polyphenols from green tea, coffee, cocoa, cinnamon and extracts of various fruits such as black currant, blueberry, apple, pomegranate and red grapes have been studied in various in vitro and animal experiments and the various compounds that have been shown to have such effects are anthocyanins, hesperidin, quercetin, isoquercitrin and saponin type homoisoflavonoids (3, 25).

The glucose-lowering effect of polyphenols can be attributed to their ability to inhibit pancreatic alpha-amylase and intestinal alpha-glucosidase enzymes and modulate intestinal glucose transporters. Additionally, they might be able to stimulate insulin secretion and suppress hepatic glucose output (25). The inhibitory effect on carbohydrate digestion and glucose absorption

has been observed to be superior to acarbose. However, data from clinical studies are limited and inconsistent and further research is needed to establish their role in blood glucose management.

Vinegar is another substance, often hyped for its potential glucose-lowering effects. Limited research evidence that is available to date has shown that vinegar taken before a carbohydrate-rich meal can lower PPG levels by 20-30% (26). A 10-30 ml dose has been used in most studies (26). However, it has been seen to be more effective with high GI carbohydrate rather than low GI ones. Though the exact mechanism of action is not yet identified, acetic acid in vinegar has been shown to inhibit the activity of sucrase and other disaccharidases slowing down gastric emptying. It might also stimulate insulin secretion and promote glucose uptake by skeletal muscles. Chlorogenic acid in apple cider vinegar has been found to inhibit glucose-6-phosphatase enzyme, thereby decreasing hepatic gluconeogenesis (26, 27). Although vinegar shows potential as a glucose-lowering agent, larger-scale clinical trials across diverse settings are necessary to confirm its effectiveness and long-term safety.

CONCLUSION

When implementing nutritional strategies to manage PPHG, a comprehensive and individualized approach that combines multiple strategies is essential. Emphasizing the concept of a mixed meal using the plate method helps patients better understand portion distribution among different food groups. A well-balanced mixed meal ensures adequate protein intake, sufficient fibre and healthy fat, which not only aid in glycemic control but also contribute to overall nutritional adequacy. Practical dietary modifications such as dividing large meals into smaller portions, replacing refined cereals with whole grains and incorporating pulses and legumes can significantly enhance carbohydrate quality. Patients should also be educated to recognize starchy vegetables such as potato, sweet potato and yam as main carbohydrate

servings rather than treating them as side dish alongside other staple carbohydrate foods such as rice, roti or bread. Functional foods containing bioactive compounds, such as cinnamon and vinegar, may serve as adjuncts in reducing PP glycemia. However, they should complement, rather than replace, a well-balanced diet and regular physical activity. A tailored, holistic approach to meal planning remains the most effective strategy for mitigating PPG spikes and improving overall glycemic control.

REFERENCES

1. Schrauwen-Hinderling VB, Carpentier AC. Molecular Imaging of Postprandial Metabolism. *J Appl Physiol*. 2018;124:504-11.
2. Zhang XX, Pan YH, Huang YM, Zhao HL. Neuroendocrine Hormone Amylin in Diabetes. *World J Diabetes*. 2016;7:189-97.
3. Pasmans K, Meex RCR, van Loon LJC et al. Nutritional Strategies to Attenuate Postprandial Glycemic Response. *Obes Rev*. 2022;23:e13486.
4. Monnier L. Is Postprandial Glucose a Neglected Cardiovascular Risk Factor in Type 2 Diabetes? *Eur J Clin Invest*. 2000;30:3-11.
5. Macedo MP, Lima IS, Gaspar JM et al. Risk of Postprandial Insulin Resistance: The Liver/Vagus Rapport. *Rev Endocr Metab Disord*. 2014;15:67-77.
6. American Diabetes Association. Classification And Diagnosis of Diabetes. *Diabetes Care*. 2016;39:S13-22.
7. Singh SK. Post-Prandial Hyperglycaemia. *Indian J Endocrinol Metab*. 2012;16:S245-7.
8. Bruce CR, Hamley S, Ang T et al. Translating Glucose Tolerance Data from Mice to Humans: Insights from Stable Isotope Labelled Glucose Tolerance Tests. *Mol Metab*. 2021;53:101281.
9. Marathe CS, Rayner CK, Jones KL et al. Relationships Between Gastric Emptying, Postprandial Glycemia and Incretin Hormones. *Diabetes Care*. 2013;36:1396-405.
10. Marathe CS, Horowitz M, Trahair LG et al. Relationships of Early and Late Glycemic Responses with Gastric Emptying During an Oral Glucose Tolerance Test. *J Clin Endocrinol Metab*. 2015;100:3565-71.
11. Brouns F, Bjorck I, Frayn KN et al. Glycaemic Index Methodology. *Nutr Res Rev*. 2005;18:145-71.
12. Jenkins DJA, Wolever TMS, Taylor RH et al. Glycemic Index of Foods: A Physiological basis for Carbohydrate Exchange. *Am J Clin Nutr*. 1981;34:362-6.
13. O'Keefe JH, Gheewala NM, O'Keefe JO. Dietary Strategies

- for Improving Post-Prandial Glucose, Lipids, Inflammation and Cardiovascular Health. *J Am Coll Cardiol*. 2008;51:249-55.
14. Goff HD, Repin N, Fabek H et al. Dietary Fibre for Glycaemia Control: Towards A Mechanistic Understanding. *Bioact Carbohydr Diet Fibre*. 2018;14:39-53.
15. Carrel G, Egli L, Tran C et al. Contributions of Fat and Protein to The Incretin Effect of a Mixed Meal. *Am J Clin Nutr*. 2011;94:997-1003.
16. Gannon MC, Nuttall FQ, Saeed A et al. An Increase in Dietary Protein Improves the Blood Glucose Response in Persons with Type 2 Diabetes. *Am J Clin Nutr*. 2003;78:734-41.
17. Bonuccelli S, Muscelli E, Gastaldelli A et al. Improved Tolerance to Sequential Glucose Loading (Staub-Traugott Effect): Size and Mechanisms. *Am J Physiol Endocrinol Metab*. 2009;297:E532-7.
18. Jovanovic A, Gerrard J, Taylor R. The Second-Meal Phenomenon in Type 2 Diabetes. *Diabetes Care*. 2009;32:1199-201.
19. Higgins JA. Whole grains, Legumes and the Subsequent Meal Effect: Implications for Blood Glucose Control and The Role of Fermentation. *J Nutr Metab*. 2012;2012:829238.
20. Gentilcore D, Chaikomin R, Jones KL et al. Effects of Fat on Gastric Emptying of and the Glycemic, Insulin and Incretin Responses to a Carbohydrate Meal in Type 2 Diabetes. *J Clin Endocrinol Metab*. 2006;91:2062-7.
21. Kamruzzaman M, Horowitz M, Jones KL et al. Gut-Based Strategies to Reduce Postprandial Glycaemia in Type 2 Diabetes. *Front Endocrinol*. 2021;12:661877.
22. Kuwata H, Iwasaki M, Shimizu S et al. Meal Sequence and Glucose Excursion, Gastric Emptying and Incretin Secretion in Type 2 Diabetes: A Randomised Controlled Crossover Exploratory Trial. *Diabetologia*. 2016;59:453-61.
23. Nishino K, Sakurai M, Takeshita Y et al. Consuming Carbohydrates After Meat or Vegetables Lowers Postprandial Excursions of Glucose and Insulin in Nondiabetic Subjects. *J Nutr Sci Vitaminol*. 2018;64:316-20.
24. Kubota S, Liu Y, Iizuka K et al. A Review of Recent Findings on Meal Sequence: An Attractive Dietary Approach to Prevention and Management of Type 2 Diabetes. *Nutrients*. 2020;12:2502.
25. Kim Y, Keogh JB, Clifton PM. Polyphenols and Glycemic Control. *Nutrients*. 2016;8:17.
26. Santos HO, de Moraes WMAM, da Silva GAR et al. Vinegar (Acetic Acid) Intake on Glucose Metabolism: A Narrative Review. *Clin Nutr ESPEN*. 2019;32:1-7.
27. Siddiqui FJ, Assam PN, de Souza NN et al. Diabetes control: Is Vinegar a Promising Candidate to Help Achieve Targets? *J Evid Based Integr Med*. 2018;23:2156587217753004.

QUESTION AND ANSWER

Q. What is the impact of non-nutritive sweeteners on glycemic control and gut health?

- A.** Non-nutritive sweeteners (NNSs) are also known as high-intensity sweeteners, as they are required in minute quantities to get the desired sweetness with low or no calorie contribution to the diet. This characteristic makes them as a valuable tool for managing caloric intake, supporting weight loss efforts and aiding in glycemic control. However, the evidence of their effectiveness remains mixed. The NNSs are Saccharin, Aspartame, Acesulfame K, Sucralose and Steviol Glycoside to name a few.

Impact on Glycemic Control: Non-nutritive sweeteners can help individuals with diabetes regulate their blood glucose levels indirectly. By switching from soda to diet soda, one can satisfy their cravings without experiencing spikes in blood glucose levels. Metabolically, the impact of its delivery in the form of the aqueous solution is similar to the one caused by water. A meta-analysis conducted in 2018 by Nichol AD et al. found that consumption of NNSs did not raise blood glucose levels compared to baseline. Although sugar substitutes provide no energy, they indirectly affect carbohydrate metabolism by impacting the digestive system. This effect of NNSs can be measured in long-term exposure compared to short-term exposure with no changes in the glucose level in the blood. Other research indicates that the consumption of NNSs may impair metabolic responses. Furthermore, as per WHO guidelines 2023, habitual consumption of these sweeteners may not necessarily translate to improved glycemic outcomes, as it could encourage higher overall energy intake through compensatory eating behaviours. Prospective cohort studies conducted on adults reported the intake of NNSs for a long time may lead to an increased risk of developing Type 2 diabetes mellitus. However, when short-term randomized controlled trials (RCTs) were conducted, no significant impact on

fasting glucose levels, fasting insulin levels or blood lipids was observed. Future research should explore differences in how factors such as age, gender and ethnicity influence people's responses to frequent consumption and biological mechanisms of NNSs over the long term.

Impact on Gut Health: The gut bacteria in the colon ferment dietary fibre to produce short-chain fatty acids, primarily propionate, butyrate and acetate. These metabolites act as an anti-inflammatory gut agents regulated via T-regulatory cells. Some studies have proposed that NNSs are associated with changes in gut microbiota, known as dysbiosis. This effect of NNSs resemble those caused by antibiotics. Gut dysbiosis, an imbalanced ratio of *Firmicutes* to *Bacteroidetes* has been linked to metabolic syndrome related problems like weight gain, impaired glucose tolerance, insulin resistance, high-fat intake, increased gut permeability and inflammatory bowel disease. In inflammatory disorders, adverse changes in the gut lining affect the system. The mechanism of action of NNSs is being explored. Future research is needed to understand the potential effects of these compounds better. Promoting a balanced gut microbiota similar to the effect of prebiotics could aid in restoring healthy conditions in microbiota-related pathological issues.

In summary, while NNSs may offer some benefits for glycemic control and serve as a tool for weight management, their overall impact on health, particularly gut health, remains complex.

Blasee R Fernaandes

Q: What is the connection between visceral fat and insulin resistance?

- A.** Visceral fat is closely linked to insulin resistance. Excess visceral fat can contribute to the development of insulin resistance. Connection between visceral fat and insulin resistance can be explained in the following ways:

Location of Visceral Fat: Visceral fat is the fat stored around internal organs such as the liver, pancreas and intestines. It is metabolically active and releases various substances that can affect your body's ability to process insulin.

Impact on Insulin Sensitivity: The insulin helps cells take up glucose from the bloodstream. When the body becomes resistant to insulin, more insulin is required to achieve the same effect. Over time, this leads to higher levels of insulin in the blood (hyperinsulinemia), which can further increase fat storage, especially in the abdominal area. This creates a vicious cycle that worsens insulin resistance. Thus, excess visceral fat around internal organs tend to disrupt insulin function through inflammation, fatty acid release and liver dysfunction, all of which contribute to insulin resistance.

Inflammation and Hormonal Disruption: Visceral fat releases inflammatory cytokines (proteins that promote inflammation) and adipokines (hormones produced by fat cells). These substances can interfere with insulin signaling pathways. Chronic low-grade inflammation caused by visceral fat can impair the action of insulin, making the body less responsive to it. Thus, contributing to insulin resistance.

Fatty Acids and the Liver: Visceral fat can also release free fatty acids into the bloodstream. The accumulation of fat in the liver by these fatty acids can lead to a condition known as non-alcoholic fatty liver disease (NAFLD). The liver plays a central role in regulating blood glucose levels and when it has been overloaded with fat, it may contribute to insulin resistance.

Rima Ved

RECIPES

STRAWBERRY AND OATS SHAKE



INGREDIENTS

60 gm Rolled Oats

60 gm Skim Curd

8 medium Almond

4 medium Strawberry

Non-nutritive sweetener as per taste

200 ml Water for the shake

METHOD

1. Soak the Oats overnight in water.
2. Drain the excess water from Oats in the following morning.

3. In a blender, add the soaked Oats, curd, water and strawberries. Blend it until smooth.
4. Add a few ice cubes for a refreshing texture.
5. Optionally, garnish with additional chopped strawberries and serve.

PROVIDES 2 SERVINGS

Nutritional Information Per Serving

| Energy (Kcal) | Carbohydrate (gm) | Protein (gm) | Fat (gm) |
|---------------|-------------------|--------------|----------|
| 150 | 21 | 5 | 5 |

SPECIAL FEATURES

- Protein-rich
- Low glycemic index

Jayshri Jain

FOUR BEAN SALAD

**INGREDIENTS**

50 gm French Beans
 50 gm Red Kidney Beans
 50 gm Chickpeas
 50 gm Edamame (Soyabean)
 100 gm Celery, finely chopped
 100 gm Onion, finely chopped
 50 gm Bell Peppers, finely chopped
 ½ tsp Garlic paste
 2 tsp Extra Virgin Olive Oil
 Lemon Juice to taste
 Black Pepper to taste
 Chilli Flakes to taste
 Salt to taste to taste

METHOD

1. Rinse kidney beans and chickpeas, then soak and drain them.

2. Bring a medium pot of water to a boil over high heat.
3. Add French beans, edamame, kidney beans and chickpeas, cooking until tender. Drain and rinse under cold water.
4. In a large bowl, mix the cooked beans with chopped celery, onion and bell peppers.
5. In a separate bowl, mix the remaining ingredients and toss with the bean mixture until well combined.

PROVIDES 4 SERVINGS**Nutritional Information Per Serving**

| Energy (Kcal) | Carbohydrate (gm) | Protein (gm) | Fat (gm) |
|---------------|-------------------|--------------|----------|
| 120 | 13 | 10 | 3 |

SPECIAL FEATURES

- Protein-rich
- Low-fat

Jayshri Jain

HOW KNOWLEDGEABLE ARE YOU?

1. Which of the following is not a health problem caused by diabetes?
 - a. Cardiovascular problem
 - b. Lung cancer
 - c. Kidney failure
 - d. Neuropathy
2. What is the first line of drug used upon failure of diet and exercise to achieve target blood glucose levels?
 - a. Pioglitazone 15 mg once daily
 - b. Metformin 500 mg twice or thrice daily
 - c. Dapagliflozin 10mg once daily
 - d. Glimepiride 4 mg day
3. The goals of the exercise plan/therapy include all the following, except
 - a. Increase sensitivity to anti-diabetic drugs
 - b. Control blood glucose levels
 - c. Control lipids (cholesterol) levels
 - d. Increase blood hemoglobin
4. Which person needs a medical checkup before initiating exercises?
 - a. An 18 year old Type 1 diabetic who wants to start playing squash
 - b. A 50 year old Type 2 diabetic who wants to switch over from tennis to swimming
 - c. A 50 year old Type 2 diabetic who wants to start learning tennis
 - d. A 40 year old non-diabetic who wants to start jogging
5. Which is the worst type of shift for a diabetic to work?
 - a. Daily evening shift (4 pm - 12 midnight)
 - b. Daily morning shift (8 am - 4 pm)
 - c. Daily night shift (12 midnight - 8 am)
 - d. Changing shifts every week
6. The best and the most important indication for a Type 1 diabetic to use insulin pump is
 - a. Recurrent hypoglycemia
 - b. HbA1c >9%
 - c. Proposed marriage
 - d. Weight loss
7. For carbohydrate counting, which foods are important?
 - a. Cereals & pulses
 - b. Chicken & fish
 - c. Diet cola
 - d. Vegetables
8. The immediate cause for infection of foot in a diabetic is most likely to be
 - a. Peripheral neuropathy
 - b. Peripheral vascular disease
 - c. Foot ulcer
 - d. Chewing tobacco
9. The predominant type of neuropathy in diabetes is
 - a. Sensory-motor
 - b. Motor
 - c. Autonomic
 - d. Cranial
10. Starvation state differs from diabetic ketoacidosis in as much as in starvation
 - a. Ketoacidosis is marked
 - b. Dehydration is present
 - c. Leukocytosis is present
 - d. Blood glucose is lower

ANSWERS:
1. b
2. a
3. d
4. c
5. a
6. a
7. a
8. b
9. a
10. d

MYTHS AND FACTS

Myth: People with diabetes cannot drink alcohol.

Fact: Moderate alcohol consumption can be safe but should be carefully monitored for blood glucose levels. People living with diabetes should be advised to avoid alcohol as a recommendation. Nevertheless, it will be unfair to social drinkers who have diabetes. Hence, physicians often permit moderate amounts of alcohol consumption and advise keeping track of their blood glucose levels.

One gram of alcohol yields seven calories. Ten per cent of the daily calorie intake may be found in one drink, depending on the concentration of alcohol present. An individual can opt for two small pegs (2 oz, 60 ml) of whisky, brandy, gin and rum or one and a half glass (5 oz, 150 ml) of wine or one glass (<12 oz, 350 ml) of beer per day. Dry wines are relatively safer than sweet wines, as they are unsweetened. Drinks should not be mixed with sweetened liquids such as ginger ale or cocktail mixes; rather, they should be mixed with water, ice or soda. Excessive drinking, more than three drinks per day, can raise HbA1c and blood glucose levels. Consistent consumption of alcohol over time can lead to weight gain, insulin resistance, pancreatitis and liver cirrhosis, which further complicates the management of diabetes.

The primary cause of concern for drinking alcohol in people with diabetes is hypoglycemia, as it inhibits glucose release, especially if consumed on an empty stomach. The liver breaks down alcohol first when it is consumed on an empty stomach. Hence, it is advised to eat while drinking alcohol. The choice of accompaniments while drinking should be made wisely. Firstly, if drinking alcohol is a must, it is recommended to drink slowly and sparingly. Secondly, opt for salads and avoid fried snacks, salted nuts and protein-rich snacks. When drinking alcohol, omit bread or fat exchange which is calorically equivalent to the alcohol consumed. Beer and wine contain a high amount

of carbohydrate, which significantly contributes to high calorie, making it undesirable for people on calorie-restricted diets. Furthermore, hypoglycemia occurs when alcohol is combined with insulin and oral hypoglycemic agents such as sulfonylureas. Here, the symptoms of hypoglycemia and alcohol intoxication appears similar and can be mistaken as one for the other.

Tanvi Gala

Myth: Type 1 diabetes mellitus only develops in children.

Fact: Type 1 diabetes mellitus (T1DM), though most commonly diagnosed in children, adolescents and young adults, can develop at any age, including adulthood. The condition arises when the immune system attacks and destroys the body's own insulin-producing cells in the pancreas. Adults may have Latent Autoimmune Diabetes in Adults (LADA). In LADA, there is indeed an autoimmune attack on the pancreas, but the process is more gradual, where the beta cell destruction occurs slowly over time. As a result, people with LADA may initially show symptoms similar to Type 2 diabetes mellitus (T2DM) such as non-ketosis proneness. They may require insulin therapy later on, if not initially because their pancreas slowly loses its capacity to produce sufficient insulin.

Rima Ved

Myth: Diabetes cannot be prevented if it runs in the family.

Fact: It is a myth that diabetes cannot be prevented if it runs in the family. One cannot change their genetic makeup, but can change their habits. By making informed lifestyle choices and following a healthy diet regimen, one can significantly minimize the risk of developing Type 2 diabetes mellitus (T2DM), even if it runs in the family. The American Diabetes Association states that the risk of developing T2DM increases due to poor eating habits and a sedentary lifestyle with lack

of physical activity. The good news is that it is possible to delay or prevent T2DM by encouraging healthy food choices, exercise and weight loss. Research shows that lifestyle intervention programs promoting healthy diets, physical activity and modest body weight loss can prevent or delay the onset of T2DM among high-risk populations such as those with impaired glucose tolerance (IGT). The Diabetes Prevention Program demonstrates that individuals can significantly decrease the risk and their likelihood of developing T2DM by implementing the following:

Lose Weight and Keep It Off: For every kilogram of weight lost, there is a 16% reduction in risk for progression to diabetes. It is possible to prevent or postpone the onset of T2DM by losing only 5 to 7% of the current body weight. For a safe and sustainable weight loss, a gradual rate of 0.5 to 1 kilogram per week is usually recommended. This facilitates weight-loss maintenance approach is easier to maintain on a

long-term basis and is considered safer than rapid weight loss.

Increase Mobility: According to the Centers for Disease Control and Prevention (CDC), adults should aim for a minimum of 150 minutes of moderate-intensity physical activity each week, as well as 2-3 days of muscle-strengthening activity per week. If one is not active, talking to a healthcare professional will help one initiate an effective exercise program.

Eat Healthy and Practice Portion Control: A nutritionally balanced, hypocaloric diet is recommended for weight loss. Consuming smaller meal portions can help in lowering calorie content. Choosing foods with lower fat content is another effective way to cut calories. Additionally, consuming water instead of sweetened beverages sweetened with sugar can further aid in weight management, hence, reducing the risk of developing T2DM.

Jayshri Jain

DIABETES EDUCATOR CERTIFICATE COURSE

Dr Chandalia's DENMARC in association with Help Defeat Diabetes Trust (HDDT) presents to you a Certificate course for Diabetes Educators (CDE)!

Help Defeat Diabetes Trust (HDDT) is a registered, non-profit public trust, having amongst its many objectives, the main objective of promoting education and awareness about diabetes among people from different fields.

Who can enroll?

Graduates in Nutrition, Doctors, Nursing, Pharmacy, Occupational and Physiotherapy.

What is the duration of the course?

6 months, including 3 months (300 Hours) of hands-on training and experience with a recognized mentor in your own town (see this on our website).

How can I do this course from my place of residence?

A suitable Mentor can be selected from the registrant's locality under whom the training can be done.

How will I get the course material?

All course material is available online on our website.

What are the course fees?

The standard fee for the course are INR 10,000/- only.

Where can I get more information about this course?

Kindly visit our website <http://www.helpdefeatdiabetes.org> or you can get in touch with us on our email id: heldefeatdiabetesinfo@gmail.com.



DIABETES EDUCATOR CERTIFICATE COURSE

HELP DEFEAT DIABETES TRUST announces

Reward of Rs. 10,000/- for securing the highest marks every year



Nature of Course: Virtual and Hands on

Duration: 6 months

Course Highlights:

- Get certificate of training in diabetes
- Get practical exposure under a recognized mentor in your own town
- Get access to 800 pages of study material and more than 18 audio & audiovisuals.

Criteria for award:

- To complete the course in given time frame i.e. 6 months.
- To secure highest marks in the current year.

For further details visit helpdefeatdiabetes.org

MEMBERSHIP FORM

Association of Diabetes Educators (ADE)

(For eligibility criteria: Check Website www.diabeteseducatorsindia.com)
(Kindly print, duly fill, scan and upload)



Name Age: Gender:

Address

.....

Telephone: Res: Office: Cell:

E-mail id:

Educational Qualifications:

.....

Work Experience:

.....

Currently employed at:

.....

Certificates attached:

.....

How do you wish to participate in the ADE activities?

- ☐ Update my knowledge and skills
- ☐ As a faculty in ADE's Educational Activities
- ☐ Organizational Activities as Office Bearer

Please pay the membership fees through NEFT / RTGS/online to the following bank account. The details are as follows:

Account name: Association of Diabetes Educators

Account type: Savings Account

Name of the bank: Bank of India

Account number: 006610110001734

IFSC Code: BKID0000066

.....
Signature



In Uncontrolled T2DM Patients***, Add

Cospiaq® S

25mg
+
100mg

empagliflozin + sitagliptin

EMPAWERING DUO for **Extended** Life

Also Available

Cospiaq®
empagliflozin

Cospiaq® M
empagliflozin + metformin HCl (Extended Release)

Cospiaq Met™
empagliflozin + metformin HCl (Immediate Release)

COMING SOON

Cospiaq® SM

empagliflozin + sitagliptin + metformin

Gift your T2DM Patients



***In T2DM for CV Risk Reduction. **For patients 45 years of age. \$As compared to patients on non-metformin therapies.



MARKETED BY: TORRENT PHARMACEUTICALS LTD.
Torrent House, Off Ashram Road, Ahmedabad - 380009, India



In T2DM, CVD & CKD

Empagreat[®]

Empagliflozin Range

^{Rx} Initiate Early in T2DM patients at high risk of CVD & CKD

DynaDuoTM

Empagliflozin + Linagliptin TABLETS

Dynamic Duo Control & Protection



