

# Mobilize, Capture, Do Not Spill

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## A fasted interval-primed low-intensity aerobic protocol for targeting visceral and ectopic lipid flux in insulin-resistant phenotypes

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**Core claim /** Do not optimize lipolysis alone. Optimize the temporal coupling between fatty-acid release and skeletal-muscle oxidative disposal.

**Keywords:** type 2 diabetes; metabolic syndrome; visceral adipose tissue; ectopic fat; FatMax; fasted exercise; lipolysis; insulin resistance; fatty acid oxidation; exercise prescription

## ABSTRACT

Type 2 diabetes mellitus (T2D), metabolic syndrome, and visceral and ectopic obesity remain unresolved targets in cardiometabolic medicine. Although aerobic exercise improves insulin sensitivity and can reduce visceral adipose tissue and intrahepatic lipid, response magnitude is heterogeneous [3]. This heterogeneity suggests that the conventional instruction to “do more cardio” may be too low-resolution for insulin-resistant phenotypes in which adipose tissue buffering, fatty-acid spillover, and skeletal-muscle oxidative capacity are misaligned.

This manuscript proposes a mechanistic exercise-nutrition protocol organized around a simple but under-specified principle: fat loss is not merely the liberation of fatty acids from adipocytes; it is the successful capture and oxidation of those fatty acids before they are re-esterified or redistributed into ectopic lipid pools. The proposed sequence combines an overnight-fasted state, a short high-intensity interval primer, a prolonged very-low-intensity FatMax-oriented aerobic block, and a post-exercise 1-2 h low-insulin window without carbohydrate or rapidly absorbed protein.

The protocol is framed as a testable hypothesis rather than an established clinical recommendation. Its strongest evidence base is modular: exercise can reduce visceral and perhaps hepatic fat in T2D; fasted aerobic exercise increases acute fat oxidation relative to fed exercise; carbohydrate ingestion can suppress lipolysis and fat oxidation during

exercise; FatMax can identify an individualized intensity for maximal fat oxidation; and post-exercise protein ingestion increases muscle protein synthesis [7]. The unproven claim is the superiority of this exact temporal sequence for reducing visceral and ectopic lipid burden. We therefore propose an 8-12 week randomized pilot trial to test whether a mobilize-capture-do-not-spill strategy outperforms duration-matched conventional aerobic exercise, interval-only exercise, and low-intensity exercise alone.

**Manuscript posture** / This is written as a hypothesis-generating protocol paper: mechanistically aggressive, empirically disciplined, and explicitly falsifiable.

## 1. INTRODUCTION

Type 2 diabetes mellitus, metabolic syndrome, and visceral and ectopic obesity remain urgent problems in cardiometabolic health; new, more precise, and more mechanistically targeted exercise methods are needed. A central limitation of the current exercise prescription paradigm is that it often treats “cardio” as a homogeneous dose of movement rather than as a time-structured metabolic intervention.

A more precise starting point is adipose tissue expandability [2]. The inability of adipose tissue to safely expand in response to chronic or repeatedly acute positive energy flux may represent a candidate upstream sub-regressor of the T2D-prone cardiometabolic decompensation phenotype. In the adipose tissue expandability model, once lipid storage capacity is exceeded, fatty acids are more likely to be displaced toward non-adipose tissues, contributing to lipotoxicity, low-grade inflammation, and insulin resistance [2].

Within the War Map framework, a sub-regressor is a target-relative, observable, modifiable, non-constitutive, and causally material biological state variable [1]. Accordingly, impaired adipose expandability can be framed not as a downstream marker but as a candidate causal control point that should be measured, perturbed, and verified if the goal is mechanistic reversal rather than mere marker management.

This paper translates that framework into a training hypothesis: the optimal session for insulin-resistant adiposity may not be the session that maximizes fatigue, calories, or acute lipolysis. It may be the session that phase-matches three metabolic events: low insulin availability, catecholamine-driven fatty-acid mobilization, and prolonged skeletal-muscle fatty-acid oxidation [4-6].

## 2. MECHANISTIC RATIONALE: SIGNAL, SINK, AND SPILLOVER

**Principle** / Maximize fatty-acid capture, not fatty-acid release.

The central danger in a metabolically unhealthy phenotype is not that fatty acids cannot leave adipose tissue. In hypertrophic and insulin-resistant adipocytes, basal non-esterified fatty acid (NEFA) release may already be elevated or poorly regulated. The

therapeutic problem is whether liberated substrate is oxidized by skeletal muscle or returns to storage and contributes to ectopic lipid burden.

Fasting lowers exogenous substrate availability and reduces the insulin context that suppresses lipolysis [4,5]. This does not make fasted exercise magical; it makes the metabolic starting condition cleaner for a lipid-flux intervention. Evidence from human exercise metabolism shows that pre-exercise carbohydrate ingestion can suppress lipolysis and limit fat oxidation during exercise, and meta-analytic evidence indicates that fasted aerobic exercise increases acute fat oxidation compared with fed exercise [4,5].

The interval primer is included to generate a strong sympathetic/catecholamine signal. High-intensity exercise is not used here primarily as a calorie-burning tool; it is used as a metabolic “ignition switch” for lipid mobilization. However, intervals alone may be incomplete for this specific target because whole-body fat oxidation is generally maximized at low-to-moderate intensities and reduced at very high intensities [6].

The low-intensity block is therefore the capture device. It is deliberately easy, often easier than the athlete or patient expects. In an insulin-resistant phenotype, the target is not hero intensity but an oxidative sink: a long period in which skeletal muscle can continuously take up and oxidize fatty acids.

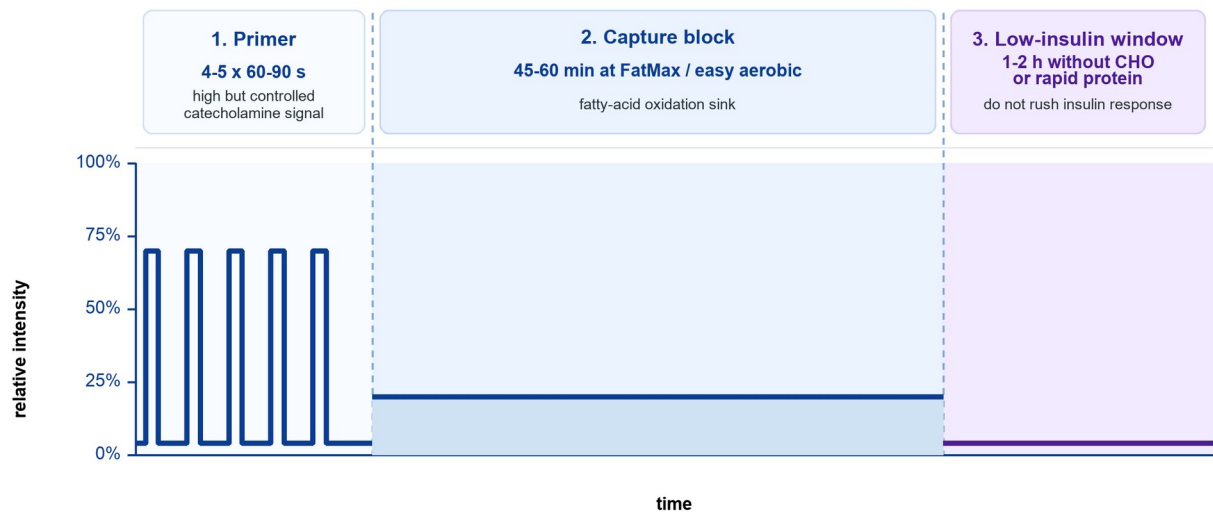


Figure 1. Protocol timeline. The intervention is designed as a signal-then-sink session: a short interval primer, followed by a prolonged low-intensity capture block, followed by a low-insulin post-exercise window.

### 3. PROPOSED TRAINING-NUTRITION PROTOCOL

Phase	Operational prescription	Primary mechanism	Do not overinterpret
Pre-session state	8-12 h overnight fast; water, electrolytes, and standardized non-caloric caffeine permitted if used	Lower insulin context; less exogenous substrate competition.	Not a universal recommendation for medication-treated

	consistently.		diabetes or hypoglycemia-prone subjects.
1. Interval primer	4-5 bouts of 60-90 s at high but controlled intensity; recovery easy enough to repeat the bout. Suggested research target: 85-95% HRmax or RPE 8-9.	Catecholamine signal; acute mobilization stimulus.	Not “all-out sprinting”; not the main calorie-burning phase.
2. Oxidative capture block	45-60 min of very easy aerobic work at individualized FatMax or below first ventilatory threshold.	Skeletal-muscle fatty-acid uptake and oxidation.	If the pace feels impressively hard, it is probably too hard for this protocol.
3. Post-exercise low-insulin window	1-2 h without carbohydrate or rapidly absorbed protein; later re-feed with adequate protein and nutrient density.	Hypothesized extension of fatty-acid oxidation and lower re-esterification pressure.	Conflicts with hypertrophy-optimized immediate protein timing; phenotype-specific hypothesis only.

## 4. MATHEMATICAL FORMALISM

The intervention can be formalized as an optimization problem over exercise-nutrition policies. The objective is not to maximize fatty-acid release, because release without oxidation may increase the probability of re-esterification or ectopic redistribution. The objective is to maximize captured lipid flux: the portion of liberated fatty acid flux that is simultaneously matched by oxidative disposal capacity.

**One-sentence principle** / Maximize fatty-acid capture, not fatty-acid release.

$$\pi^* = \arg \max_{\pi \in \Pi} \mathbb{E}_{\theta} \left[ \int_0^{T_{\pi} + \tau} \min(\dot{F}_{\text{rel}}^{\pi}(t), \dot{F}_{\text{ox}}^{\pi}(t)) e^{-\lambda_I \mu^{\pi}(t) - \lambda_C C^{\pi}(t)} dt - \eta \int_0^{T_{\pi} + \tau} (\dot{F}_{\text{rel}}^{\pi}(t) - \dot{F}_{\text{ox}}^{\pi}(t))_+^2 dt \right]$$

Equation 1. Candidate policy objective for a mobilize-capture protocol.

Term	Interpretation
$\pi$	The complete exercise-nutrition policy: fasting state, interval primer, low-intensity block, and post-exercise feeding timing.
$F_{\text{rel}}(t)$	Rate of fatty-acid release from adipose stores.
$F_{\text{ox}}(t)$	Rate of fatty-acid oxidation by skeletal muscle.
$\min(F_{\text{rel}}, F_{\text{ox}})$	Captured lipid flux: only the portion of released substrate that can be oxidized counts as useful flux.

I(t)	Insulin exposure, penalizing conditions that suppress lipolysis and shift substrate storage dynamics.
C(t)	Exogenous carbohydrate or rapidly absorbed protein exposure, representing substrate competition and insulinogenic pressure.
positive spillover term	A penalty when fatty-acid release exceeds oxidation capacity, interpreted as re-esterification or ectopic-distribution risk.

## 5. HYPOTHESES AND PREDICTIONS

**Primary hypothesis.** Compared with duration-matched conventional steady-state aerobic exercise and interval-only exercise, the fasted interval-primer plus FatMax capture protocol will produce larger reductions in visceral adipose tissue, waist circumference, intrahepatic lipid, fasting insulin, triglyceride-to-HDL ratio, and postprandial glucose/insulin exposure in insulin-resistant adults.

**Mechanistic hypothesis.** The interval primer increases acute lipolytic signaling, while the subsequent low-intensity block increases the probability that liberated fatty acids are oxidized rather than re-esterified or redirected toward ectopic depots.

**Performance-adaptation hypothesis.** The protocol may improve FatMax intensity and submaximal respiratory exchange ratio (RER), indicating a shift toward greater lipid oxidation at a given absolute workload. VO2max improvements may occur but are not the main purpose of the protocol.

**Nutrition-timing hypothesis.** The 1-2 h post-exercise delay of carbohydrate and rapidly absorbed protein may preserve a lower-insulin environment long enough to extend fatty-acid oxidation. This is a deliberately testable and controversial claim, not a settled rule.

## 6. EVIDENCE MAP: WHAT IS ESTABLISHED VERSUS SPECULATIVE

Claim	Evidence status	Reference anchor
Exercise can reduce visceral and perhaps hepatic fat in adults with T2D.	Supported by systematic review/meta-analysis; effect sizes vary.	[3]
Fasted aerobic exercise increases acute fat oxidation versus fed aerobic exercise.	Supported for acute substrate use; long-term body composition superiority is not established.	[5]
Pre-exercise carbohydrate can suppress lipolysis and limit fat oxidation during exercise.	Supported by controlled human physiology study.	[4]
FatMax identifies an individualized intensity at which fat oxidation is maximal.	Supported as a measurement concept/protocol; varies by training status and phenotype.	[6]
Immediate protein after endurance exercise	Supported; relevant trade-off against	[7]

increases MPS and net protein balance.	the low-insulin window.	
The exact sequence proposed here outperforms matched alternatives for visceral/ectopic fat.	Unproven. This is the core falsifiable hypothesis.	Future trial

## 7. PROPOSED PILOT STUDY

**Design.** An 8-12 week randomized controlled pilot trial in adults with insulin resistance, elevated waist circumference, metabolic syndrome, T2D not requiring hypoglycemia-prone medication adjustment, or imaging/biomarker evidence suggesting visceral or ectopic lipid excess.

**Arms.** (1) Conventional moderate continuous training; (2) interval-only training; (3) low-intensity FatMax training only; (4) fasted interval-primer plus FatMax capture plus delayed carbohydrate/rapid-protein window.

**Dose matching.** Total weekly exercise time and estimated energy expenditure should be matched as closely as practical. The distinguishing variable is sequence and substrate context, not simply total movement.

**Primary endpoints.** MRI- or DEXA-derived visceral adipose tissue, intrahepatic lipid by MRI-PDFF or magnetic resonance spectroscopy, waist circumference, fasting insulin, HOMA-IR, triglycerides, HDL-C, and CGM-derived postprandial glucose AUC.

**Secondary endpoints.** VO<sub>2</sub>max, FatMax intensity, submaximal RER, fasting NEFA, beta-hydroxybutyrate, ALT/AST/GGT, subjective hunger, sleep quality, adherence, and adverse events.

**Exploratory endpoints.** Adiponectin, hsCRP, IL-6 response, lipidomics/metabolomics, post-exercise NEFA clearance kinetics, and wearable-derived recovery markers.

## 8. SAFETY, LIMITATIONS, AND REVIEWER-PROOFING

This protocol should not be interpreted as medical advice or as a replacement for established diabetes care. People using insulin, sulfonylureas, or other hypoglycemia-relevant therapies should not attempt fasted exercise or delayed post-exercise feeding without clinical supervision and glucose monitoring. Physical activity can lower glucose for many hours after exercise, and hypoglycemia risk depends on medication, starting glucose, intensity, duration, and prior nutrition [9].

The post-exercise low-insulin window is the most controversial element. Protein after endurance exercise can increase myofibrillar protein synthesis and whole-body net protein balance [7]. Therefore, delaying protein is not appropriate for every goal. It is proposed here only as a phenotype-specific lipid-flux strategy, with total daily protein preserved [7,8].

The model also risks being too elegant. Human physiology is messy: NEFA kinetics, adipose depot biology, liver fat, muscle insulin sensitivity, menstrual status, medications, sleep, stress, and prior training all influence response. The protocol should be treated as a falsifiable control-system hypothesis, not as a guaranteed fat-loss hack.

The strongest claim that can currently be made is this: the components are individually plausible and evidence-anchored, but the complete sequence requires direct randomized testing.

## **9. PRACTICAL EXECUTION NOTES FOR A RESEARCH PROTOCOL**

Intensity control. Use gas exchange testing to individualize FatMax where possible. If unavailable, use an intensity below the first ventilatory threshold: nasal-breathable, conversational, and deliberately easy.

Primer control. Avoid all-out sprinting in sedentary or high-risk participants. The primer should be hard enough to create a signal but controlled enough to avoid injury, excessive lactate accumulation, or premature session termination.

Nutrition standardization. Standardize the last evening meal before fasted testing sessions. Record caffeine, sleep, medications, menstrual phase where relevant, and prior-day training load.

Adherence safeguard. The protocol should feel counterintuitive: the hardest part is often keeping the long block easy. The mantra is: ignite, then oxidize.

## **10. CONCLUSION**

The proposed mobilize-capture-do-not-spill protocol reframes aerobic exercise for insulin-resistant adiposity as a problem of lipid-flux coordination rather than calorie expenditure alone. A short interval primer supplies the signal, a prolonged low-intensity block supplies the oxidative sink, and a post-exercise low-insulin window attempts to reduce immediate substrate and insulin competition.

If supported empirically, this model would not replace standard exercise recommendations; it would refine them for a specific phenotype: individuals in whom adipose expandability, visceral fat, liver fat, and insulin resistance suggest a failure of lipid buffering and oxidative disposal. The protocol is intentionally bold, but its scientific value depends on whether it can be measured, falsified, and improved.

## **CONTRIBUTIONS**

Denis Varvanets: conceptualization, mechanistic protocol design, literature synthesis, formal model development, writing - original draft, and writing - review and editing. The author approved the submitted version for publication.

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## DATA AND SUPPLEMENTARY MATERIAL ACCESSIBILITY

No new datasets were generated or analyzed for this hypothesis/protocol manuscript. No supplementary material is associated with this version.

## COMPETING INTERESTS

The author declares no competing interests.

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