Restoring Metabolic Health

Ketosis as a Tool: Personalised, Measurable, Sustainable

Dorian Greenow | SwissRe | February 2026

Disclaimer, Conflict of Interest / Bias

Nutritional Ketosis since 2015

Founder and CEO/CFO Keto-Mojo

President and Co-Founder of the Ketogenic Foundation

Advisory Board Member, Keto Live Center, Non-Profit

Advisory Committee Member, Metabolic Terrain Institute of Health, Non-Profit

But first the ABC's…

***A**cknowledge bias*

***B**e curious*

***C**onscientious research*

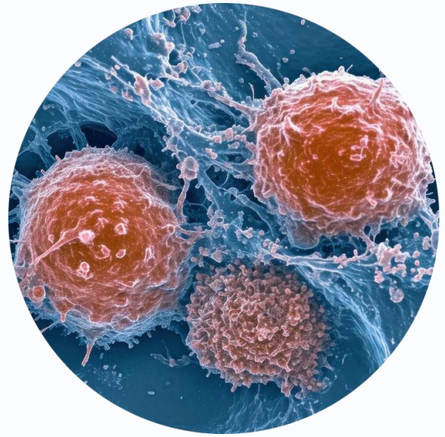
Only 7% of Americans Are Metabolically Healthy

Metabolic dysfunction is the root cause of multiple non-communicable diseases

Metabolic Dysfunction Drives Chronic Disease

Type 2 Diabetes

Cardiovascular Disease



Certain Cancers

Cognitive Decline

Fatty Liver Disease

The Common Thread: Insulin Resistance

- 208 healthy, non-overweight adults (avg. age 50)
- Grouped by insulin sensitivity; matched for BMI, age, smoking
- Followed for 6 years

- Zero hospitalisations in the insulin-sensitive group
- 1 in 3 hospitalised in the insulin-resistant group
- Insulin resistance outpredicted age, smoking, and exercise for chronic disease risk

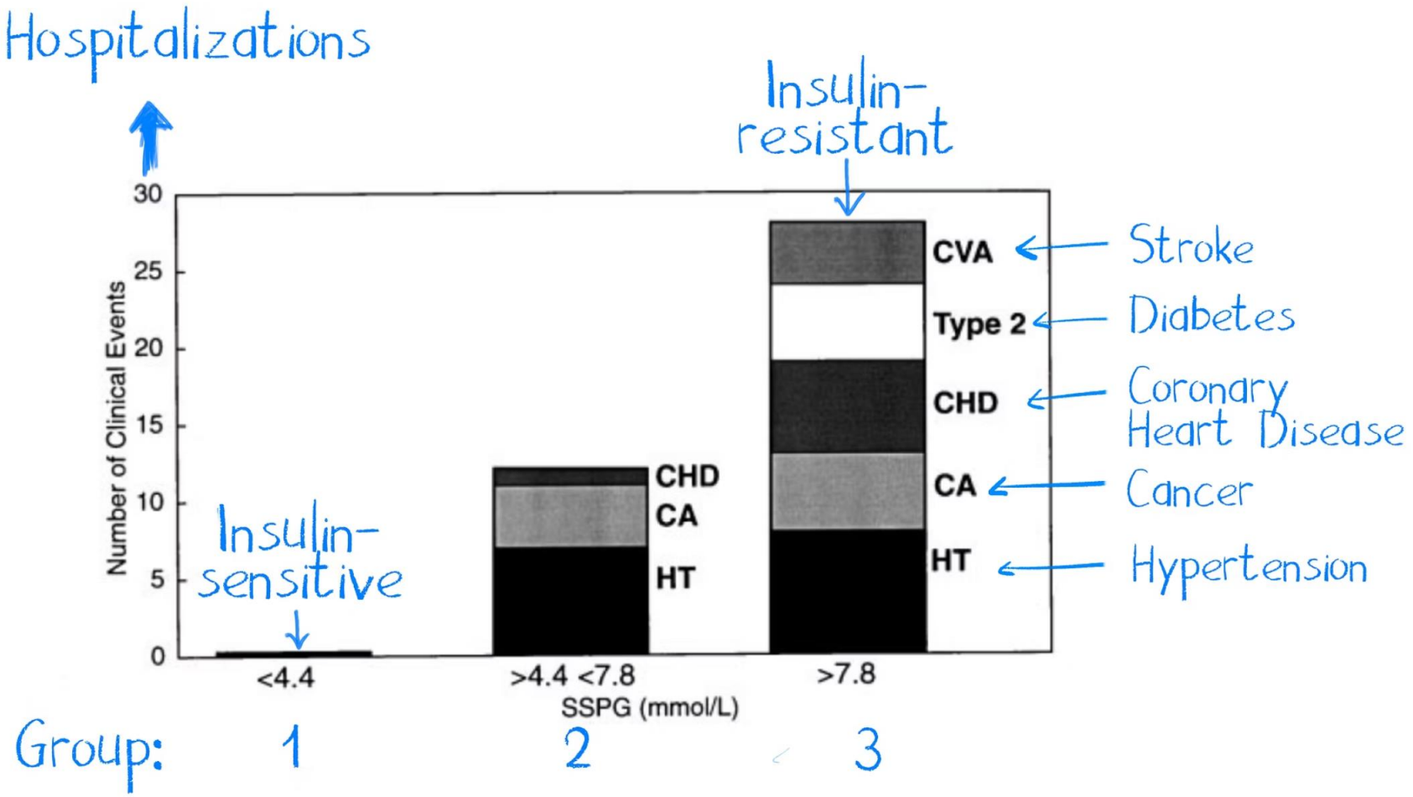


FIG. 1. The number of clinical events observed, as a function of insulin resistance tertile at baseline. CA, Cancer; Type 2, type 2 diabetes. These were 28 events in the highest tertile (SSPG > 7.8 mM), 12 in the intermediate tertile (SSPG > 4.4 < 7.8 mM), and none in the most insulin-sensitive tertile (SSPG < 4.4 mM).

Image from https://doi.org/10.1210/jcem.86.8.7763 , F. S. Facchini et al: Insulin Resistance as a Predictor of Age-Related Diseases.

Insulin Resistance Comes Silently

Hyperinsulinemia precedes loss of glucose control—by the time blood sugar rises, the problem has been developing for years.

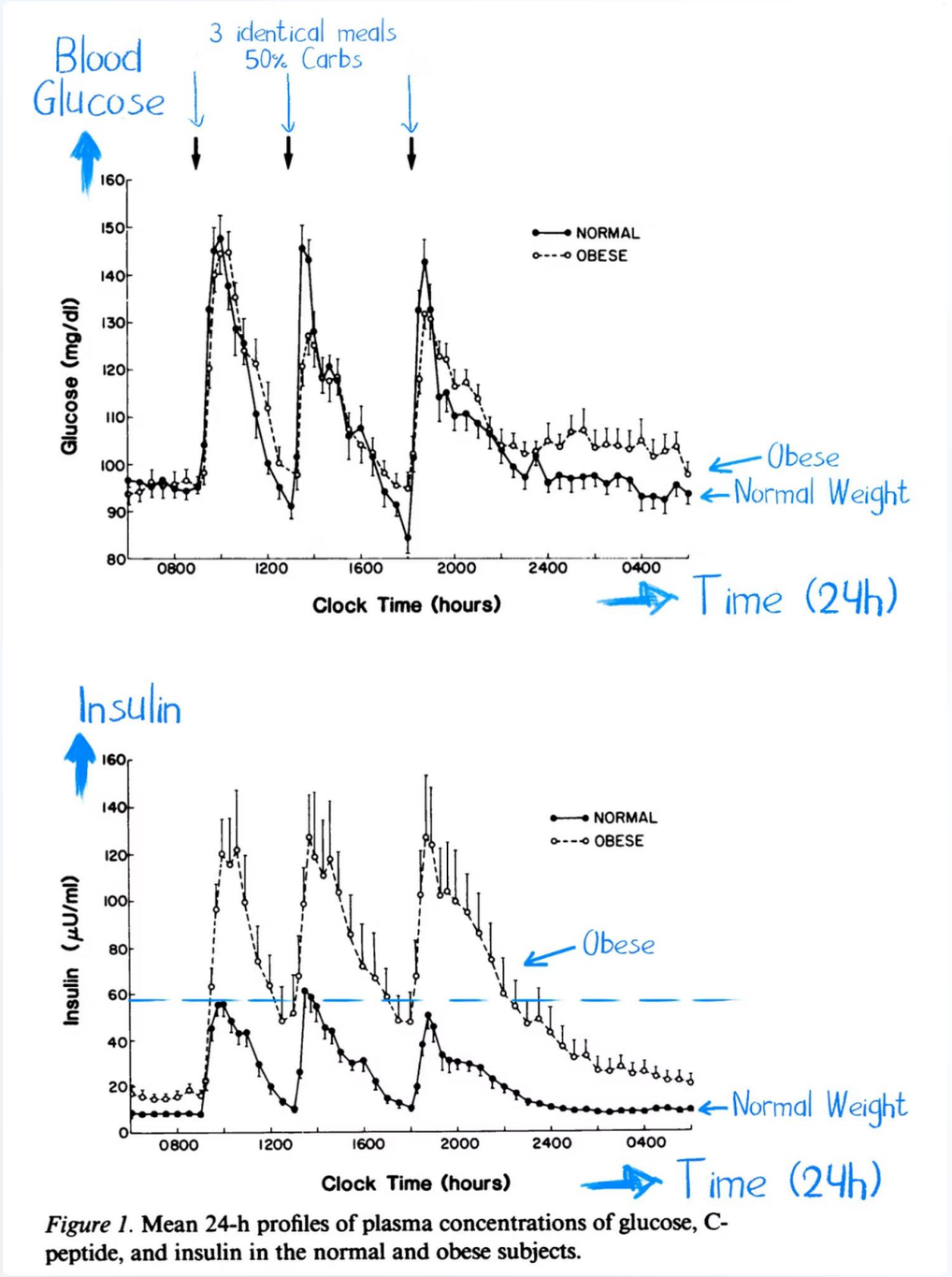


Figure 1. Mean 24-h profiles of plasma concentrations of glucose, C-peptide, and insulin in the normal and obese subjects.

Image from https://doi.org/10.1172/JCI113339, K. S. Polonski et at.: Twenty-four-hour profiles and pulsatile patterns of insulin secretion in normal and obese subjects.

A Self-Reinforcing Problem

The Lever: Carbohydrate Intake

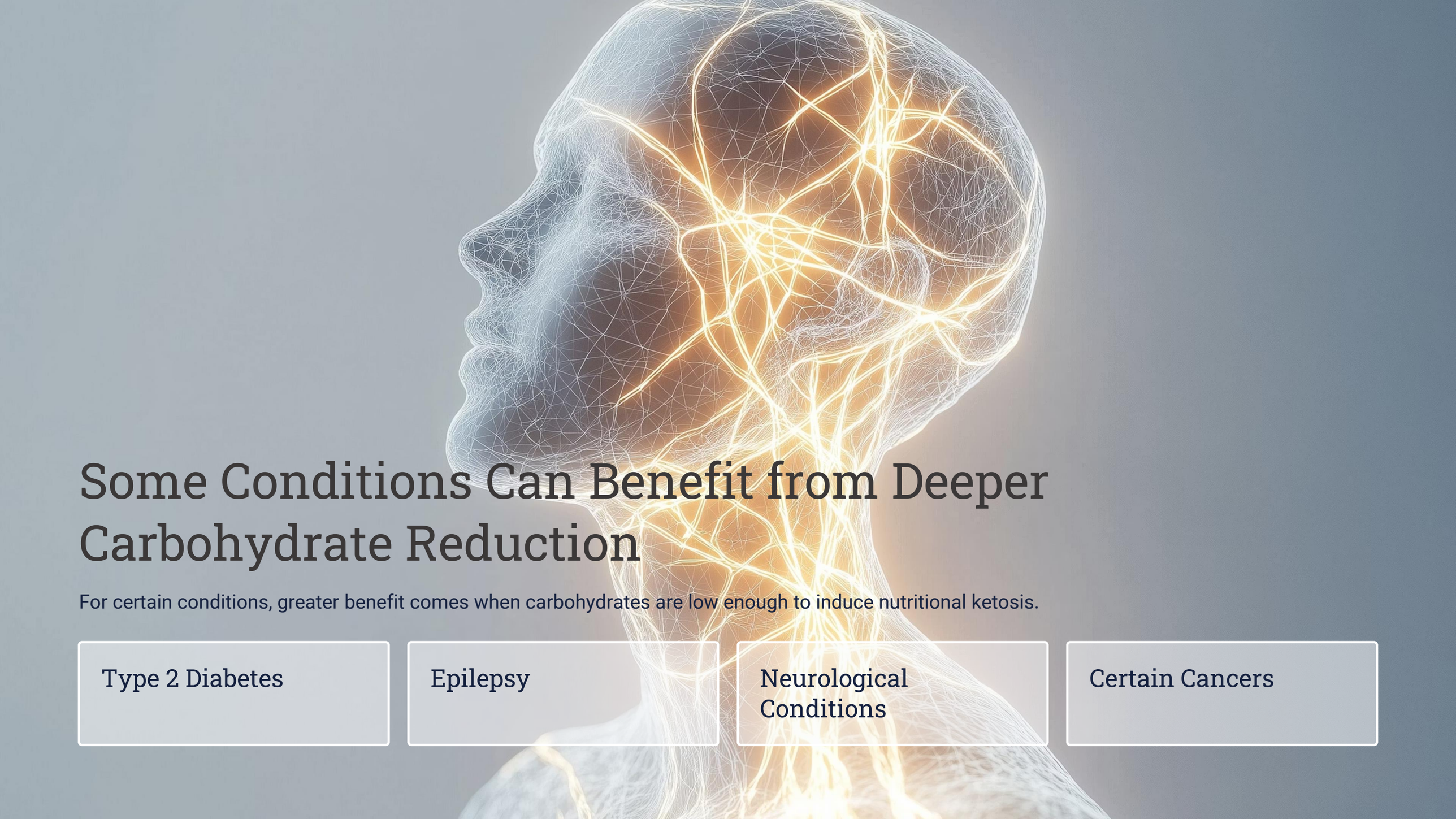
Carbohydrates drive insulin more than any other macronutrient. Reducing carbohydrate intake lowers insulin demand, allowing the cycle to reverse.

Small Changes, Real Impact

Even modest carbohydrate reduction improves insulin sensitivity. People don't need to adopt the most restrictive approach to benefit—any movement along the spectrum helps.

Personalization Matters

People have different levels of carbohydrate tolerance based on their metabolic state, genetics, activity level, and health history. What works for one person may not work for another.



Some Conditions Can Benefit from Deeper Carbohydrate Reduction

For certain conditions, greater benefit comes when carbohydrates are low enough to induce nutritional ketosis.

- Type 2 Diabetes
- Epilepsy
- Neurological Conditions
- Certain Cancers

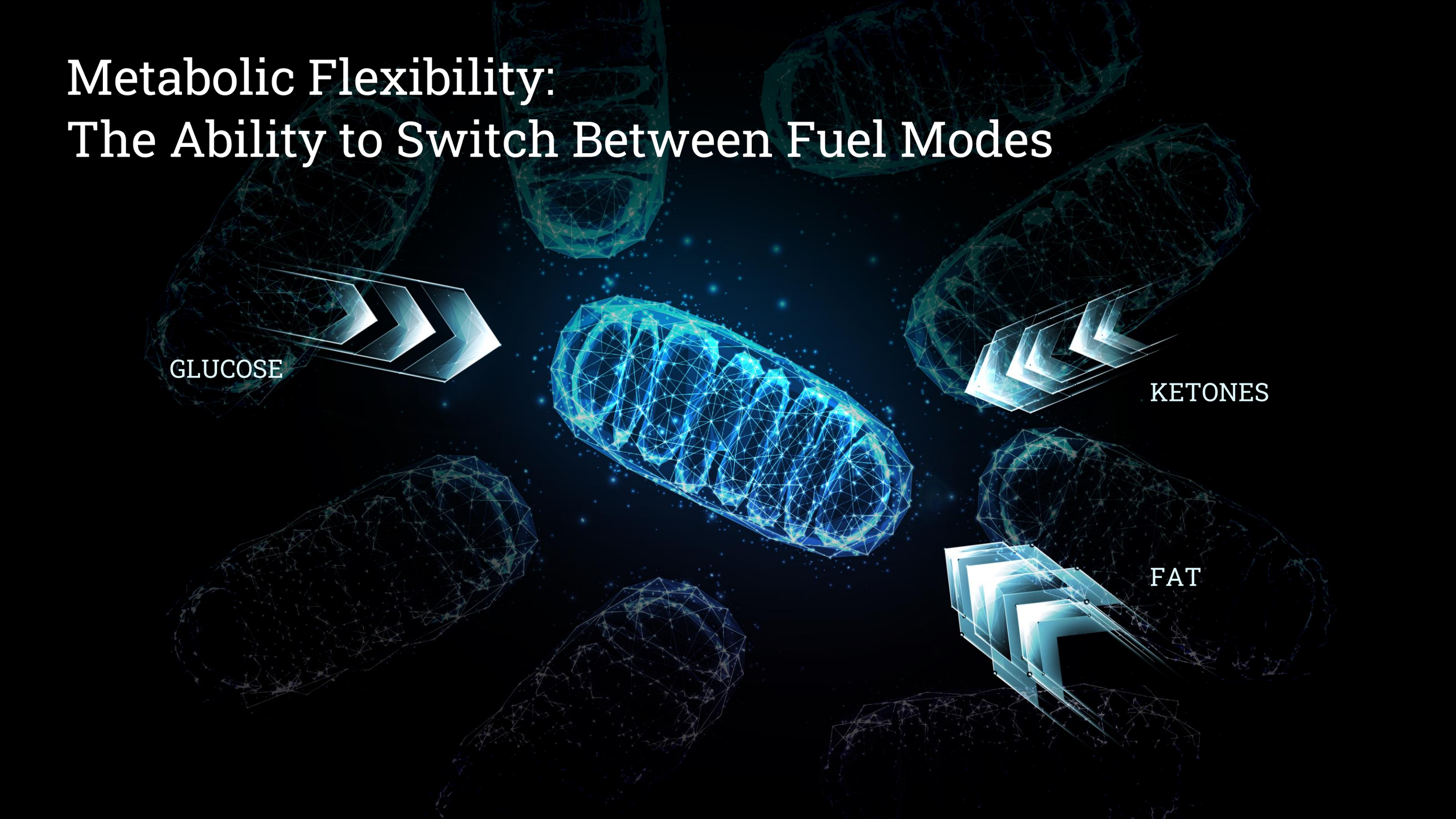
The Metabolic Switch

Nutritional ketosis occurs either through lower carbohydrate intake, exercise or fasting.

The body shifts to burning fat and producing ketones as an alternative fuel source.

Typically achieved below ~50g carbs/day, though individual thresholds vary.

Metabolic Flexibility:
The Ability to Switch Between Fuel Modes



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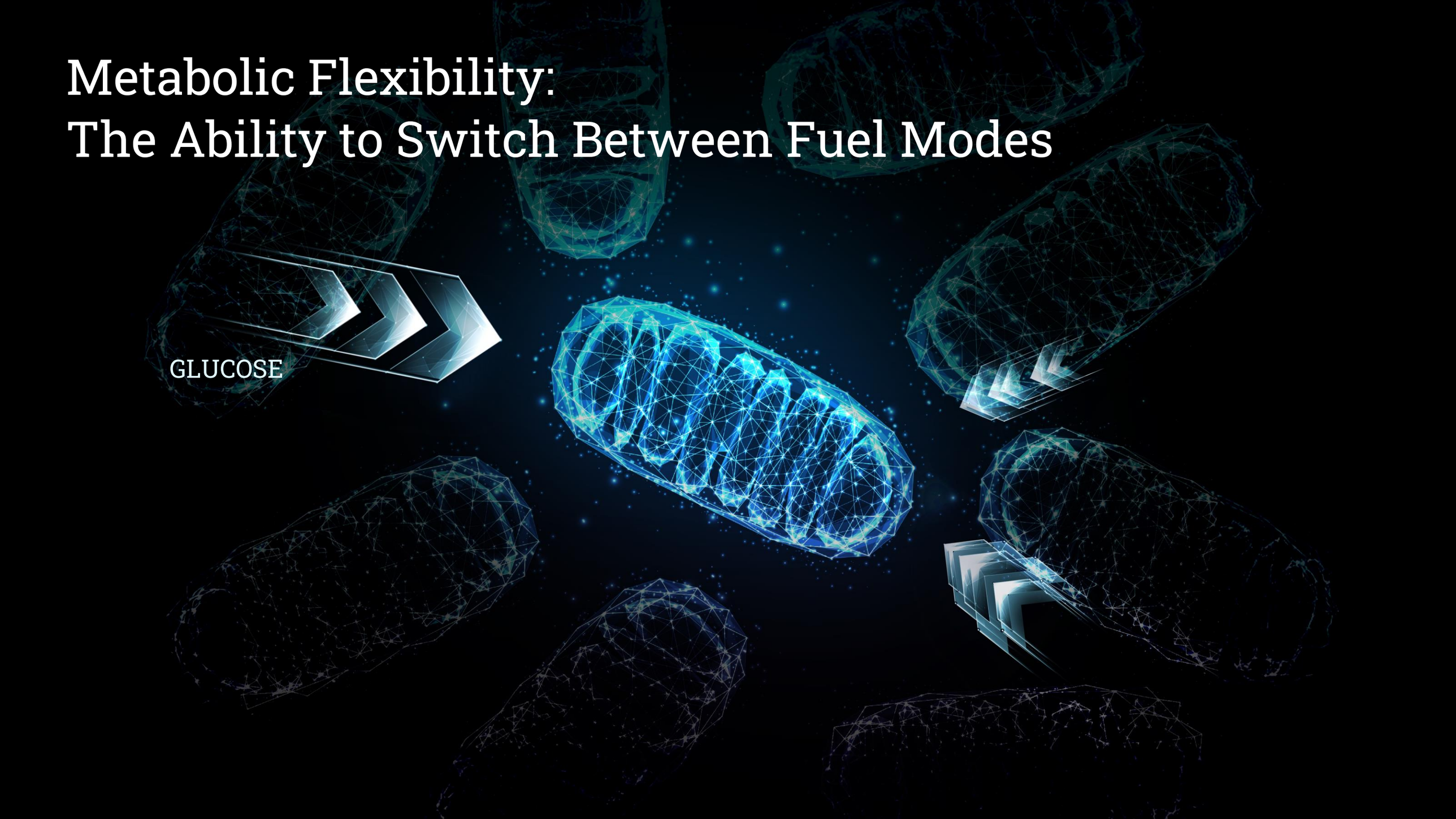

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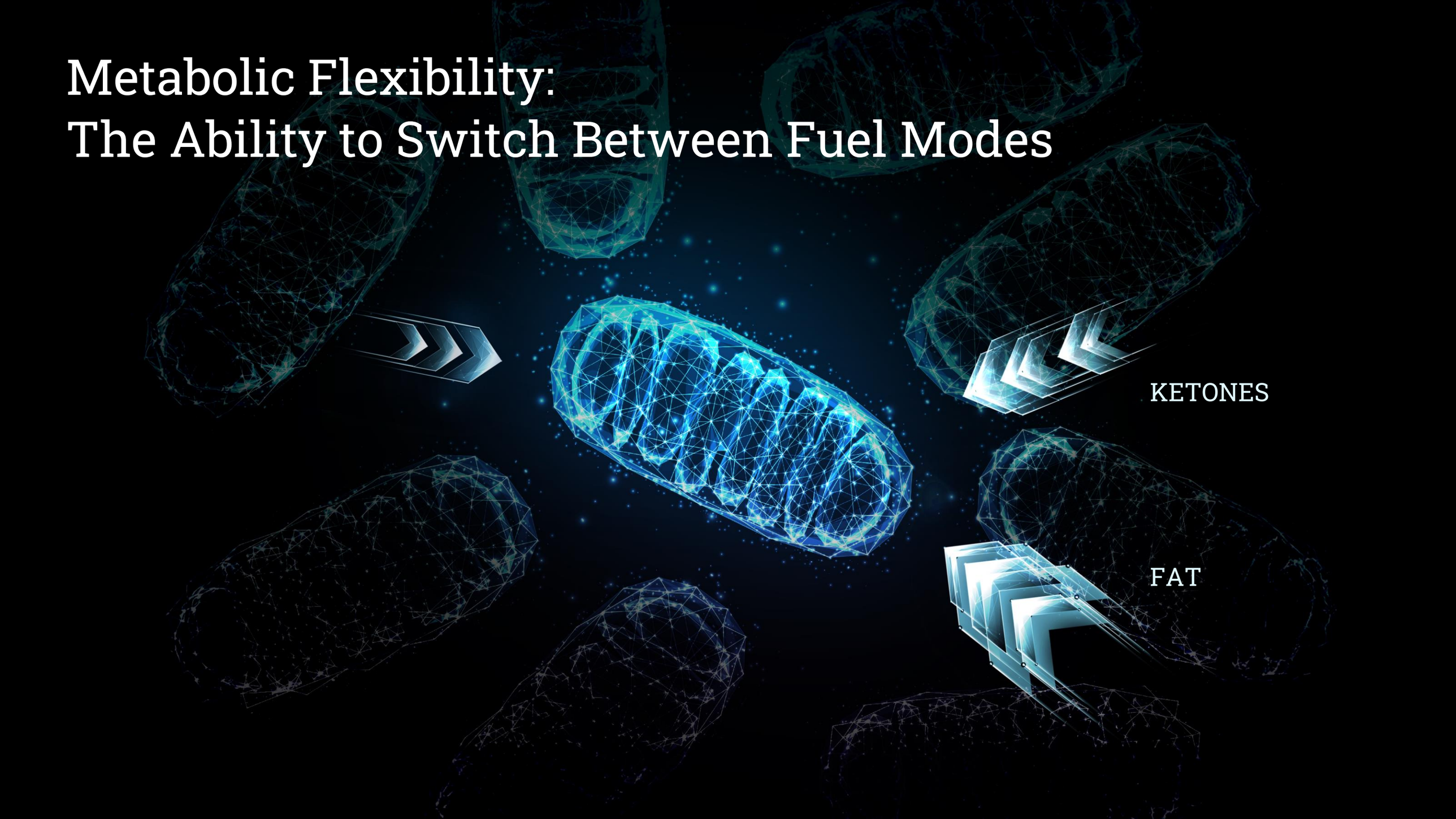

Three Main Fuels: Glucose, Fat, and Ketones

→ Metabolic flexibility means the body can switch smoothly between fuel modes.

→ A simple test: after an overnight fast, a flexible metabolism will produce ketones. If it doesn't, the body is stuck in carb-burning mode.

Ketosis: Our Metabolic Starting Point

Newborns are in ketosis—it's our first metabolic state.

Ketones are critical for early brain development.

This isn't an extreme intervention; it's a return to a natural physiological state.

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Ketone Bodies in the Fetus and Newborn During Gestational Diabetes and Normal Delivery

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ABSTRACT

Background: Authors successfully treated gestational diabetes by a very low carbohydrate diet without insulin and other drugs. Increased ketone bodies seemed to play an essential role in energy metabolism, and the fetus and newborn also showed hyperketosis. It is necessary to clarify how much ketone bodies were present in the placenta and umbilical cord in the fetus and newborn and the pregnant mother with or without gestational diabetes.

Subjects and Methods: All cases were patients of Muneta OB/GYN Clinic in Chiba, where about 700 deliveries were done every year, 90% normal and 10% gestational diabetic. Blood of 313 mothers and babies at health check-up postpartum, 192 samples of placenta and cord blood at the delivery, and 122 cases were obtained at the time of miscarriage. Abbott's kit measured βHB, and 101 samples obtained at the post partem health check-up were biochemically analyzed for both βHB and glucose. The IBM-SPSS did the statistical analysis.

Results: βHB in Mothers' and newborns' blood at four days postpartum was 0.062 and 0.244 mmole/L (median), respectively, and glucose was 4.55±0.81 mmole/L. βHB was high throughout the pregnancy; In the placenta, βHB in the first-, second-and-third trimester was 1.95±0.9 mmole/L, 2.82±0.49 mmole/L, 1.87±0,65 mM/L, respectively. In the cord blood, it was 2.3±1.13 mmole/L, 1.36±0.76 mmole/L, and 0.69±0.6 mmole/L, respectively. Placental βHB at the delivery was 1.99±0.78 mmole/L, and that of the umbilical cord was 0.75±0.36 mmole/L. In the first trimester miscarriage, βHB in spontaneous abortion was 1.84±0.85 mmole/L, while it was 2.09±0.94 mmole/L in artificial abortion. Aborted cases in the second trimester showed 1.96±0.38 mmole/L βHB and 3.74±0.75 mmole/L glucose in the cerebrospinal fluid.

Discussion: Our data showed βHB and glucose concentration in the human fetus and newborn under the normal physiological condition. βHB was present in the placenta and umbilical cord blood throughout fetal life and after birth. Different concentrations between the placenta and umbilical cord blood suggested the fetus's uptake for energy and intrauterine growth. High βHB in the cerebrospinal fluid suggested the effects on neuronal development.

Keywords: 3-hydroxybutyric acid, Ketone bodies fetus, Placenta, Umbilical cord, Cerebrospinal fluid, Miscarriage, Gestational diabetes

Abbreviations: βHB: 3-hydroxybutyric acid

The Decline of Metabolic Flexibility

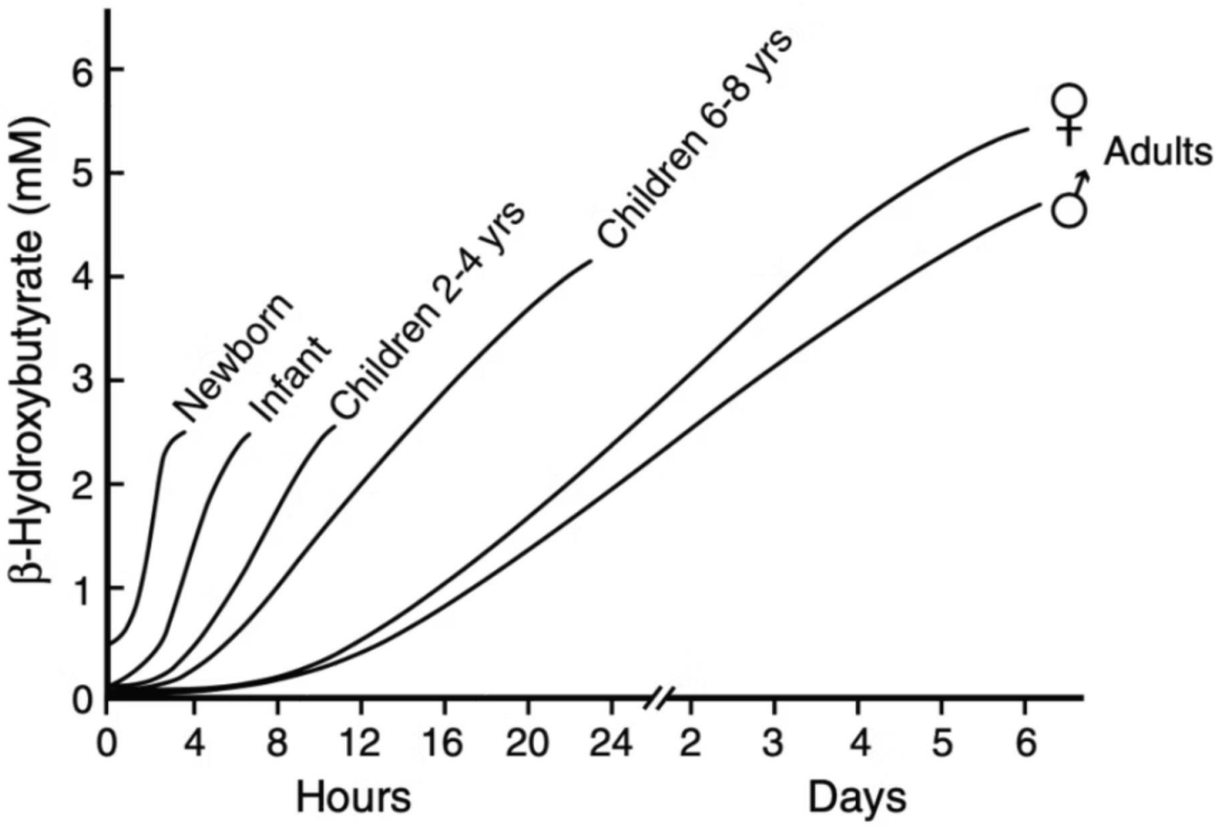
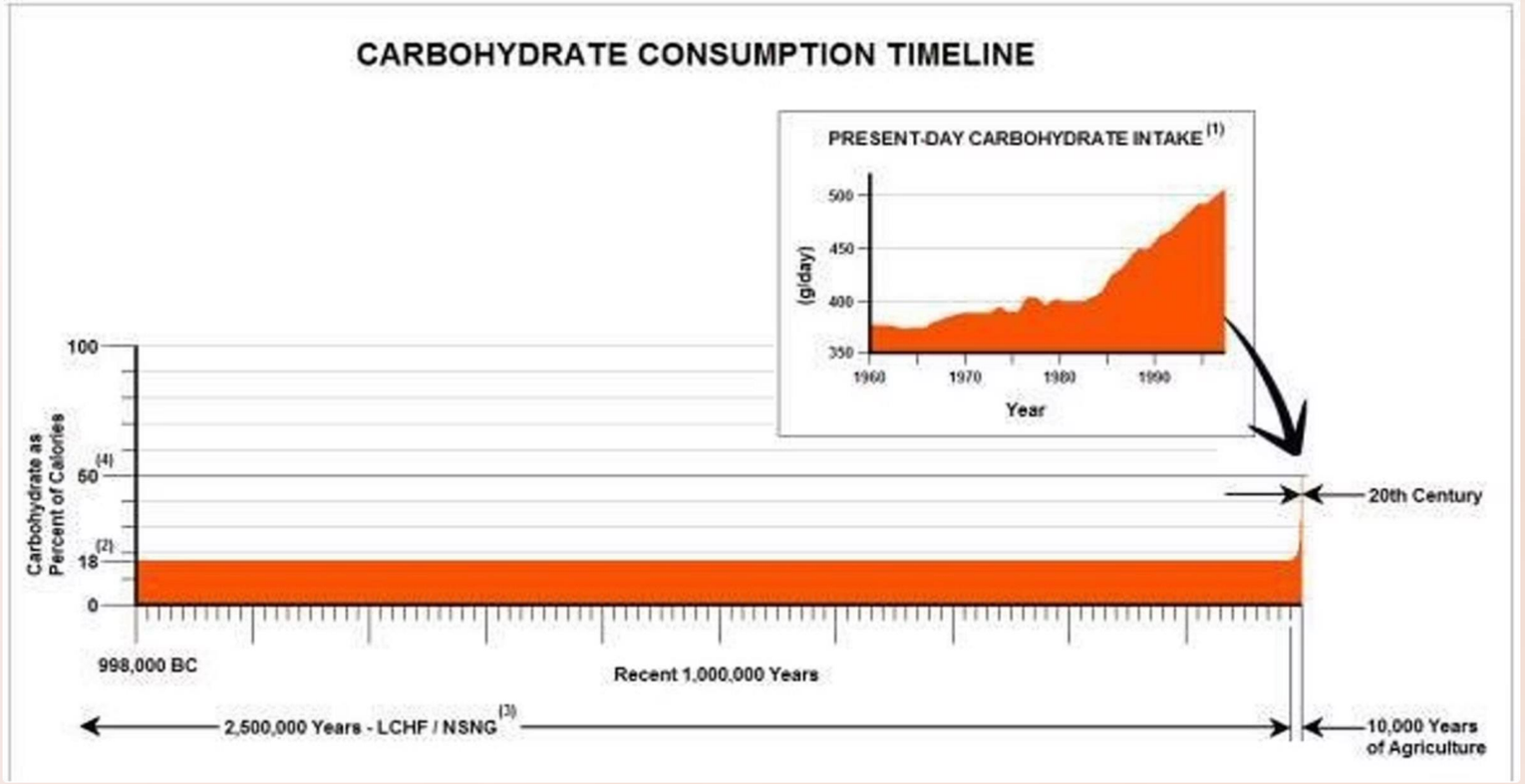


Figure 6 Levels of β-hydroxybutyrate in starving subjects of different ages (5, 13, 30, 54, 59, 66). Not shown is the accelerated ketosis in fasting pregnant or lactating women or in any subject with marked renal glucosuria requiring increased gluconeogenesis, e.g., when the renal threshold is surpassed, as in type 1 diabetes (40), or with genetic renal glucosuria or chemical inhibition of tubular reabsorption of glucose (phlorizin administration).

Image from https://doi.org/10.1146/annurev.nutr.26.061505.111258 , George F. Cahill, Jr.: Fuel Metabolism in Starvation.

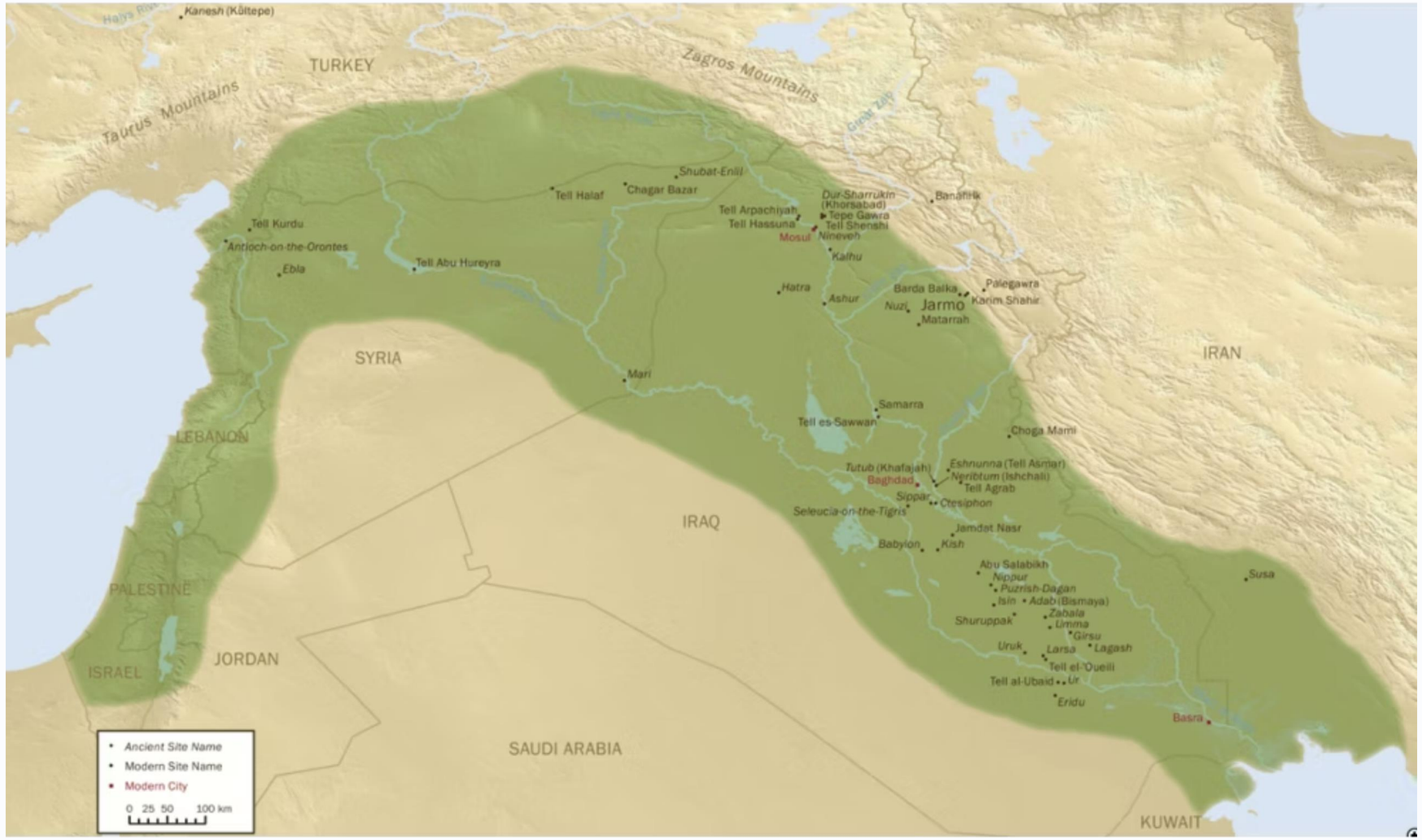
Built for Carbohydrate Scarcity, Living in Abundance

For most of human history, carbohydrates were scarce and seasonal. Our bodies evolved to switch between glucose and ketones depending on availability.



The Globalisation of Carbohydrates Over the Last 450 Years

Cereal Production & the Cradle of Civilization

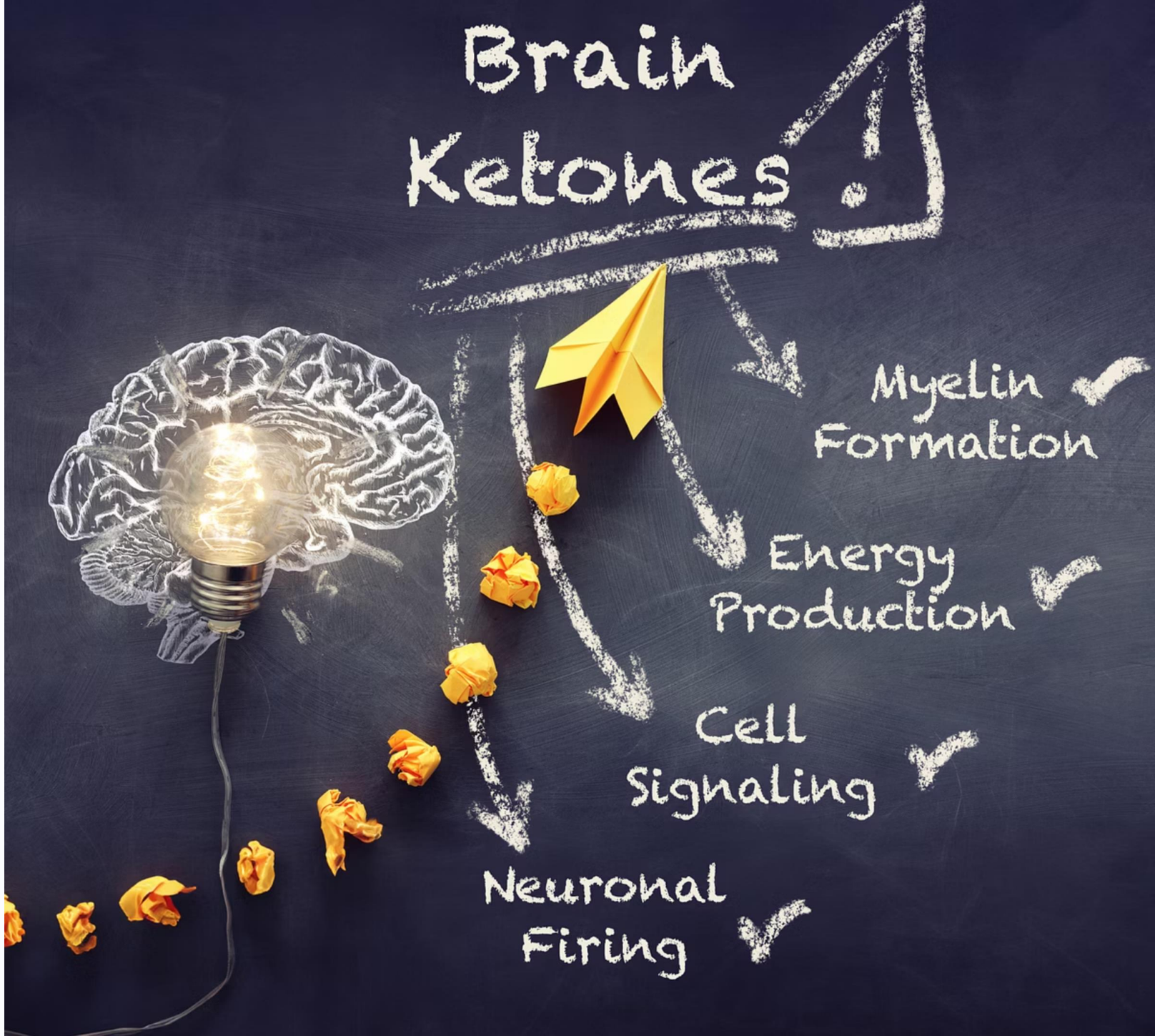


This Is Your Brain on Sugar

Not a Lack of Willpower—A Hijacked Brain

Ketosis: Unique Advantage for the brain

Ketones play a key role in optimal brain function. The brain not only actively transports ketones across the blood-brain barrier—it also produces them locally in astrocytes.



N. Auestad, R. A. Korsak, J. W. Morrow, J. Edmond: Fatty Acid Oxidation and Ketogenesis by Astrocytes in Primary Culture, Journal of Neurochemistry, https://doi.org/10.1111/j.1471-4159.1991.tb11435.x

We can help by changing our messaging and food environment toward real food and better choices!

The Ketogenic Metabolic Health Triangle

How Can We Utilise Today's Technology?

We have the technology to move beyond one-size-fits-all dietary advice. Biomarker feedback allows individuals to see how their bodies respond—enabling personalisation and improving adherence.

Keto Is Uniquely Measurable

Ketogenic diets are the only dietary approach with a built-in biomarker. You can objectively test whether someone is in the metabolic state—no guessing, no relying on food diaries alone.

Personalisation Through Measurement

With objective data, interventions can be calibrated to the individual. Some people need strict carbohydrate reduction; others see benefits with modest changes. Testing reveals the minimum effective dose.

01

Collect Objective Data

Measure individual biomarker responses

02

Calibrate Intervention

Determine minimum effective dose

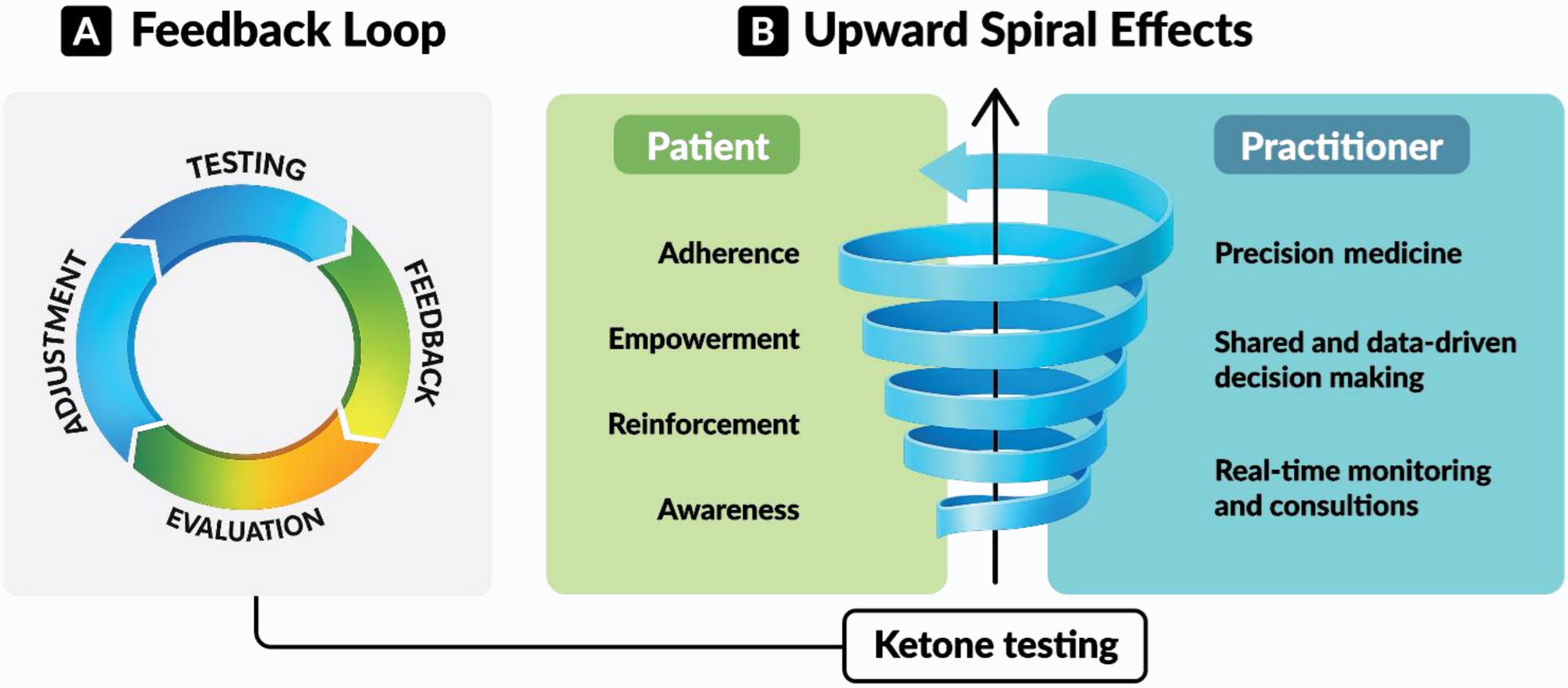
03

Personalise Approach

Tailor carbohydrate levels to individual needs

The Key to Long-Term Success

When people see their own data and understand their individual threshold, they're more likely to sustain the change. It's no longer an arbitrary prescription—it's a personalised approach based on their biology.



C: Fante, F. Spritzler, L. Calabrese, N. Laurent, C. Roberts, S. Deloudi: The role of β-hydroxybutyrate testing in ketogenic metabolic therapies. Front. Nutr. 2025;12:1629921

Metabolic Disease Is Addressable, Not Inevitable

Metabolic dysfunction is not a foregone conclusion of modern life. With the right tools—personalised carbohydrate management, biomarker feedback, and metabolic flexibility assessment—we can intervene earlier and more effectively.

Thank You

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