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Introduction

Type 2 Diabetes Mellitus (T2DM) is a chronic metabolic disorder defined by chronic hyperglycaemia due to a gradual decrease in insulin sensitivity and a decline in insulin production by the pancreatic cells, called beta cells ([American Diabetes Association, 2024](#)). T2DM is a progressive illness characterized by high interaction of genetic, physiological, and environmental risk factors, in contrast to Type 1 diabetes, which is caused by a progressive autoimmune destruction of the pancreatic beta cells resulting in absolute insulin deficiency ([WHO, 2016](#)). There is also certain non-modifiable risk factors identified, such as older age, family history and ethnic groups including South Asian, African-Caribbean and Black African populations ([NHS, 2025](#)). Tobacco smoking, high intakes of ultra-processed foods and refined carbohydrates, central adiposity, and physical inactivity are the most important modifiable risk factors responsible for T2DM prevalence globally ([Davies et al., 2022](#)).

In primary care, the practice nurse is likely to see patients who have difficulty controlling their blood glucose levels because they do not understand how the process works in their body. As long-term hyperglycaemia progresses, it can cause microvascular complications (including retinopathy, nephropathy and neuropathy) and macrovascular complications (such as coronary heart disease, peripheral arterial disease and stroke) ([NICE, 2026](#)).

This report aims to review physiological evidence and create an educational tool for use by patients at their primary care clinic. It examines the regulatory mechanisms involved in maintaining normoglycaemia, explains how ATP is generated in cells, reviews the pathophysiology of T2DM and identifies lifestyle factors, and describes the clinical value of monitoring and recording blood glucose parameters and how this can enable patients to participate in their disease management in a safe, self-managed way. It also outlines evidence-based recommendations based on national clinical guidelines to support the use of these principles in clinical practice.

Section 1: Anatomy & Physiology

Maintenance of blood glucose concentration (Homeostasis) Plasma glucose levels need to be tightly regulated in a narrow range (around 4.0— 7.8 mmol/L), and under physiological conditions, glucose is controlled mainly by the endocrine pancreas through a negative feedback loop ([Hall and Hall, 2020](#)). The two major types of cells in the islets of Langerhans

that regulate glucose homeostasis are alpha cells, which release glucagon, and beta cells, which produce insulin (Stanfield, 2017).

After eating, complex carbohydrates are broken down to simple sugars in the gastrointestinal tract and then enter the bloodstream. This causes a rise in blood sugar, which in turn triggers the pancreas beta cells to release insulin. The binding of insulin to the specific tyrosine kinase receptors on target cell membranes occurs in Skeletal Muscle and Adipose Tissue. This binding activates an intracellular signalling pathway that results in the movement of storage vesicles containing Glucose Transporter 4 from the cytoplasm to the cell membrane allowing glucose to move into cells by passive diffusion (Shulman, 2000).

In liver (Hepatocytes) insulin promotes the storage of glucose as glycogen (glycogenesis) and suppresses the release of glucose from stored glycogen (glycogenolysis) or the formation of glucose from non-carbohydrate sources (gluconeogenesis) (Hall and Hall, 2020). If, on the other hand, you skip a meal or exercise, your blood glucose drops, which stops insulin production and triggers a release of glucagon from the pancreas. Glucagon acts on hepatocytes to increase glucose production by glycogenolysis and gluconeogenesis to ensure adequate fuel supply to vital organs, especially the brain.

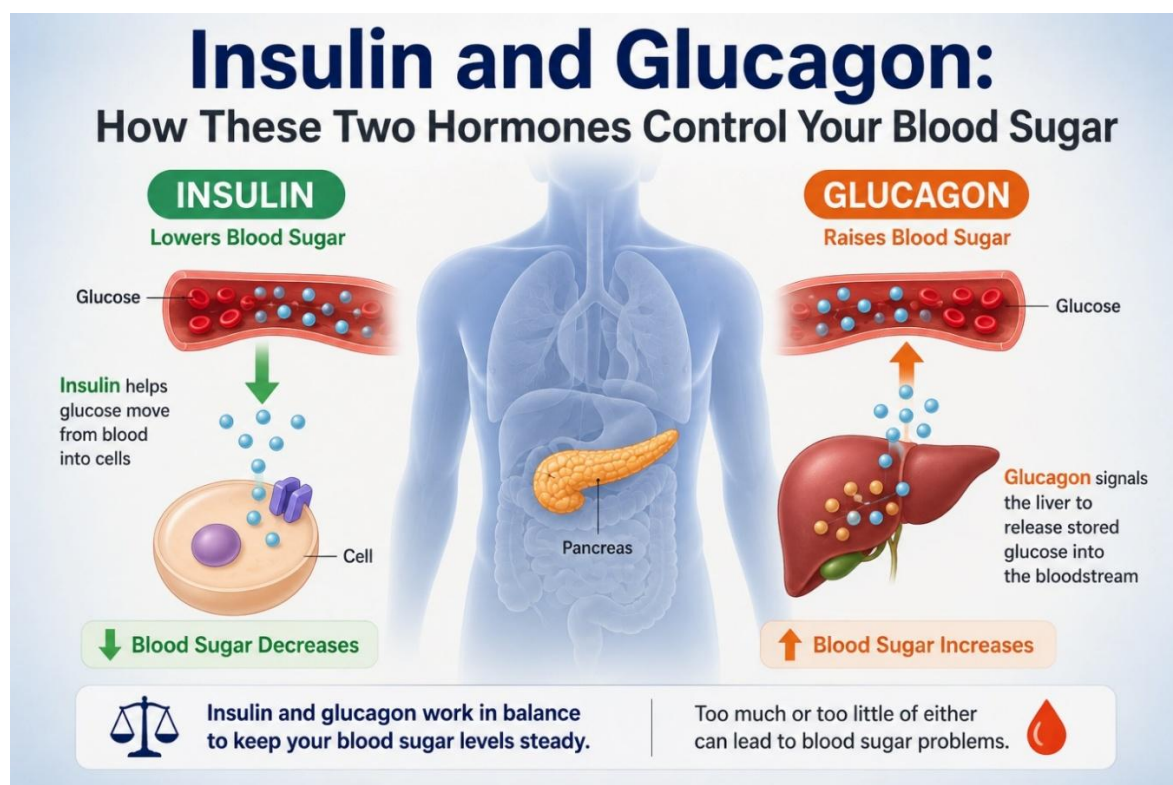
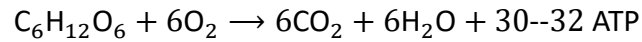


Figure 1: How blood sugar is maintained in the body (Organic Gyaan, 2026).

Glucose is converted by cellular respiration in systemic body cells to adenosine triphosphate (ATP), the chemical currency for cellular energy. In overall cellular respiration the balanced equation is:



Glucose is completely oxidized in four steps:

1. Glycolysis (Cytoplasm): Glucose (a 6-carbon molecule) is broken down into 2 Pyruvate (3-carbon molecules) producing 2 NADH and 2 ATP as net products. This initial process takes place in the absence of oxygen (anaerobic) (Stanfield, 2017).
2. Pyruvate crosses the mitochondrial membrane and is converted to Acetyl-coenzyme A (Acetyl-CoA) and releases a molecule of carbon dioxide (CO_2) as a waste product.
3. Acetyl-CoA is consumed in a cycle (Krebs cycle) of enzymes resulting in the production of 2 ATP (or GTP), high energy electron carrier molecules (NADH and FADH_2) and some CO_2 .

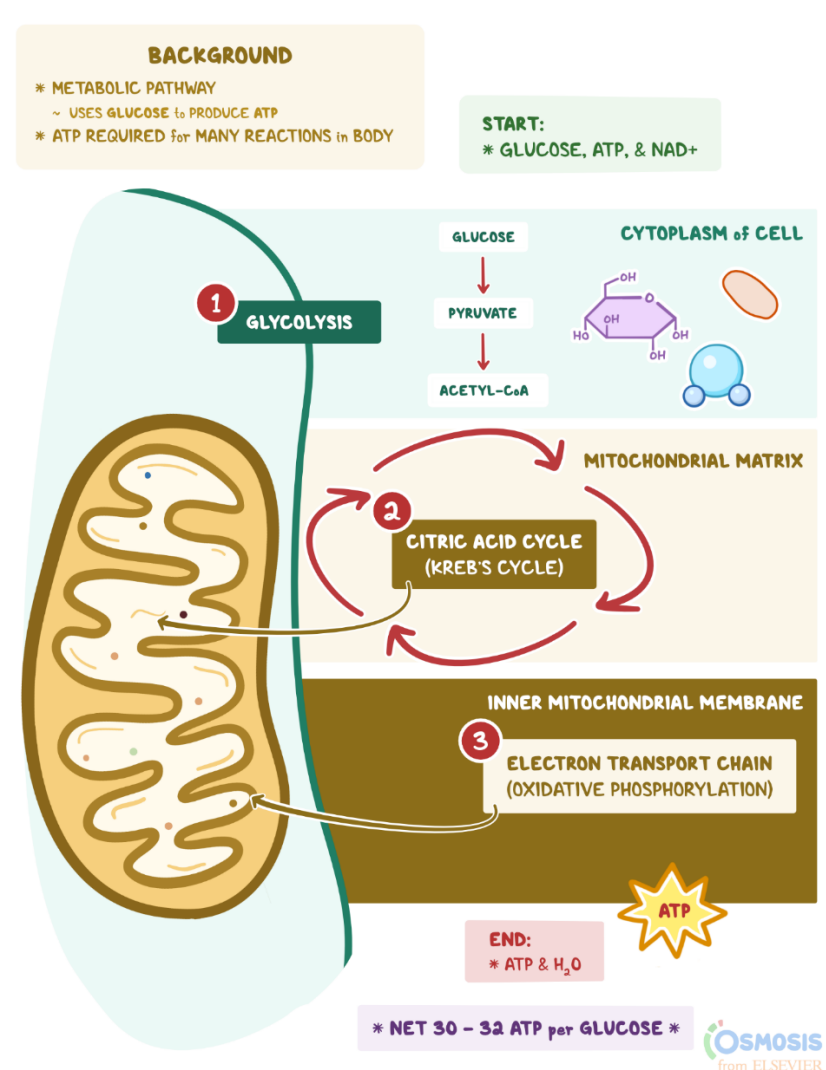


Figure 2: Cellular Respiration process (Osmosis from Elsevier, 2025).

4. The high-energy electrons from NADH and $FADH_2$ are transported in complexes bound to the inner mitochondrial membrane, where they pump hydrogen ions (H^+) across the membrane to form a proton gradient. Molecular oxygen (O_2) serves as the final electron acceptor, which reacts with protons to produce water (H_2O). Most cellular energy is produced by chemiosmosis through ATP Synthase (26-- 28 ATP per glucose molecule) (Hall and Hall, 2020).

Section 2: Pathophysiology & Impact

Type 2 Diabetes Mellitus is a structural and functional metabolic failure of blood glucose metabolism, caused by two key interrelated features, peripheral insulin resistance and beta-cell dysfunction (Davies et al., 2022).

In early stages of disease, there is reduced response of target tissues (skeletal muscle, adipose tissue and liver) to circulating insulin. In the cells, excessive free fatty acids and inflammatory cytokines abolish intracellular insulin signalling pathways, which results in less GLUT4 transporters moving to the cell membrane (Shulman, 2000). This leads to lower uptake of glucose into skeletal muscle following a meal, and increased production of glucose by the liver in the presence of high blood glucose levels.

This is compensated for by hyper-secretion of insulin (hyperinsulinemia) by the pancreatic beta cells in order to maintain a normal plasma glucose level. With time, continuous demand for metabolic, lipotoxicity, and stress on the cells result in progressive beta-cell dysfunction and a decrease in beta-cell mass (Taylor, 2019). If insulin production is not adequate to overcome the peripheral resistance, hyperglycaemia occurs.

Diets containing ultra-processed foods and refined carbohydrates cause quick rises in blood glucose, putting a strain on the pancreatic beta cells. With chronic overconsumption of calories, ectopic fat accumulates in the liver and skeletal muscle, and directly impairs insulin signalling (Taylor, 2019).

Physical Inactivity: Skeletal muscle takes up about 80% of postprandial glucose uptake (Shulman, 2000). Without activity the expression of GLUT4 decreases in muscle. On the other hand, physical activity causes the contraction of the muscles which stimulates the other pathway (AMP-activated protein kinase, or AMPK) that moves GLUT4 to the cell membrane, thus making exercise one of the most effective means for enhancing glucose disposal.

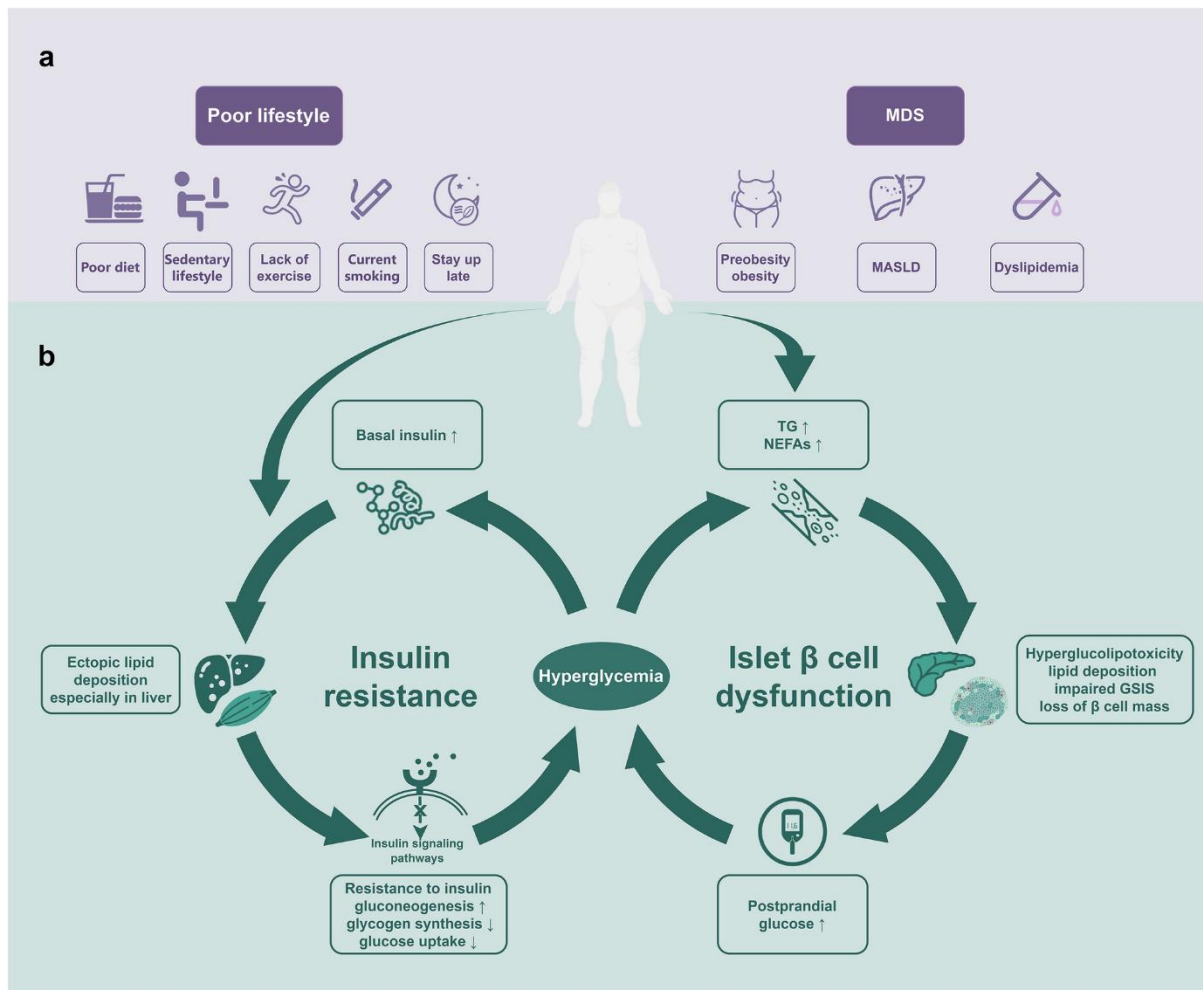


Figure 3: Causes of T2DM (Lu et al., 2024).

Tobacco Smoking: Tobacco smoke contains nicotine and ROS that cause low-grade inflammation and systemic oxidative stress. This raises the levels of counter-regulatory stress hormones (including cortisol and catecholamines) that inhibit the activity of insulin, raise peripheral insulin resistance and promote vascular endothelial injury (American Diabetes Association, 2024).

Section 3: Monitoring & Data Use

Objective clinical measures are crucial in evaluating metabolic control, managing treatment and avoiding long-term vascular complications in the management of Type 2 Diabetes Mellitus (T2DM). Glycated haemoglobin (HbA1c) is the most important marker for assessing long-term glycaemic control. Physiologically, glucose in plasma combines non-enzymatically with haemoglobin in red blood cells at a rate directly related to the plasma glucose level. The average lifespan of erythrocytes is approximately 120 days, which means that HbA1c gives a reliable estimation of average glycemia over the last 2–3 months (Hall and Hall, 2020).

The national clinical guidance states that an HbA1c reading of 48 mmol/mol, or 6.5% (or 53 mmol/mol) for people taking therapies that can cause hypoglycaemia), is a diagnosis of T2DM (NICE, 2026). Primary care providers should monitor their patient's HbA1c routinely every 3 to 6 months to assess the effectiveness of treatment. This longitudinal data is important because it provides evidence to support escalating the use of pharmaceuticals, such as starting metformin, SGLT2 inhibitors, or GLP-1 receptor agonists (Davies et al., 2022).

Daily self-monitoring of blood glucose (SMBG) using finger-prick capillary testing offers immediate, real-time feedback on glucose levels at a given moment. For most people, target ranges for capillary blood glucose tests are below 8.5 mmol/L after eating, and 4.0-7.0 mmol/L before eating (NICE, 2026). Capillary reading can help patients see what meal portions, exercise and medications do to their blood sugar. Moreover, monitoring urgent information allows the person to detect and act on hypoglycaemia (< 4.0 mmol/L) or highs after eating. In addition to glycaemic control, annual monitoring of surrogate microvascular markers like the urine Albumin Creatinine Ratio (ACR) for screening of diabetic nephropathy provides complete risk management and promotes active participation in patient individualization plans.

Section 4: Evidence-Based Lifestyle Recommendations

Lifestyle change is the core base of T2DM care, following NICE Guideline NG28 (Type 2 diabetes in adults: management).

1. In weight management and glycaemic control, one of the first steps is to make dietary adjustments.
 - Recommendation: Eat a low-glycaemic index (GI) diet, including plenty of high-fibre foods, whole grains, vegetables and lean proteins, and less of refined sugars and sugar-sweetened drinks (NICE, 2026). Initial weight loss is suggested to start with an average 5 – 10%.
 - Physiological Mechanism: In high GI foods, the high fibre content slows the emptying of the stomach and the passage of glucose into the blood via the small intestine, helping to diminish the postprandial rise in blood sugar and the acute strain on pancreatic beta cells. In addition, clinical trials show that caloric restriction has been

shown to lower fat levels in the liver and pancreas, which in turn can lead to restored normal insulin sensitivity and beta-cell function ([Lean et al., 2018](#); [Taylor, 2019](#)).

2. Structured Exercise and Physical Activity.

- Recommendation: Engage in at least 150 minutes of moderate intensity aerobic exercise (e.g. brisk walking, swimming, cycling) spread over at least 3 days a week and at least 2–3 sessions of resistance training a week ([NICE, 2026](#); [Davies et al., 2022](#)).
- Physiological Mechanism: When physiological activity occurs aerobically, muscle contraction leads to the activation of AMPK signalling pathways. This facilitates GLUT4 translocation to the membrane of muscle cells without the need of insulin, which rapidly decreases circulating blood glucose (Power and Slattery, 2021). Resistance exercise enhances systemic insulin sensitivity for up to 48 hours after the activity and increases the total amount of skeletal muscle tissue available to take up glucose.

3. Smoking Cessation

- Recommendation: Quitting smoking completely, with the assistance of NHS stop smoking services and pharmacotherapy if suitable ([NICE, 2026](#)).
- Physiological Mechanism: Peripheral insulin resistance is greatly due to systemic inflammation and oxidative stress, which decreases when one quits smoking, and has a significant impact on long-term microvascular and macrovascular risk ([American Diabetes Association, 2024](#)).

Conclusion

Type 2 Diabetes Mellitus is a chronic metabolic disorder, which is associated with insulin resistance and pancreatic beta-cell dysfunction. Hyperglycaemia interferes with the normal use of glucose for energy production and can, over time, cause damage in the vascular network throughout the body. The knowledge that some of the basic metabolic processes such as function of the endocrine pancreas, GLUT4 translocation, and aerobic ATP production by oxidative phosphorylation are impaired by poor diet, physical inactivity and smoking puts this in perspective.

HbA1c is an important clinical data monitoring instrument along with SMG and renal markers that is fundamental to T2DM treatment. The measures offer objective information to help make pharmaceutical adjustments and prevent acute and chronic complications. As indicated in the clinical guidelines, structured lifestyle interventions specifically targeting low-GI dietary

patterns, regular exercise (aerobic and resistance training) and smoking cessation directly target the major contributors to insulin resistance by promoting uptake of glucose independent of insulin and decreasing ectopic accumulation of lipids.

T2DM is managed well by multidisciplinary primary care teams. In addition to prescribing treatments, practice nurses function as educators, informing patients of their condition. Practitioners can help individuals understand how to take care of themselves as they work toward a healthy lifestyle and possibly avoid preventable chronic conditions by interpreting the physiological concepts and making them "user friendly".

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