Blood Sugar & Insulin

What Everyone Should Know About Glucose, Metabolism, and Long-Term Health

Dr. Yves J. Hilpisch[1]

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Abstract

Blood glucose and insulin are among the most discussed—and most misunderstood—markers in modern health culture. This paper builds a mental model of glucose metabolism as a regulatory system rather than a score to be minimised. It explains what normal blood sugar looks like after a meal, why insulin resistance can hide behind normal glucose readings for years, how the major tests (fasting glucose, HbA1c, the oral glucose tolerance test, insulin measurements, and continuous glucose monitoring) reveal different aspects of the same system, and which lifestyle factors most strongly shape long-term metabolic health. The paper separates established evidence from plausible but less-confirmed optimisation strategies, examines five popular claims about insulin and glucose, and closes with a hierarchy of what actually matters most.

[1]Get in touch: https://linktr.ee/dyjh. Web page: https://hilpisch.com. Research, structuring, drafting, and visualizations were assisted by different LLMs as co-writing tools under human direction. Comments and feedback are welcome.

Contents

1 Introduction

You can think of your blood glucose system as the body's energy grid: glucose is the electricity, insulin is the control signal, and your liver, muscle, fat tissue, gut, and pancreas are the producers, storers, sensors, and consumers that keep supply and demand in balance. Most popular health advice flattens this system into a single number—how high your glucose rises after a meal—and then sells you ways to flatten that number. This paper takes a different view: what matters for long-term health is not a perfectly flat glucose curve, but a regulatory system that can efficiently handle food, fasting, exercise, stress, illness, and changing energy demands.

The subject matters because glucose regulation sits at the intersection of energy, cardiovascular risk, diabetes prevention, body composition, fitness, and daily behaviour. Yet contemporary glucose culture often over-simplifies it. It can make a banana look suspicious because a sensor line rises, while making a low-carbohydrate meal look healthy because the same line stays flat. That is measurement without physiology.

This paper is written for educated general readers interested in health, longevity, fitness, and prevention, and for supporters who translate evidence into practical routines. Readers with diagnosed diabetes, prediabetes, pregnancy, cardiovascular disease, kidney disease, or medication questions should treat it as background education only. This paper is educational and does not provide individual medical advice.

The aim is to build one durable mental model: glucose metabolism is a system, not a score. The paper first explains the physiology, then shows how normal dynamics differ from insulin resistance, how common tests reveal different slices of the system, which determinants deserve priority, and where popular claims about glucose and insulin go wrong.

2 Executive Summary: The Most Important Things to Know

Before the details, here are the ten conclusions the rest of the paper builds toward. Together they form the paper's recurring idea: glucose metabolism is a system, not a score.

1. **Glucose is essential fuel, not a toxin.** Your body runs on it, stores it, and manufactures it when you do not eat.

2. **Insulin is an essential regulatory hormone, not an enemy.** A healthy insulin rise after a meal is normal physiology, not a problem to be suppressed.

3. **Blood glucose normally rises after eating.** A temporary post-meal excursion is expected, even in the metabolically healthiest people.

4. **The size of a glucose rise alone does not determine whether a meal is healthy.** A flat glucose trace can coexist with a poor diet.

5. **Insulin resistance means tissues require a stronger insulin signal to produce a given metabolic response.** It is a property of the response, not of the insulin itself.

6. **The pancreas can compensate for insulin resistance for years.** Apparently normal glucose does not necessarily imply optimal insulin sensitivity.

7. **Muscle, liver, adipose tissue, and pancreas play distinct roles** in glucose regulation, and can become insulin resistant to different degrees.

8. **Physical activity, body composition, diet quality, sleep, and genetics** all shape metabolic health; no single factor explains it.

9. **Fasting glucose, HbA1c, an oral glucose tolerance test, insulin measurements, and CGM reveal different aspects** of the system; no single test captures it all.

10. **Long-term metabolic capacity matters more than eliminating every short-term glucose excursion.**

3 The Glucose–Insulin System

Before judging glucose curves, it helps to know what the system is for. This section explains why blood glucose exists, what insulin actually does, and why insulin never works alone.

3.1 Why We Have Blood Glucose

Glucose is one of the body's fundamental energy substrates. Standard physiology texts describe the same basic control problem: maintain circulating glucose within a narrow enough range to supply tissues while avoiding sustained excess.[1] Dietary carbohydrate is digested into glucose and other sugars, which enter the bloodstream through the gut. Cells use some of it immediately for energy; the rest is stored. Skeletal muscle and the liver store glucose as glycogen—muscle glycogen serves the muscle itself, while liver glycogen acts as a shared reservoir that can be released back into the blood between meals. When dietary carbohydrate is scarce, the liver manufactures new glucose from amino acids, glycerol, and lactate through gluconeogenesis, maintaining the glucose supply required by tissues such as red blood cells and by the brain, which normally relies heavily on glucose but can also use ketone bodies during prolonged fasting.

Two complementary points follow, and the whole paper depends on keeping both in view. The body's ability to manufacture glucose does not mean that carbohydrate-containing foods are inherently undesirable. And the fact that glucose is essential does not mean that chronically elevated blood glucose is harmless: sustained high glucose damages blood vessels and nerves over the years. Essential and harmless are different properties.

3.2 The Energy Grid Analogy

A useful mental model is an electricity grid:

- the **bloodstream** is the distribution grid carrying immediately available energy;
- the **liver** is the balancing station that stores, releases, and manufactures glucose;
- **glycogen** is short-term storage;
- **adipose tissue** is the large long-term energy store;
- **muscle** is a major consumer and site of glucose disposal, with its own local storage;
- **hormones** are the control signals coordinating all of it.

Note that a healthy electricity grid is not one in which electricity never moves. It is one in which supply and demand remain controlled. That distinction anticipates the later discussion of glucose "spikes" (Section 4.4): movement on the grid is not itself a malfunction.

3.3 What Insulin Actually Does

Insulin is produced by the pancreatic beta cells and signals nutrient availability. Modern reviews of insulin action emphasise coordinated effects across muscle, liver, adipose tissue, and vascular and neural systems rather than a single blood-sugar switch.[2] After a meal, rising glucose—together with amino acids and gut-derived hormones such as the incretins—stimulates insulin secretion. Insulin then coordinates a whole-body shift from mobilisation to storage: it promotes glucose uptake, particularly in muscle and adipose tissue; stimulates glycogen synthesis; suppresses excessive hepatic glucose production; and influences protein and lipid metabolism.

This is broader than the popular characterisation of insulin as "the hormone that lowers blood sugar." Lowering blood glucose is one consequence of a general signal: *energy is available; use it and store it.* Two corrections follow. First, a healthy insulin rise following a meal is normal physiology: severe insulin deficiency impairs normal nutrient handling and tissue storage, precipitating dangerous hyperglycemia and systemic metabolic disturbances. Second, protein also stimulates insulin secretion, which already undermines simple stories in which insulin rises are uniquely caused by, and harmful because of, carbohydrate.

3.4 Insulin Does Not Work Alone

Insulin's counterpart is glucagon, produced by the pancreatic alpha cells. In the fed state, relatively stronger insulin signalling favours nutrient utilisation and storage. During fasting, lower insulin and relatively stronger glucagon signalling permit the liver to maintain circulating glucose through glycogen breakdown and glucose production. The two hormones form a feedback controller: one acts like the accelerator for storage, the other like the accelerator for release.

Other signals modulate the system as well. Cortisol and adrenaline raise glucose availability during stress and illness, growth hormone influences overnight metabolism, and the autonomic nervous system adjusts both hormone secretion and hepatic glucose output. Figure 1 summarises the model: a distribution network with storage, consumers, and hormonal feedback. Keep this picture in mind—every measurement discussed later is a partial view of it.

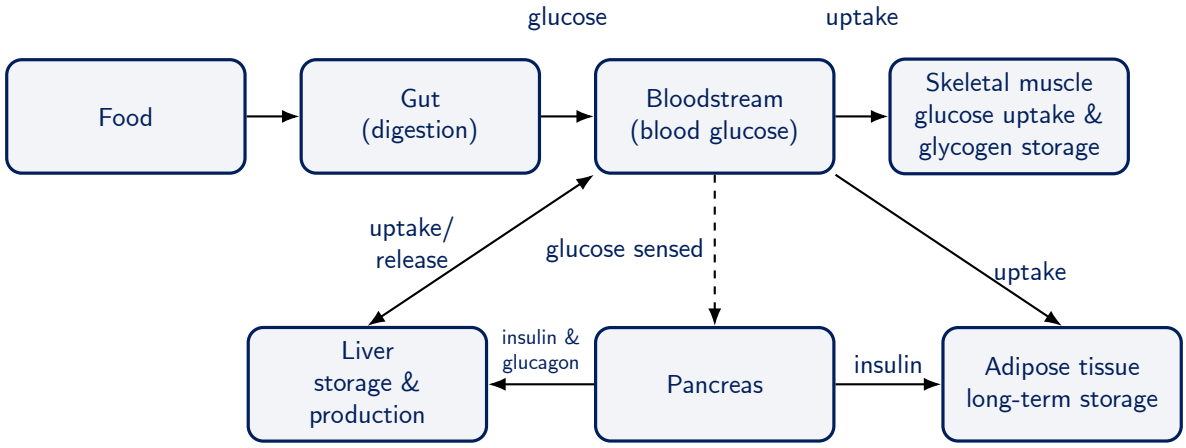

Figure 1: The glucose–insulin system as a regulatory network. The pancreas senses blood glucose and adjusts insulin and glucagon; the liver stores, releases, and produces glucose; muscle and adipose tissue take up and store nutrients. Solid arrows show glucose movement, dashed arrows hormonal feedback.

4 What Normal Blood Sugar Looks Like

Now that the system is in place, the question becomes empirical: what does normal actually look like? This section walks through a meal, explains why curves differ, and asks whether "spikes" are a problem.

4.1 What Happens After a Meal

Consider an ordinary mixed meal—vegetables, rice, chicken, olive oil, and fruit. Within minutes, digestion converts digestible carbohydrates into absorbable monosaccharides, including glucose, which enter the circulation through the gut. Rising glucose, amplified by incretin hormones, stimulates insulin secretion. Within the first hour, insulin suppresses hepatic glucose production and switches the liver from releasing glucose to storing it, while muscle and other tissues take

up nutrients. Glucose reaches a temporary peak, typically within the first hour, and then moves back toward its pre-meal level over the following two to three hours as uptake continues and the insulin signal gradually declines.

This sequence has a name in the clinical literature: the *postprandial* (after-meal) *glycemic excursion*. The essential point is that a temporary increase in blood glucose after eating is expected physiology. The system is doing precisely what it was built to do: handle an incoming load and return to baseline.

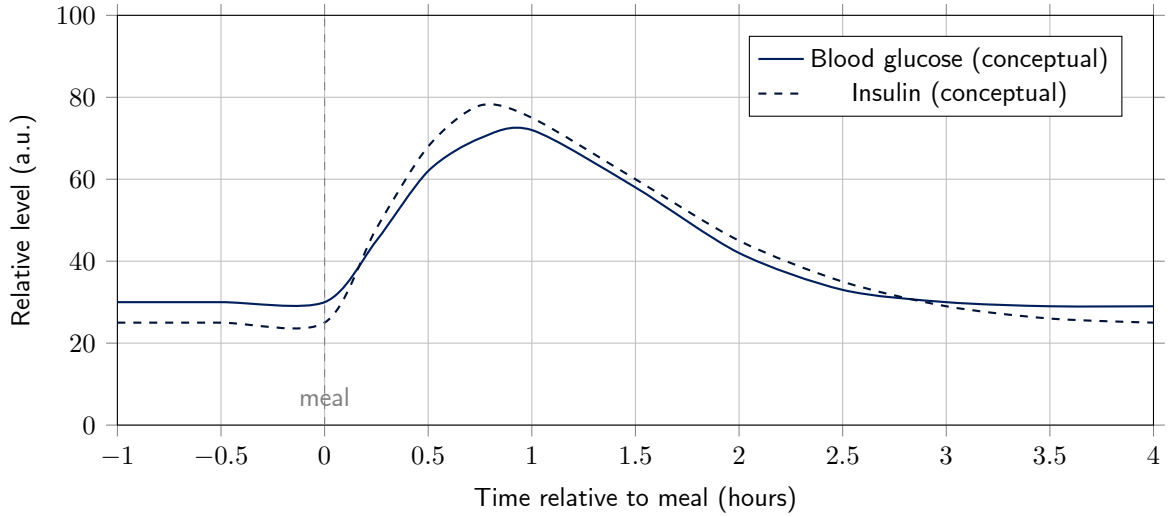

Figure 2: A normal meal response: conceptual glucose and insulin curves after a mixed meal. Both rise, peak within about an hour, and return toward baseline within two to three hours. This is a conceptual illustration of the pattern, not a universal reference value—magnitudes and timings vary substantially between people, meals, and days.

4.2 Why Different Meals Produce Different Curves

The shape of the curve depends strongly on the food. The main food-related factors are the amount of digestible carbohydrate, the carbohydrate structure and degree of processing, fiber content, the food matrix (whole versus ground, solid versus liquid), and the accompanying protein and fat. Protein, fat, fiber, and food structure can alter gastric emptying, nutrient absorption, hormonal responses, and therefore the resulting glucose curve.

Two comparisons make this concrete. An apple and a glass of apple juice come from the same underlying food, but the apple arrives with intact cell structure and fiber, takes longer to eat, is absorbed more slowly, and satisfies appetite more effectively; the juice delivers its sugars in seconds. White bread eaten alone often produces a faster, higher excursion than the same bread eaten as part of a mixed meal with protein, fat, and vegetables, because the surrounding meal changes delivery, absorption, satiety, and hormonal signalling.

The concepts of *glycemic index* (how quickly a fixed amount of carbohydrate from a food raises glucose relative to a reference) and *glycemic load* (index scaled by the amount of carbohydrate actually served) capture part of this. They are useful research constructs, with limits. A systematic review and meta-analysis of randomised trials found modest improvements in glycemic control and several cardiometabolic markers from lower-GI/GL dietary patterns in people with diabetes.[3] The evidence concerns dietary patterns, not proof that every individual high-GI food is harmful—and results in people with diabetes should not be extrapolated to healthy populations without qualification.

4.3 Why the Same Meal Can Affect Two People Differently

Glucose response depends on far more than the food itself. The same meal lands differently depending on insulin sensitivity, beta-cell function, recent exercise and muscle glycogen levels, sleep, stress, previous meals, time of day, medications, gastric emptying, genetics, age, and body composition. The same person can even respond differently to the same meal on different days—a poorly slept Tuesday is not a well-slept Saturday.

This context dependence leads to a principle that recurs throughout the paper: a glucose curve describes a response in a particular context. It is not necessarily a verdict on the food that produced it. Section 7 returns to the full set of influences; Figure 2 should therefore be read as "one meal in one context," never as "the response to this food."

4.4 Are "Glucose Spikes" Bad?

This is one of the central contemporary questions, and the honest answer avoids both extremes. It is simply not true that glucose spikes do not matter: chronic hyperglycemia in diabetes is clearly associated with harm, and impaired postprandial glucose regulation has recognised clinical relevance. But it is equally untrue that every glucose spike causes damage.

Large continuous-monitoring datasets show what healthy curves actually look like. In a multicentre study of healthy, non-diabetic participants, CGM profiles showed substantial physiological variability with normal post-meal excursions.[4] In the Framingham-based community cohort, normoglycemic participants spent on average about 87% of CGM time between 70 and 140 mg/dL (3.9–7.8 mmol/L)—and still recorded some values above commonly promoted consumer "limits."[5] Healthy glucose is not perfectly flat.

An analogy helps calibrate intuition, with the explicit caveat that it is illustrative rather than physiologically exact. A rise in heart rate during exercise is not in itself cardiovascular pathology; a rise in glucose after eating is not in itself metabolic pathology. In both cases, what matters is context, magnitude, duration, recovery, and the underlying physiology. A system that never responded would be far more worrying than one that responds and recovers.

5 When the System Becomes Less Responsive

Normal physiology established, the paper now turns to pathology-in-the-making. This section defines insulin sensitivity and resistance, explains the compensation that can hide decline for years, and traces the road from resistance to type 2 diabetes.

5.1 Insulin Sensitivity and Insulin Resistance

Insulin sensitivity is simply how strongly the body responds to a given insulin signal. Clamp studies and physiological reviews consistently identify skeletal muscle as the major site of insulin-stimulated postprandial glucose disposal.[6, 2] Insulin resistance is the converse: a state in which more insulin is required to produce a given metabolic effect. Importantly, insulin resistance is not necessarily uniform throughout the body. Skeletal muscle, liver, and adipose tissue can become insulin resistant to different degrees and through partly different mechanisms, and pancreatic beta cells can fail independently. Modern reviews emphasise exactly this tissue specificity and the inter-organ crosstalk that drives progression toward type 2 diabetes.[7] The multi-organ picture replaces the older idea of insulin resistance as a single switch you either have or lack.

5.2 Compensation: Why Normal Glucose Can Hide a Problem

Here is one of the paper's key insights. Imagine two people with the same fasting glucose. Person A regulates that glucose with modest insulin secretion; Person B requires substantially more insulin to achieve the same number. The glucose reading alone cannot distinguish them—the difference is invisible unless insulin itself is measured or challenged.

The progression runs conceptually as follows. With high insulin sensitivity, only modest insulin is required. As sensitivity declines, the pancreas may compensate by secreting more insulin, and glucose can remain normal—sometimes for years. As deterioration continues, compensation becomes increasingly difficult, and when beta-cell function can no longer keep up, glucose rises persistently. This is a useful canonical trajectory rather than a mandatory sequence: the relative timing and severity of insulin resistance and beta-cell dysfunction vary substantially among individuals. Figure 3 sketches the canonical pattern: glucose often deteriorates relatively late, after hidden rises in resistance and insulin.

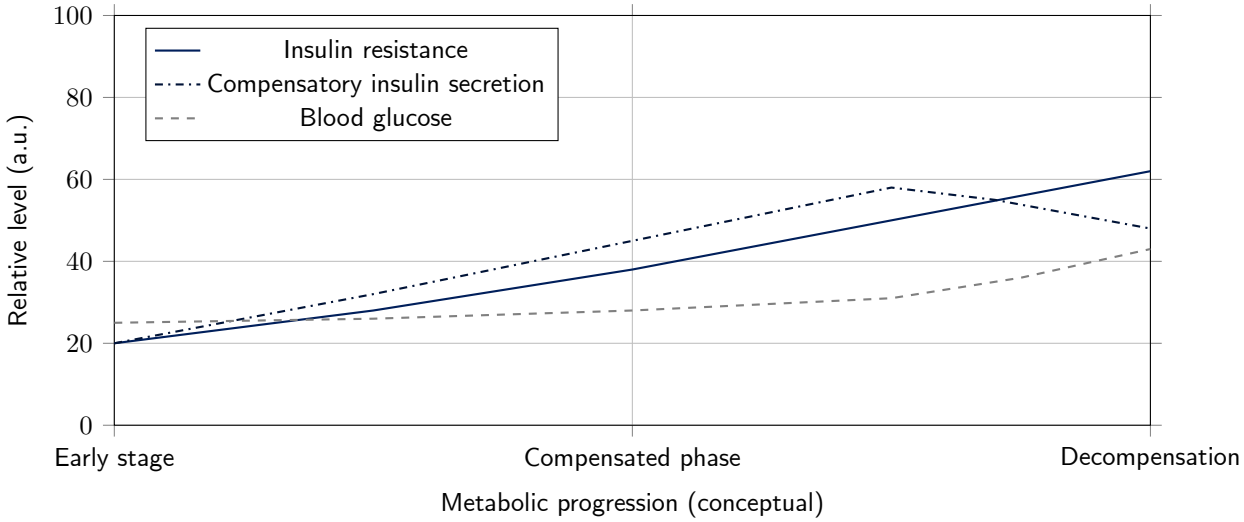

Figure 3: Compensation before hyperglycemia (conceptual model—individual trajectories differ). Insulin resistance may rise; insulin secretion may compensate for years; blood glucose often deteriorates relatively late, when beta-cell compensation fails. This is why "my glucose is normal" and "my metabolism is maximally healthy" are not identical statements.

5.3 Why Insulin Resistance Develops

Insulin resistance is multifactorial, and honest writing about it resists single-cause stories. The strongest relevant factors are genetic susceptibility, chronic energy surplus, excess adiposity where present (especially visceral and ectopic fat, including liver fat), adipose-tissue dysfunction, physical inactivity, loss of muscle mass and fitness, aging, sleep disruption, and certain medications and diseases.[8, 6, 2]

One distinction deserves particular care: body weight and fat distribution are not the same thing. Body mass index (BMI) is an imperfect proxy. People with the same BMI can differ substantially in visceral fat, liver fat, muscle mass, fitness, and metabolic risk. Statements such as "sugar causes insulin resistance" or "being overweight causes diabetes" both discard this causal complexity—each is a slogan standing in for a multifactorial process with a strong dose of individual variation.

5.4 From Insulin Resistance to Type 2 Diabetes

Type 2 diabetes emerges from the interaction of insulin resistance, insufficient compensatory insulin secretion, progressive beta-cell dysfunction, dysregulated hepatic glucose production, and broader multi-organ metabolic dysfunction.[7] The current American Diabetes Association (ADA) classification explicitly characterises type 2 diabetes in terms of progressive loss of adequate beta-cell insulin secretion, frequently occurring against a background of insulin resistance.[9]

The contrast with type 1 diabetes matters because popular discussions sometimes talk about "insulin" as though excess insulin itself were the disease.

Type 1 vs. Type 2 Diabetes

Type 1 diabetes is primarily autoimmune destruction of the beta cells, leading to severe insulin deficiency; people with type 1 diabetes depend on insulin replacement for survival. In type 1 diabetes, the insulin signal becomes severely deficient. In type 2 diabetes, tissues commonly become less responsive while beta cells progressively lose the ability to provide sufficient compensatory insulin.

6 Measuring Glucose and Metabolic Health

The system has a normal side and a deteriorating side; the next question is how to observe it. The major tests answer different questions, and confusing them produces most of the popular misunderstandings about glucose.

6.1 Different Tests Answer Different Questions

Fasting glucose is a snapshot under standardised conditions. HbA1c is an indirect, longer-term measure reflecting average glycemic exposure over the preceding weeks to months. The oral glucose tolerance test (OGTT) is a standardised stress test: a fixed 75-gram glucose load reveals how the system handles a challenge. Insulin measurements add information about the hormonal effort behind the observed glucose result. Continuous glucose monitoring (CGM) is a movie: high-frequency measurement of interstitial glucose dynamics throughout ordinary life.

An analogy ties them together. No single camera captures the entire system: fasting glucose is a snapshot, HbA1c a long exposure, the OGTT a stress test, CGM a movie, and insulin measurement information about the control signal behind the observed result. Figure 4 shows how each camera should be interpreted.

Measure	Best viewed as	Main limitation
Fasting glucose	Baseline snapshot	Limited view of post-meal dynamics
HbA1c	Longer-term exposure	Indirect; hides temporal variation
OGTT	Standardised challenge	Artificial test, not everyday life
Insulin	Hormonal response	Limited assay standardisation/interpretation
CGM	Continuous dynamics	Interstitial measurement; interpretation in healthy people uncertain

Figure 4: Five ways of looking at glucose metabolism. Each measure is useful when its question is clear: snapshot, exposure, challenge, hormonal effort, or continuous dynamics. The limitations are not defects; they are reminders that no single measure captures the whole regulatory system.

6.2 Fasting Glucose, HbA1c, and OGTT

For nonpregnant adults, the current ADA criteria define diabetes through laboratory findings including fasting plasma glucose of at least 126 mg/dL (7.0 mmol/L), HbA1c of at least 6.5%,

a 2-hour plasma glucose of at least 200 mg/dL (11.1 mmol/L) during a 75-gram OGTT, or, under appropriate symptomatic circumstances, a random plasma glucose of at least 200 mg/dL (11.1 mmol/L).[9] Prediabetes corresponds to a fasting glucose between 100 and 125 mg/dL (5.6 and 6.9 mmol/L), HbA1c of 5.7–6.4%, or 2-hour OGTT glucose of 140–199 mg/dL (7.8–11.0 mmol/L). In the absence of unequivocal hyperglycemia, diagnosis generally requires confirmation by repeat testing; one borderline reading is not a diagnosis.

Numerical discussions run in both unit systems, so keep one conversion in mind: $mmol/L \approx mg/dL/18$. Table 1 lists common values.

Table 1: Common blood glucose values in both unit systems.

mg/dL	70	90	100	126	140	180	200
mmol/L	3.9	5.0	5.6	7.0	7.8	10.0	11.1

HbA1c comes with its own caveat, recognised in laboratory guidance for diabetes diagnosis and monitoring:[10] the measure estimates glycemic exposure through glycation of haemoglobin in red blood cells, so anything that changes erythrocyte lifespan (anaemia and other haematological conditions, haemoglobin variants, pregnancy, kidney disease) or the assay itself can shift the reading without any change in true glycemia. HbA1c is valuable precisely because it is a long exposure; it is imperfect because it is an indirect one.

6.3 What About Insulin?

Fasting insulin and derived indices such as the homeostatic model assessment of insulin resistance (HOMA-IR) can provide useful information. HOMA-IR is roughly proportional to fasting insulin times fasting glucose. In research, the hyperinsulinemic-euglycemic clamp remains the reference technique against which simpler measures are judged.[11, 12]

But interpretation limits are real. Insulin assays are less standardised than glucose diagnostics; proposed thresholds differ across populations; fasting insulin does not capture all forms of insulin resistance; and tissue-specific resistance cannot be reduced to a single number. The reasonable takeaway is to understand why measuring insulin is interesting—it sees the compensation that glucose alone misses (Figure 3)—without treating a single fasting insulin value as a definitive metabolic-health score.

6.4 CGM: Powerful but Easy to Overinterpret

A CGM sensor sits in the subcutaneous tissue and measures glucose in the interstitial fluid—not the blood itself. The two track each other, with a physiological lag of minutes and additional sensor uncertainty, which matters for fast-changing excursions. For people with diabetes, CGM has been transformative, enabling titration of therapy and detection of dangerous excursions that spot tests miss. Large diabetes trials and meta-analyses support improvements in glycemic management in insulin-treated populations, while this evidence does not automatically transfer to metabolically healthy users.[13, 14]

Its use among people without diabetes is more nuanced. CGM can reveal personal glucose responses, broad patterns, and associations with meals or exercise, and some users may find that information behaviourally useful. Whether such use materially improves long-term health outcomes in people without diabetes remains uncertain. It should not be sold as a universal health-optimisation system. The current ADA standards of care state that evidence is insufficient to support CGM for screening or diagnosis of prediabetes or diabetes.[9] Laboratory testing remains the diagnostic standard.

The deeper lesson is general: more measurements do not automatically create more knowledge. Interpretation matters, and a CGM trace without context—sleep, exercise, meal compo-

sition, timing—can mislead as easily as inform.

7 What Most Strongly Shapes Metabolic Health?

From measurement back to physiology in motion. Rather than cataloguing dozens of "hacks," this section organises the evidence around the major determinants of metabolic health.

7.1 Food: Quality, Quantity, and Context

The metabolic impact of food cannot be summarised by whether it spikes glucose. What matters is the whole picture: total energy intake, carbohydrate quantity and quality, the balance of whole versus highly processed foods, fiber, protein, and fat, food structure and energy density, satiety, and the overall dietary pattern. Dietary guidelines and prospective evidence consistently support higher-quality, fiber-rich dietary patterns and caution against sugar-sweetened beverages and highly refined, energy-dense patterns.[15, 16, 17]

A deliberately provocative comparison makes the point. Compare Meal A—lentils, vegetables, and fruit—with Meal B—processed meat and high-fat cheese with virtually no carbohydrate. Meal B might well produce the flatter immediate CGM trace. That establishes nothing about nutritional superiority: CGM does not directly measure dietary fiber, micronutrients, LDL cholesterol, blood pressure, total energy balance, or long-term cardiovascular outcomes. Optimising the glucose curve is not synonymous with optimising the diet.

Meal order is worth mentioning only as an illustrative secondary intervention: eating vegetables and protein before carbohydrate can influence acute postprandial responses in some experiments. Such effects are real but belong several evidential levels below broad diet quality, energy balance, and physical activity.

7.2 Exercise and Skeletal Muscle

Skeletal muscle is a major site of glucose disposal, particularly after meals and during physical activity, and contracting muscle can stimulate glucose uptake through mechanisms that are not identical to insulin signalling—which is why muscular activity can substantially increase glucose uptake even when insulin action is impaired. Regular exercise improves insulin sensitivity, glucose handling, cardiorespiratory fitness, body composition, and muscle function. Exercise-position statements and meta-analyses support both aerobic and resistance training for glycemic control in type 2 diabetes and for cardiometabolic risk reduction more broadly.[18, 19] Both broad categories matter: aerobic activity (walking, running, cycling, swimming) builds the oxidative machinery, while resistance training maintains and builds metabolically active muscle, which becomes especially important with age. Recent systematic-review evidence continues to support beneficial glycemic effects of resistance training in people with type 2 diabetes, although effect sizes depend on population and training design.[20]

A practical example shows both the power and the limits of the evidence. Consider the post-meal walk: lunch, then a 10–20 minute walk, then back to work. Post-meal activity measurably reduces the acute glucose excursion.[21] But an acute glucose effect is not proof of improved long-term clinical outcomes specifically because the walk occurred after rather than before the meal. Keeping that distinction—acute effect versus long-term benefit—is what sound evidence interpretation looks like in practice.

Acute Effect ≠ Long-Term Benefit

"Walking after a meal reduces the postprandial glucose excursion" does not automatically establish "walking specifically after meals prevents cardiovascular disease or increases lifespan." Likewise, "changing food order lowers an acute glucose peak" does not establish "food order is an important determinant of long-term health." Both claims may still be worthwhile habits—but for the right reasons, at the right level of confidence.

7.3 Body Composition and Energy Storage

The amount and location of stored energy matter. Subcutaneous fat under the skin, visceral fat around the internal organs, and ectopic fat inside organs such as the liver have very different metabolic consequences, and adipose tissue is an active metabolic organ—releasing hormones and inflammatory signals—not merely passive storage. Skeletal muscle, meanwhile, is the largest site of postprandial glucose disposal.

A storage-capacity framing ties these together: when chronic energy surplus contributes to increasing visceral and ectopic fat accumulation—particularly in the liver—insulin signalling and metabolic regulation can deteriorate in susceptible individuals. This is why two people with the same BMI and different fat distribution can have very different metabolic health.

Weight loss deserves careful wording. For people with overweight or obesity and elevated type 2 diabetes risk, meaningful weight reduction can substantially improve metabolic health. The landmark Diabetes Prevention Program (DPP) randomised 3,234 high-risk adults to an intensive lifestyle intervention (targeting at least 7% weight loss and at least 150 minutes of weekly physical activity), metformin, or placebo.[22] During the trial, the lifestyle intervention reduced diabetes incidence by 58% relative to placebo and metformin by 31%; in absolute terms, incidence was approximately 4.8 cases per 100 person-years with lifestyle intervention versus 11.0 with placebo (7.8 with metformin). These findings concern a high-risk study population; they do not imply that weight loss is appropriate for everyone.

7.4 Sleep, Stress, and Daily Rhythm

Metabolism does not reset independently at every meal. The clearest demonstration comes from sleep research: in a randomised crossover study, experimental sleep restriction impaired whole-body and adipocyte insulin signalling, with the body requiring more insulin to achieve a similar glucose result.[23] The study is small and mechanistic in purpose—its lesson is the direction of the effect, not a population-wide effect size. Once again, compensation is the mechanism through which the hidden cost appears first.

Stress physiology acts in the same direction. Experimental and epidemiological work links circadian disruption, sleep curtailment, and stress-hormone activation with impaired glucose regulation, though effect sizes and individual vulnerability vary.[24, 25] Cortisol, adrenaline, acute illness, psychological stress, and normal circadian variation can all raise glucose in the absence of any food—which is why glucose can be elevated on a stressful, sleep-deprived morning despite identical meals. Note what this does *not* imply: nothing here licenses rigid rules such as "never eat carbohydrate after 6 p.m." Circadian biology is real; simple clock rules built on it mostly are not.

Figure 5 collects the full set of influences around a single meal. The glucose response is a property of the whole context, not of the food alone.

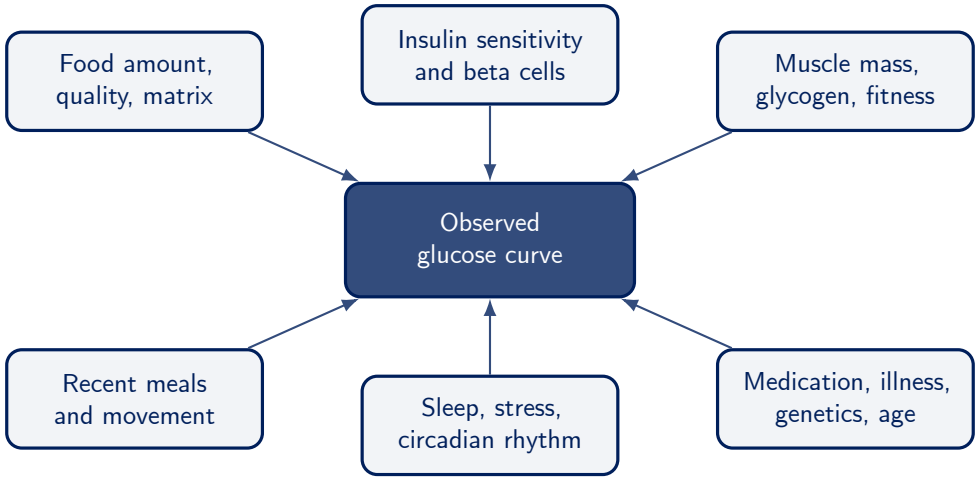

Figure 5: What determines my glucose response? Food matters, but the observed curve is co-produced by insulin sensitivity, beta-cell function, muscle, recent activity, sleep, stress, timing, previous meals, medications, illness, genetics, and age. A glucose curve describes a context, not a verdict on a food.

8 What Actually Matters Most?

With the determinants on the table, the evidence can be turned into a hierarchy. Not everything with a measurable effect deserves equal attention.

8.1 The Big Levers

The strongest general priorities for long-term metabolic health are regular physical activity, cardiorespiratory fitness, maintaining skeletal muscle, avoiding chronic excessive energy intake, improving excess visceral and ectopic adiposity where appropriate, a high-quality fiber-rich diet built on minimally processed foods, adequate sleep, avoiding prolonged physical inactivity, and appropriate evidence-based medical screening where risk warrants it. This is not a claim that every item carries equal evidence for every population and outcome—it is a claim about where the weight of the evidence sits.

8.2 Useful Optimizations

Below the big levers sit genuinely useful refinements: replacing highly refined carbohydrate sources with less processed alternatives, reducing sugar-sweetened beverages, increasing fiber, breaking up prolonged sitting, walking after meals, combining carbohydrate with protein, fat, and fiber appropriately, and adjusting portion sizes. Each can add value. All of them remain subordinate to the major levers.

8.3 The Fine-Tuning Trap

At the bottom of the hierarchy sit contemporary optimisation practices: obsessing over the exact order of foods, minimising every CGM peak, pursuing arbitrary glucose variability scores, testing individual foods repeatedly, and trying to achieve an unnaturally flat CGM trace. Some of these have legitimate uses in specific situations. The problem arises when someone optimises a secondary biomarker while neglecting the major determinants of health. Figure 6 arranges the three levels; the caption is the instruction.

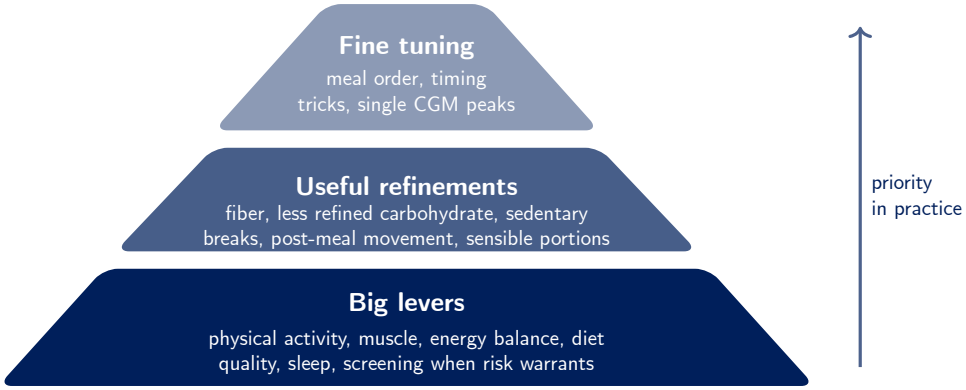

Figure 6: The metabolic health pyramid. The wider base carries the most weight: preserve the major determinants first, use refinements second, and treat CGM-driven fine tuning as optional context rather than the main project.

9 Five Claims Worth Examining

The ideas above can now be turned on the claims that circulate most widely. Five suffice to cover the popular landscape.

9.1 "Insulin Makes You Fat"

Verdict: incomplete and misleading. Insulin promotes nutrient storage and suppresses lipolysis in the fed state—that is normal physiology, and it is transient. Long-term accumulation of body fat cannot be explained simply by whether insulin rises after meals. Protein can also stimulate substantial insulin secretion, illustrating why insulin response alone cannot explain the long-term effect of a food on body weight. Insulin is part of the energy-storage machinery, not the master switch of body fat.

9.2 "Carbohydrates Cause Insulin Resistance"

Verdict: too simplistic. Acute insulin secretion after carbohydrate ingestion and chronic insulin resistance are different phenomena, and the development of resistance tracks energy balance, adiposity, ectopic fat, inactivity, genetics, sleep, and overall dietary quality—not carbohydrate as a category. Carbohydrate sources range from lentils and whole fruit to refined starches and sugar-sweetened beverages; treating all of them as one metabolic exposure is unhelpful at best.

9.3 "Every Glucose Spike Is Harmful"

Verdict: not supported as a general statement. Normal physiology includes postprandial excursions, and healthy people without diabetes show them on CGM.[4, 5] Chronic hyperglycemia is clearly harmful—but that fact should not be extrapolated automatically to every temporary rise in metabolically healthy individuals. What matters is magnitude, duration, recovery, baseline risk, and the surrounding diet and activity pattern.

9.4 "The Flatter the CGM Curve, the Healthier the Food"

Verdict: false as a general rule. The lentils-versus-processed-low-carbohydrate comparison from Section 7 settles the direction: a flatter glucose trace can accompany a meal—or an overall diet—that is inferior on other dimensions of nutritional and cardiovascular health. State it succinctly: CGM measures glucose, not health.

9.5 "A Normal HbA1c Means My Metabolism Is Optimal"

Verdict: not necessarily. HbA1c is valuable, and nobody should rush into elaborate insulin testing on the strength of one normal or borderline value. But remember what HbA1c reflects: glycemic exposure, not insulin requirement. Compensatory hyperinsulinemia can precede overt dysglycemia by years (Figure 3), and HbA1c carries biological and measurement limitations of its own. Normal glucose and optimal metabolism are related but not identical claims.

10 Conclusion: A Better Mental Model

The best way to finish is not with another list of diet rules, but with four concepts that together define metabolic health.

Regulation. Can the body maintain glucose appropriately across meals, fasting, and activity?

Responsiveness. How effectively do tissues respond to insulin, and can the pancreas generate an appropriate insulin response when required? For a comparable metabolic challenge and glucose response, a lower insulin requirement can indicate greater insulin sensitivity. Adequate beta-cell capacity remains essential.

Capacity. Can liver, muscle, adipose tissue, and pancreas handle changing energy demands, such as a big meal, a long hike, or a poor night's sleep, without falling behind?

Resilience. How well does the system adapt to meals, fasting, exercise, sleep loss, illness, and stress, and how quickly does it return to baseline?

Together these four return the paper to its central thesis: good metabolic health is not a perfectly flat glucose curve. It is a robust regulatory system. Blood glucose and insulin are observable parts of that system, not enemies to be minimised at all costs. The objective is not to eliminate glucose or insulin responses; it is to preserve the body's ability to generate the right metabolic response, of the right magnitude, at the right time.

11 Glossary

Key terms in alphabetical order for quick reference.

Beta cells Pancreatic cells that produce insulin; their progressive dysfunction defines the transition to overt type 2 diabetes.

Blood glucose The concentration of glucose in the blood; the system's observable state variable.

Continuous glucose monitoring (CGM) A wearable sensor measuring interstitial glucose at high frequency; a movie of glucose dynamics, currently not an established diagnostic test for prediabetes or diabetes.

Ectopic fat Fat stored inside organs such as the liver or pancreas; excess ectopic fat is associated with metabolic dysfunction and insulin resistance.

Gluconeogenesis The liver's manufacture of new glucose from amino acids, glycerol, and lactate.

Glucagon Pancreatic alpha-cell hormone that raises blood glucose by stimulating hepatic glycogen breakdown and glucose production; insulin's counterpart.

Glycemic excursion The temporary rise and fall of blood glucose after a meal.

Glycemic index (GI) A measure of how quickly a fixed amount of carbohydrate from a food raises blood glucose relative to a reference food.

Glycemic load (GL) The glycemic index scaled by the amount of carbohydrate in the actual serving.

Glycogen The storage form of glucose; muscle glycogen serves the muscle, liver glycogen the whole body.

HbA1c Glycated haemoglobin; an indirect, longer-term measure of average glycemic exposure over the preceding weeks to months.

Hepatic glucose production The liver's release of glucose into the blood, from glycogen breakdown and gluconeogenesis.

HOMA-IR Homeostatic model assessment of insulin resistance; a research index roughly proportional to fasting insulin times fasting glucose.

Hyperinsulinemia Abnormally elevated insulin levels, often compensating for insulin resistance.

Incretins Gut-derived hormones that amplify insulin secretion after oral nutrients.

Insulin Pancreatic beta-cell hormone signalling nutrient availability; promotes glucose uptake, glycogen synthesis, and storage.

Insulin resistance A state in which more insulin is required to produce a given metabolic effect.

Insulin sensitivity How strongly the body responds to a given insulin signal.

Interstitial glucose Glucose in the fluid between cells; what CGM measures, with a lag relative to blood glucose.

OGTT Oral glucose tolerance test; a standardised 75-gram glucose challenge revealing how the system handles a load.

Postprandial Occurring after a meal.

Prediabetes Metabolic state between normal regulation and diabetes: fasting glucose 100–125 mg/dL, HbA1c 5.7–6.4%, or 2-hour OGTT glucose 140–199 mg/dL.

Type 1 diabetes Autoimmune destruction of beta cells leading to severe insulin deficiency.

Type 2 diabetes Progressive loss of adequate beta-cell insulin secretion, frequently against a background of insulin resistance.

Visceral fat Fat stored around internal organs; excess visceral fat is generally more strongly associated with metabolic risk than subcutaneous fat.

12 Disclaimer

This paper is provided for educational and informational purposes only. It does not constitute medical advice, diagnosis, or treatment, and it is not a substitute for individual consultation with a licensed physician or other qualified health professional. Interpretation of glucose, insulin, HbA1c, or CGM data—and any decision about screening, medication, or treatment—belongs in a clinical context, particularly for readers with diagnosed diabetes, prediabetes, cardiovascular, kidney, or other chronic conditions.

The evidence summarised here comes from studies with specific populations, protocols, and endpoints; reported associations do not guarantee individual outcomes. Reference ranges and

diagnostic criteria evolve, consumer glucose devices measure interstitial rather than blood glucose, and no number in this paper—glucose, insulin, or variability metric—should be interpreted as a definitive metabolic-health score without professional context.

If you experience red-flag symptoms such as excessive thirst and urination, unexplained weight loss, blurred vision, severe fatigue, or fruity-smelling breath, seek medical care promptly.

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