Human Fat-Based Metabolism

人类脂供能系统

Canonical PDF archive
alitaos.com remains the canonical web definition source. GitHub Markdown is the versioned public archive.

Fat-Based Metabolism / Human FBM

Contents

1. Abstract

Human Fat-Based Metabolism (Human FBM) is the formal human branch of Fat-Based Metabolism. It defines a long-term metabolic operating structure, not a diet identity, short-term weight endpoint, or ketone target.

The current canonical web source is a 7-chapter, 37-leaf structure covering definition, failed high-carbohydrate frames, species boundaries, energy regulation, feeding and adaptation, observable-output backtrace, and clinical/claim boundaries.

Human FBM is not ketogenic-diet identity, not a weight-loss method, not a high-fat label, and not medical care or a diagnostic system.

2. Canonical Source

- Human FBM canonical source: https://alitaos.com/en/fat-based-metabolism/human
- Public GitHub archive: https://github.com/dujf921/fat-based-metabolism

alitaos.com remains the canonical web definition source. The public repository stores versioned Markdown source documents.

3. Core Definition

Human Fat-Based Metabolism is a long-term metabolic operating structure in which low exogenous carbohydrate input, reduced long-term insulin occupancy, sufficient total energy, and stable lean mass allow fatty acids to become the dominant direct energy substrate instead of repeated carbohydrate scheduling.

Base causal chain:

> lower exogenous carbohydrate input → reduced long-term insulin occupancy → rising fatty-acid direct energy → energy sufficiency → stable lean mass → long-term metabolic steady state

Validity is judged by structural variables and boundary conditions, not by ketone level, short-term weight change, or diet labels.

4. Why Human FBM Is Not Ketogenic Diet

Human FBM is not defined by ketogenic-diet identity. Ketogenic framing often centers carbohydrate restriction and ketone production. Human FBM centers long-term substrate structure under low insulin occupancy and energy sufficiency.

Ketones may appear, but ketone level is not a primary validity variable or success proof. High ketone level does not automatically indicate a better metabolic structure.

5. Why Human FBM Is Not a Weight-Loss Method

Human FBM does not use weight loss as its primary target. Weight change must be decomposed into body fat F, lean mass L, water, and gastrointestinal content before structural interpretation.

Weight decline driven by low energy should not be interpreted as Human FBM success. Valid structure requires both energy sufficiency and lean-mass stability.

6. Food Structure, Not Diet Label

Human FBM evaluates food structure, not ingredient names or marketing labels. The same label can carry very different exogenous carbohydrate input, insulin occupancy, total energy, and lean-mass trends.

Structural judgment must return to control variables to explain output differences under the same label.

7. Why High-Carbohydrate Frames Fail

The problem with high-carbohydrate default frames is not that carbohydrate presence is inherently toxic. The problem is that repeated exogenous carbohydrate input maintains insulin occupancy and carbohydrate scheduling as the long-term axis, compressing long-term fatty-acid direct-energy capacity.

These frames often reduce post-meal volatility, hunger rhythm, and body-fat scheduling to willpower or calorie arithmetic, while missing substrate architecture and occupancy state.

8. Exogenous Carbohydrate Input and Insulin Occupancy

Exogenous carbohydrate input is carbohydrate burden introduced by external food intake, excluding endogenous glucose production. Repeated input shapes long-term insulin occupancy.

Insulin occupancy describes how strongly insulin occupies long-term energy allocation. Under higher occupancy, carbohydrate scheduling dominates; under reduced stable occupancy, fatty-acid direct energy can carry greater long-term responsibility.

9. Fatty Acids as Primary Energy Substrate

Primary energy substrate is the substrate class carrying dominant direct energy duty in long-term operation. Human FBM targets transition from repeated carbohydrate scheduling toward fatty-acid dominance.

Fatty acids as primary energy substrate require low insulin occupancy, energy sufficiency, and stable lean mass at the same time. This is not a high-fat-diet label. High fat percentage with high occupancy, insufficient energy, or lean-mass decline does not establish stable structure.

10. Body Fat F, Lean Mass L, Energy Intake I, and Energy Expenditure E

- Body fat F: stored energy pool and fatty-acid availability buffer.
- Lean mass L: metabolic machinery and structural tissue that must remain stable.
- Energy intake I: total energy entering the system.
- Energy expenditure E: maintenance and output cost.

Weight change must first be decomposed into F, L, water, and gastrointestinal content shifts. A single weight number cannot replace structural judgment.

11. Leptin and Energy Feedback

Leptin feedback helps evaluate whether energy sufficiency and body-fat scheduling remain aligned. In Human FBM, consistency between feedback rhythm and input structure, insulin occupancy, and total energy matters more than a single appetite reading.

Loss of appetite does not automatically indicate steady-state establishment; appetite dysregulation may also reflect stress output.

12. Feeding, Digestion, and Adaptation

Fat digestion, bile-salt dispersion, pancreatic processing, and small-intestine absorption jointly determine whether fatty acids can enter a stable fueling pathway. Fat and protein together raise post-meal satiety and change gastric-emptying rhythm.

Switching from high-carbohydrate scheduling to low-insulin fatty-acid fueling requires re-matching substrate mobilization, enzyme systems, appetite signals, and electrolyte state. Adaptation is a transition phase, not long-term steady state and not structural failure.

13. Electrolyte and Water Boundary

After lowering exogenous carbohydrate input, glycogen reserves and water-binding state change. Water status is coupled to sodium, potassium, and magnesium scheduling and cannot be judged outside energy architecture and adaptation phase.

Water issues cannot be resolved by “drink more water” alone; total energy and electrolyte variables must be judged together.

14. Adaptation Output and Stress Output

Adaptation output is short-term volatility during scheduling-structure switching. Stress output arises from insufficient total energy, insufficient protein, excessive training load, electrolyte insufficiency, or clinical boundary.

The key judgment is not whether discomfort exists, but whether discomfort declines as structural conditions are restored. Not all volatility should be classified as adaptation, and stress output should not be misread as structural failure.

15. Observable Output Index

Chapter 6 is a backtrace index from observable output to variables to system state. It provides no execution protocol and no medical-care advice.

- Body-fat change: weight must be decomposed; low-energy weight decline is not structural success.
- Hunger and satiety: separate stable fatty-acid fueling from low-energy stress suppression.
- Post-meal sleepiness: no single-cause attribution; return to multi-variable backtrace.
- Energy stability: numbness from energy insufficiency is not energy stability.
- Performance boundary: high-intensity explosive demand relates to glycolytic need; competitive contexts enter specialized boundaries.

16. Clinical Boundary

Human FBM is structural nutrition language, not a diagnostic system, cannot replace medical supervision, and enters clinical boundary when disease, acute symptoms, severe metabolic disturbance, or medically supervised states appear. Structural backtrace should stop in those contexts.

This framework provides no diagnostic conclusion, no medical-care promise, and no outcome guarantee.

17. Medication Boundary

Medications can alter glucose, insulin, appetite, water, electrolytes, weight, and energy outputs. With glucose-lowering drugs, insulin, diuretics, or hormone medications, ordinary Human FBM interpretation cannot be applied directly.

Medically supervised medication contexts cannot be overridden by diet architecture alone. This remains structural nutrition language, not a diagnostic system, and cannot replace medical supervision.

18. Pregnancy, Eating-Disorder, and Underweight Boundary

Pregnancy, lactation, eating-disorder risk, and severe underweight states are not suitable for direct guidance from ordinary Human FBM pages. Control variables include development needs, endocrine context, behavioral risk, and total-energy safety monitoring.

This boundary defines stop conditions only and provides no execution advice. Structural backtrace should stop and the case enters clinical boundary.

19. Extreme Athletic Load Boundary

Extreme endurance, strength, or explosive training significantly changes energy demand and glycogen demand. Human FBM can serve as baseline metabolic-structure language but cannot replace specialized sport-nutrition design.

When competitive output is prioritized, judgment enters specialized sport-nutrition boundary. A single generic fatty-acid framing cannot cover all athletic populations.

20. Claim Boundary

Human FBM may state structural causality and may not state disease-management outcomes or universal population applicability.

Permitted: lower exogenous carbohydrate input may reduce insulin occupancy and raise fatty-acid fueling contribution.

Not permitted: disease-management outcomes, unbounded population applicability, or deterministic efficacy promises.

21. Canonical Links

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