

Type 2 Diabetes Mellitus: A Case Study on Physiology, Monitoring and Lifestyle Management in Primary Care

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1. Introduction

A long-term metabolic disorder, Type 2 Diabetes Mellitus (T2DM) develops when the tissues gradually stop responding to insulin. The result is hyperglycaemia, and if uncontrolled, can lead to the destruction of the pancreatic cells that make insulin (Basak and Laskar, 2024). Lifestyle factors are the most predominant causes of this disease, and the International Diabetes Federation (IDF) forecasts that by 2050, 853 million adults will be living with the condition (IDF, 2025). In contrast to T1DM, the risk of developing T2DM is driven largely by daily habits (e.g. obesity, physical inactivity, unhealthy eating, smoking) but there are also fixed risks that cannot be changed (e.g. older age, ethnicity, a parent or sibling with diabetes, previous gestational diabetes) (NHS, 2025; Lima, Moreira and Sakamoto-Hojo, 2022). This report explains how glucose is kept in balance and how cells generate energy, the pathophysiology of the disease and its related conditions, glucose tracking, and research-backed lifestyle changes which a practice nurse can apply in day-to-day clinics (NHS, 2025).

2. Blood Sugar Regulation in a Healthy Body

2.1 Normal glucose range

The adult brain requires a constant and stable supply of glucose to function properly. Blood glucose levels must be tightly regulated by the body to ensure that the brain, and other tissues with high glucose requirements, receives a constant supply of glucose to function optimally. In healthy adults, fasting glucose is held at roughly 3.9–5.6 mmol/L (70–100 mg/dL), while a reading taken two hours after eating should stay under 7.8 mmol/L, the threshold above which glucose tolerance is classed as impaired (Betts et al., 2022; World Health Organization, 2006). Low and high glucose are each harmful: hypoglycaemia will affect the brain and hyperglycaemia will damage blood vessels and nerves.

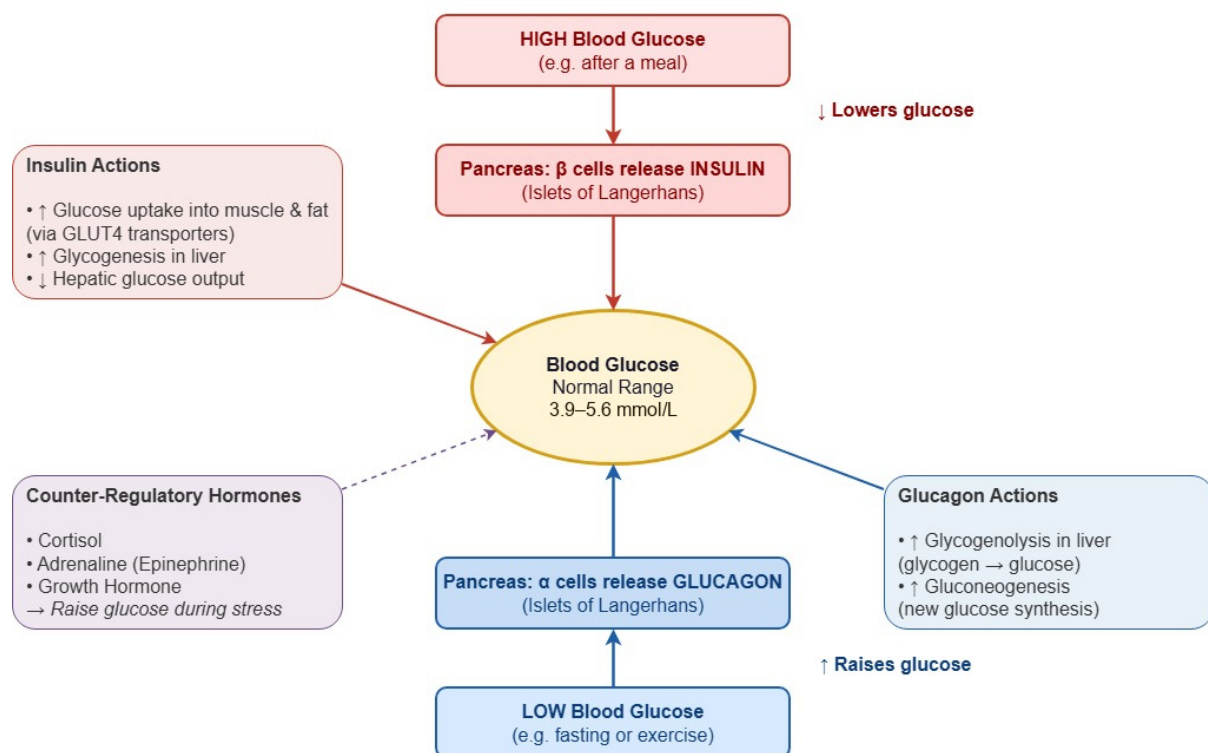
2.2 The pancreas and its hormones

Two islet hormones made by the pancreas are chiefly responsible for keeping glucose stable: insulin, which comes from β cells, and glucagon, which is secreted by α cells (Betts et al., 2022). Insulin release is stimulated by an increase in blood glucose. In the presence of insulin, muscle and adipose tissues take up glucose by transporting glucose transporter 4 (GLUT4) to the membrane (Betts et al., 2022). Insulin also promotes hepatic glycogenesis and inhibits hepatic glucose output (Betts et al., 2022). Between meals, glucagon signals the liver to convert its stored glycogen back into sugar and to carry out gluconeogenesis, so that circulating glucose rises again (Betts et al., 2022).

2.3 Negative feedback loop

Glucose regulation is a classic illustration of a self-correcting (negative feedback) system. As glucose concentration climbs, pancreatic β cells secrete insulin to bring it down by promoting the uptake and storage of glucose in cells and the liver (Lima, Moreira and Sakamoto-Hojo, 2022). On the other hand, when glucose falls, pancreatic α cells secrete glucagon, which raises the level by prompting the liver to discharge its stored sugar into the circulation. Stress and exercise increase the levels of so-called “counter regulatory” hormones, adrenaline, cortisol and growth hormone to protect against hypoglycaemia.

2.4 Visual representation



Negative feedback: insulin lowers glucose; glucagon raises glucose — maintaining homeostasis

Figure 1: Opposing actions of insulin and glucagon in maintaining glucose balance.
Adapted from Betts et al. (2022).

Figure 1 summarises this loop: insulin from β cells lowers glucose, glucagon from α cells raises it, and the counter-regulatory hormones help hold glucose within its normal 3.9–5.6 mmol/L window (Betts et al., 2022).

3. Cell Metabolism - How Oxygen and Glucose Produce Energy

All cells need a constant supply of energy to function, for example to produce muscle contraction, signals for nerves and make biosynthesis. This energy is held in adenosine triphosphate (ATP), the molecule that every cell uses to pay for its energy needs.

Glucose ($\text{C}_6\text{H}_{12}\text{O}_6$) + 6 $\text{O}_2 \rightarrow$ 6 CO_2 + 6 H_2O + usable energy as ATP. This takes place in three successive stages (Betts et al., 2022).

3.1 Glycolysis

Glycolysis takes place in the cytoplasm and does not need oxygen. During this stage, the six-carbon glucose molecule is split into two pyruvic acid molecules, each containing three carbons. In the process, 2 ATP are produced and 2 NADH are made (Betts et al., 2022). This process yields very little energy and is suitable for short spurts of activity when there is no oxygen present.

3.2 Krebs cycle (citric acid cycle)

When oxygen is available, pyruvate is changed into acetyl-CoA, which then feeds into the Krebs cycle. Two carbon dioxide molecules are released for every cycle of the Krebs cycle, and each turn also generates the reduced carriers NADH and FADH_2 together with a little ATP (Betts et al., 2022). These reduced coenzymes act as shuttles, carrying energy-rich electrons on to the final stage.

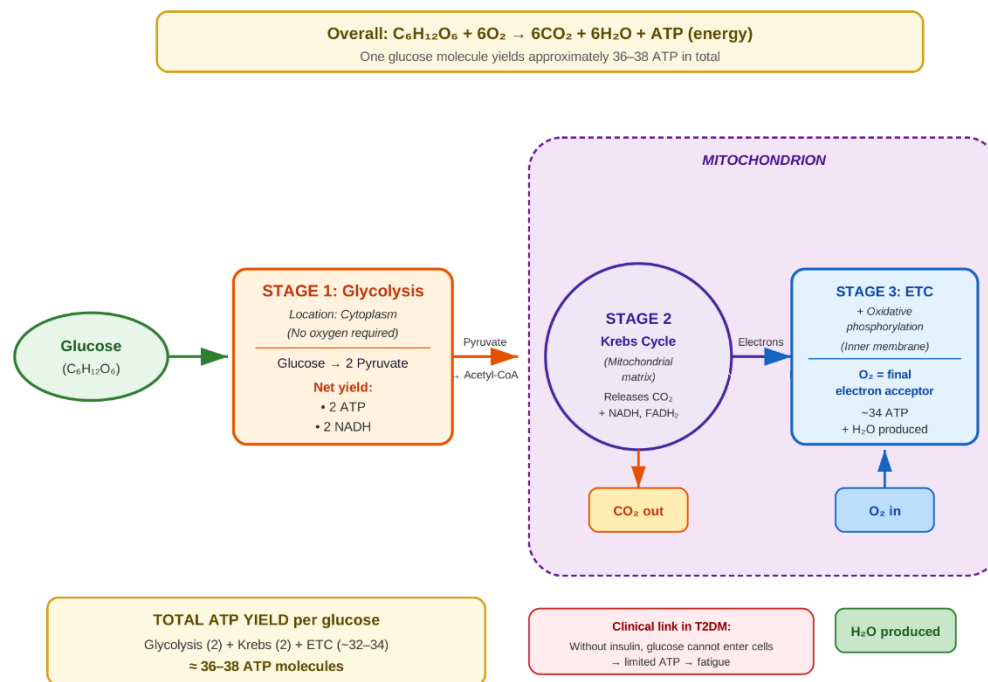


Figure 2: The three stages of aerobic cellular respiration. Adapted from Betts et al. (2022).

3.3 Final stage: electron transport and ATP synthesis

The final and most productive stage takes place across the inner membrane of the mitochondria. Here, electrons donated by NADH and $FADH_2$ pass along a chain of protein complexes, and the energy released drives ATP synthesis. Oxygen is the last molecule to accept these electrons, and it joins with hydrogen ions to form water (Betts et al., 2022). This stage is the most effective in ATP production, and about 36-38 ATP molecules are produced for every glucose molecule.

3.4 Clinical link

Important to note, however, is that insulin is required for the glucose to get into most cells of the body. In T2DM, the problem with glucose signalling becomes that, even though blood glucose is plentiful, cells can no longer access them efficiently, therefore decreasing ATP production which, in turn, increases the fatigue that is often reported by people with T2DM (Betts et al., 2022).

4. Pathophysiology - What Goes Wrong in T2DM

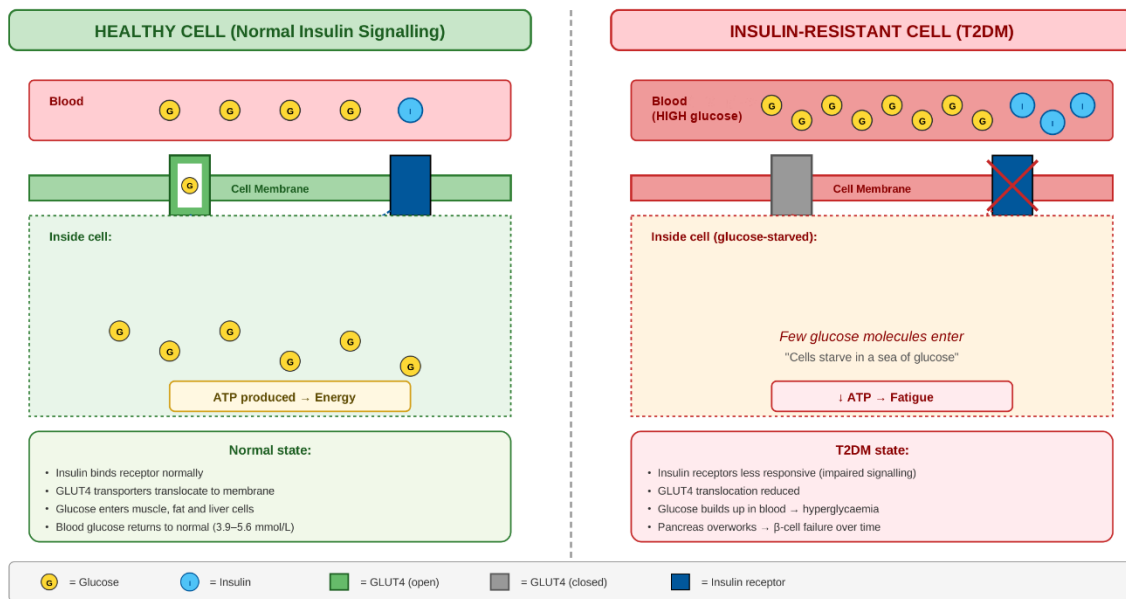


Figure 3: Insulin signalling in a healthy cell compared with an insulin-resistant cell.
Adapted from Lima, Moreira and Sakamoto-Hojo (2022).

4.1 Insulin resistance and β -cell dysfunction.

Insulin resistance is the core problem in T2DM, involving less sensitivity of cells in skeletal muscle, adipose tissue and liver to the action of insulin. At first, the pancreas tries to make up for this and releases more insulin, giving rise to hyperinsulinemia. However, as the disease progresses, the body's insulin production goes down and the β cells can no longer secrete enough insulin to meet the body's demand, leading to relative insulin deficiency and continued hyperglycaemia (Basak and Laskar, 2024). Chronic hyperglycaemia also leads to oxidative stress and low-grade inflammation, thereby impairing insulin signalling (Lima, Moreira and Sakamoto-Hojo, 2022).

4.2 Short-term symptoms

Patients with poorly controlled T2DM typically present with polyuria (excessive urination due to osmotic diuresis), polydipsia (excessive thirst), tiredness, blurred eyesight and cuts that take longer to heal (Basak and Laskar, 2024). These symptoms can also build up over time and are sometimes thought to be symptoms of stress or normal signs of ageing.

4.3 Long-term complications

Uncontrolled hyperglycaemia leads to extensive vascular damage. Damage to small vessels (microvascular) can cause foot ulceration, nerve damage in the limbs (peripheral neuropathy) and kidney disease (nephropathy, which can lead to end stage renal disease), and these

problems are among the leading causes of adult blindness. Damage to larger vessels (macrovascular) raises the risk of heart attack, stroke and poor circulation in the legs (Lima, Moreira and Sakamoto-Hojo, 2022). T2DM also increases the susceptibility of an infection because of the impaired immune function.

4.4 Impact of lifestyle factors

Lifestyle choices are large contributors to T2DM. High consumption of foods with a high glycaemic index, such as refined sugar and carbohydrates, leads to high glucose levels post consumption, weight gain, and insulin resistance. Inactivity contributes by decreasing the body's ability to remove glucose (through GLUT4-mediated glucose transport in skeletal muscle), the body's most significant glucose sink. Cigarette smoking has been linked to insulin resistance, although a recent case-control study found no significant direct association in healthy adults and suggested that smoking worsens insulin resistance through other contributing factors (Kaplan and Sezgin, 2023). Visceral adipose tissue further impairs insulin signalling by releasing pro-inflammatory signalling molecules, including TNF- α and IL-6 (Lima, Moreira and Sakamoto-Hojo, 2022).

5. Monitoring and Use of Routine Health Data

5.1 Methods of measurement

Regular and correct blood glucose monitoring is important for the management of T2DM. Glycated haemoglobin (HbA1c) is the primary method for long-term monitoring in primary care in the UK and averages blood glucose levels over the last 2–3 months. Adults managed through diet and exercise, or with one drug that does not cause hypos, should aim for an HbA1c of 48 mmol/mol (6.5%), whereas those taking a drug that can trigger low glucose are given a goal of 53 mmol/mol (7.0%), as set out by the National Institute for Health and Care Excellence (2026). Some patients also check their own levels at home (SMBG) with finger prick meters to give real time information, and continuous glucose monitoring (CGM) is becoming more common in patients with more complex glycaemic patterns. Blood pressure, weight, BMI, lipid profile, foot health and retinal screening are also measured as part of the annual review (NHS, 2025).

5.2 Why tracking matters

Documenting the blood glucose record enables the patient and healthcare provider to determine trends and patterns: for example, post meal spikes, dawn hyperglycaemia and the impact of exercise, which can then be used to tailor the healthcare plan, diet, or activity. Holmes-Truscott et al. (2023), pooling the findings of many earlier studies, reported that

planned and regular home glucose testing led to a small yet statistically significant decrease in HbA1C levels in adults with non-insulin treated T2DM, as well as improvement in diabetes self-efficacy. The findings underscore the importance of monitoring beyond the numbers, emphasizing its behavioural aspect and its potential to engage the patient and empower them with knowledge for decision-making.

5.3 Routine data at system level

In addition to this, routine data are collected in primary care – through the GP incentive scheme known as QOF and the yearly national audit of diabetes care – and are used to inform service design and quality improvement in general practice (NHS England, 2026). A continuous cycle of information that leads to improvements in care pathways, resource allocation and in public health policy, as individual-level data contribute to population-level information. This is significant for the practice nurse because each of the HbA1c readings taken in clinic is a part of a national picture of the health system's diabetes control.

6. Evidence-Based Lifestyle Recommendations

6.1 Dietary modification

The primary treatment for T2DM is diet. A Mediterranean-style eating pattern, built on plenty of plant foods (vegetables, fruit, wholegrains and pulses), olive oil as the main fat, and seafood eaten in moderation, was significantly effective in decreasing HbA1c, fasting glucose and body weight in adults with T2DM when compared to control diets, according to Zheng et al. (2024). Other steps are generally recommended, such as cutting out refined carbohydrates, sugar-sweetened drinks and processed foods (NHS, 2025). Any dietary plan should be realistic, culturally acceptable and achievable for the patient.

6.2 Weight management

The greatest lifestyle intervention for T2DM has become weight loss. DiRECT was the first diabetes remission clinical trial, showing that clinically meaningful diabetes remission can be achieved with structured weight management in UK primary care. At 5 years, 13% of extension participants were in remission, and of those in remission at year 2, 26% remained in remission at year 5 (Lean et al., 2024). There is evidence to support a structured weight loss programme, such as the NHS low-calorie 'Path to Remission' scheme, as an integral part of contemporary diabetes management.

6.3 Physical activity

Exercise has been shown to have glycaemic benefits independent of insulin, such as contraction-induced GLUT4 translocation in skeletal muscle (Laktineh et al., 2026). UK

guidance advises adults to complete at least two and a half hours of moderate cardio activity each week, plus muscle-strengthening work on two days (NHS, 2025). Even modest increases in daily walking produce measurable improvements in insulin sensitivity.

6.4 Smoking cessation

Smoking cessation is an important intervention that is underemphasized. Bellanca et al. (2024) show that smoking alters the metabolism of antidiabetic drugs by inducing liver enzymes, so quitting can improve medication efficacy, although doses may need adjusting after cessation. Patients should be encouraged to attend NHS Stop Smoking Services regularly, be provided with appropriate pharmacotherapy and receive behavioural counselling support (NHS, 2025).

6.5 Structured education

Lastly, structured education programmes like DESMOND and X-PERT provide patients with knowledge and confidence to manage their condition, thus enhancing their HbA1c levels, dietary habits and mental health (NHS, 2025).

7. Conclusion

In summary, T2DM arises when insulin becomes steadily less able to adequately regulate blood sugar, which in turn damages blood vessels. It is associated with breakdown of the interaction of the pancreas, liver, muscle and adipose tissue, which is normally under the negative feedback control of insulin and glucagon. The breakdown occurs in a stepwise fashion and progressively results in poor blood sugar and cellular energy regulation. Evidence-based lifestyle changes (Mediterranean diet, weight loss, physical activity, stopping smoking and structured education) that are available to practice nurses facilitate improvement in blood sugar regulation and possibly even remission (Lean et al., 2024). The increasing global diabetes prevalence forecasts (IDF, 2025) requires that practice nurses bridge the knowledge of clinical physiology and behavioural science to promote informed self-management.

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