MAXACADEMY

THE HEALTH-AFTER-50 PLAYBOOK

DR MAX

LIVE LONGER, STRONGER

THE HEALTH-AFTER-50 PLAYBOOK DR MAX

THE HEALTH-AFTER-50 PLAYBOOK

Insulin Resistance, Fatty Liver, and the First 7 Days of Taking Back Your Metabolic Health

Dr Max
MAXACADEMY
First Edition — 2026

MAXACADEMY is the publishing brand of Dr Max.

Published by MAXACADEMY

First published: 2026

MAXACADEMY.com

MEDICAL DISCLAIMER

A note on the nature of this book

This book is health education. It is not medical advice. It is not a diagnosis. It is not a treatment plan. It does not replace clinical care.

The information in these pages is intended to help readers understand how the body changes after 50 and what the clinical evidence supports in the areas of nutrition, movement, and recovery. It is not intended — and should not be used — as the basis for personal medical decisions made without clinical guidance.

If any of the following apply to you, please discuss lifestyle changes with your clinician before acting on anything in this book:

• Cardiovascular disease, chest pain, or unexplained shortness of breath with exertion

• Diabetes or elevated blood sugar managed with medication

• Advanced liver disease or abnormal liver function

• Kidney disease or reduced kidney function

• A diagnosed sleep disorder, including sleep apnea

• Major orthopedic limitations, joint disease, or recent surgery

• Use of long-term prescription medications, including glucose-lowering drugs, blood pressure medications, blood thinners, or other regularly prescribed medicines

• Any condition for which you have been advised to restrict physical activity

Do not stop, reduce, or adjust any medication based on information in this book. Any medication-related decisions should be made with, and under the supervision of, a qualified healthcare provider.

If you experience emergency symptoms — including chest pain, sudden severe shortness of breath, loss of consciousness, or sudden neurological changes — seek immediate medical care. Do not consult this book.

Dr Max writes as a clinical physical therapist and health educator, not as a replacement for the reader's own clinician.

The author and MAXACADEMY are not responsible for personal medical decisions made without appropriate clinical guidance.

A note before you begin

This book is designed to be read in sequence, not opened at random.

Chapters 1 through 4 explain the metabolic system — the biological shift that occurs after 50, the two conditions that most commonly develop within that shift (insulin resistance and metabolic dysfunction-associated fatty liver disease), and the relationship between them. These chapters provide the map. Chapters 5, 6, and 7 describe what the evidence supports across three practical areas: nutrition, movement, and recovery. Chapter 8 translates this into a concrete starting point: a structured first-week protocol.

Reading the chapters in sequence matters because each chapter builds on the one before. The practical recommendations in Chapters 5 through 8 are more meaningful when the biological reasoning behind them is understood.

One principle applies throughout: use this book to support your conversations with your clinician — not to replace them. The goal of this education is to give you enough understanding of the underlying mechanism that those conversations become more specific, more informed, and more productive.

No part of this book constitutes individualized clinical guidance. Bring questions that arise from reading it to your own clinician.

A NOTE FROM DR MAX

Most adults over 50 receive advice that is not wrong. Watch your blood sugar. Lose some weight. Move more. Keep an eye on those liver enzymes. These are reasonable recommendations. They are also, very often, delivered without the explanation that would make them coherent.

What drives the metabolic shift after 50? What is insulin resistance, and why does a normal fasting glucose not rule it out? What is fatty liver disease, how common is it, and what does the updated science actually say about it? Why do nutrition, movement, and sleep all matter specifically — not generally, but for the metabolic context that most adults in this age group are operating in?

This book answers those questions. It explains the mechanism behind the metabolic shift after 50, introduces insulin resistance and fatty liver disease (MASLD) — and the relationship between them — and presents what the evidence supports across three practical areas: nutrition, movement, and recovery.

This is not a transformation program. It does not promise specific clinical outcomes. It does not replace clinical care. It is a briefing — the one most adults in this age group were never given and, once given, tends to change how they think about every other piece of health advice they receive.

Use it as education. Use it alongside your clinical relationships. And if it helps you ask better questions at your next appointment, it has done exactly what it was designed to do.

Dr Max

Clinical Physical Therapist | Health Educator | MAXACADEMY

TABLE OF CONTENTS

INTRODUCTION

The Briefing You Were Never Given

Something changes around age 50. Most adults notice it — in how the body responds to food, how energy shifts, how weight accumulates differently than it did before. They do the things they were told to do. They eat reasonably. They exercise more or less as they always have. And yet the picture keeps changing.

The standard advice usually follows. Lose some weight. Watch what you eat. Keep an eye on your blood sugar. Take a walk. Monitor those liver enzymes. These are not bad recommendations. But they are typically given without the one thing that would make them coherent: an explanation of what is actually happening in the body, and why.

That explanation is what this book provides.

Why Common Advice Feels Incomplete

The fragmentation of standard lifestyle advice after 50 is not a failure of medicine. It is a consequence of how clinical time works. A note about mildly elevated liver enzymes rarely includes a full explanation of what fatty liver disease is, how it develops, or why it matters in the context of broader metabolic function. A recommendation to watch blood sugar rarely comes with an explanation of what insulin resistance is, why it develops in this decade of life, or why a normal fasting glucose result

does not necessarily confirm that insulin function is working efficiently [B2].

The result, for many people, is a collection of partial instructions without a map. Eat better — but better toward what, and for which specific metabolic reason? Move more — but which kind of movement matters most, and why? These are not unreasonable questions. They are precisely what an informed adult should want to understand.

The Missing Briefing

This book provides the mechanism. It explains the biological shift that occurs after 50, introduces the two metabolic conditions most likely to be developing within that shift, and describes what the evidence supports across the three practical areas most relevant to addressing them: nutrition, movement, and recovery.

This book does not diagnose and does not prescribe. It is health education — structured, evidence-referenced, and designed to give readers enough understanding of their own biology that the advice they already receive begins to make more sense.

CHAPTER 1

What Actually Happened: The Metabolic Shift After 50

The Metabolic Baseline Shift

The term metabolic baseline refers to the background conditions that determine how the body processes and manages energy at any given point in adult life. This includes how much energy the body requires at rest, how efficiently muscle tissue takes up glucose and responds to insulin, how the body distributes and stores fat, and how well it adapts to the demands placed on it across each day.

This baseline is not fixed throughout adult life. It shifts.

In many adults, the years following 50 mark a period during which several interacting biological changes compound. None of these changes is catastrophic on its own. Each is a gradual process — invisible from one week to the next, legible only when viewed across years. But when these processes interact, the cumulative effect on the body's operating conditions is meaningful.

Muscle Loss: The Quiet Loss of Metabolic Capacity

Skeletal muscle is not simply structural tissue. It is metabolically active — meaning it consumes energy, participates in hormonal

signaling, and plays a direct role in how the body processes glucose and manages insulin demand.

The gradual, age-related loss of skeletal muscle mass — known as sarcopenia — begins in early adulthood and continues at a measurable rate across decades [A2]. In the post-50 context, this decline becomes increasingly relevant to metabolic function. As the volume of metabolically active muscle tissue decreases, the body's resting energy demand falls, and with it the site capacity for insulin-mediated glucose uptake.

Visceral Fat: The Location That Changes Everything

With age, fat tends to shift toward central and visceral distribution — stored not under the skin but around the internal organs. Visceral adipose tissue is hormonally active — releasing inflammatory molecules, contributing to a low-grade systemic inflammatory environment, and interfering with the signaling pathways that govern insulin sensitivity [A3]. Its volume is consistently associated with insulin resistance and metabolic dysfunction [A3].

Same Habits, Different Results

If skeletal muscle mass has gradually declined — reducing the body's resting energy demand and the tissue available for glucose disposal — and if visceral adiposity has gradually increased — introducing a more inflammatory metabolic environment — then the same food intake, activity level, and recovery patterns that maintained metabolic equilibrium at 38 will produce different results at 56. Not because anything

dramatic changed in behavior. Because the system is now operating under different constraints.

VA1 — POST-50 METABOLIC SHIFT: THE KEY CHANGES

MUSCLE CHANGES: Gradual sarcopenia reduces resting metabolic rate and insulin-mediated glucose uptake capacity [A2].

FAT REDISTRIBUTION: Visceral fat accumulates around internal organs, releasing inflammatory molecules that impair insulin signaling [A3].

THE RESULT: Same inputs → different metabolic outputs. Understanding the shift is what makes an informed response possible.

CHAPTER 2

The Hidden Driver: Understanding Insulin Resistance

How Insulin Works — and What Resistance Means

Think of insulin as a key and the cell's response as the lock. Under normal conditions, the key fits precisely: the signal is received, the cell responds, glucose is absorbed. In insulin resistance, the lock becomes less responsive. The key still exists — in fact, more of it is produced — but it takes considerably more effort to produce the same result [B2].

At a biological level, this reduced cellular responsiveness develops through multiple contributing factors. The chronic low-grade inflammatory state generated by visceral adipose tissue contributes to impaired insulin signaling [A3]. Reduced skeletal muscle mass decreases the body's total capacity for insulin-mediated glucose disposal.

The Compensation Trap

When cells respond less effectively to insulin, the pancreas compensates — increasing insulin output to achieve the cellular response no longer occurring efficiently. For a period, this compensation works. Blood glucose stays within a broadly normal range. Standard fasting glucose tests may return results

that appear acceptable [B2].

But the compensation itself carries consequences. Elevated circulating insulin promotes fat storage, particularly in visceral locations, and contributes to the metabolic conditions that make hepatic fat accumulation more likely [B2].

VA3 — THE INSULIN RESISTANCE SPECTRUM

Stage 1 — Efficient Response: Cells respond well. Normal insulin output. Normal post-meal glucose.

Stage 2 — Early Compensation: Cells slightly less responsive. Pancreas increases output. Blood glucose stays normal. Often asymptomatic.

Stage 3 — Active Compensation: Significant resistance. Pancreas working substantially harder. Fasting glucose may still appear normal.

Stage 4 — Compensation Limit: Pancreas can no longer compensate. Blood glucose rises. Pre-diabetes / Type 2 diabetes territory.

Insulin resistance is a spectrum, not a binary. A normal fasting glucose does not confirm efficient insulin function — it confirms the pancreas is still compensating.

VA4 — BLOOD PANEL: QUESTIONS TO BRING TO YOUR CLINICIAN

These are conversation prompts, not self-diagnostic instructions.

• **Fasting insulin and HOMA-IR** — provides a measure of insulin function that fasting glucose alone does not capture.

• **Fasting glucose and HbA1c** — HbA1c reflects average blood glucose over ~3 months.

• **Fasting triglycerides and HDL cholesterol** — can provide useful metabolic context when interpreted by your clinician.

• **Liver enzymes (ALT and AST)** — mildly elevated levels alongside metabolic risk factors carry clinical significance. Chapter 3 explains the mechanism.

The most useful question: 'What does this mean in the context of my other metabolic risk factors?'

CHAPTER 3

The Silent Organ: What Fatty Liver Disease Actually Is

Most people first learn they may have a liver concern not from a symptom — but from a blood test flagging mildly elevated liver enzymes, or from an abdominal ultrasound performed for an unrelated reason. The liver is often called the silent organ: it carries significant pathology with few, if any, perceptible symptoms in early stages.

A New Name for a Common Condition

The condition most commonly referred to as fatty liver disease now has a more precise name: MASLD, which stands for metabolic dysfunction-associated steatotic liver disease [C1]. Until 2023, this condition was known as non-alcoholic fatty liver disease — NAFLD. The name was changed following an international consensus process [C1] [G3]. If you received a NAFLD diagnosis in the past, it refers to the same condition.

MASLD is defined by the presence of excess fat within liver cells — hepatic steatosis — alongside at least one cardiometabolic risk factor, in the absence of other causes of liver disease [C1]. It is the most common cause of chronic liver disease in the world, affecting approximately one-third of adults globally [C1].

How It Develops

When insulin resistance develops, the liver receives altered signaling that promotes fat synthesis within hepatic cells. Elevated circulating insulin — the compensatory response from Chapter 2 — is a direct driver of hepatic fat accumulation through de novo lipogenesis [C2]. The result is a liver accumulating fat in a metabolic context it cannot efficiently clear.

VA5 — THE MASLD SPECTRUM

Simple Steatosis — Early MASLD: Excess fat within liver cells. No significant inflammation or liver cell damage. Most relevant to this book's metabolic education.

MASH — Metabolic Dysfunction-Associated Steatohepatitis: Fat accumulation with active liver cell inflammation and injury.

Fibrosis: Scar tissue develops from sustained inflammation. Requires clinical monitoring and specialist involvement.

Cirrhosis / Advanced Fibrosis: Extensive scarring. Requires specialist hepatology management.

SAFETY NOTE

This book focuses on early-stage metabolic education. Advanced liver disease — fibrosis, cirrhosis, hepatitis, or other diagnosed liver conditions — requires specialist clinical care and is outside the scope of this book.

If you have been diagnosed with hepatitis, cirrhosis, advanced fibrosis, or another liver disease, this chapter is educational context only. Your care should be managed by a specialist clinician.

CHAPTER 4

Why These Two Conditions Are Running the Show

Insulin resistance and MASLD are not coincidentally common. They share biological drivers and reinforce each other in a self-sustaining cycle.

The Bidirectional Relationship

Insulin resistance promotes hepatic fat accumulation. But a liver carrying excess fat — particularly at the stage of active inflammation — generates inflammatory signals that circulate systemically, contributing to further impairment of insulin signaling [C1]. The arrow runs in both directions. Each condition deepens the metabolic context in which the other operates.

The Shared Upstream Driver

Visceral adiposity sits upstream of both conditions simultaneously. It secretes inflammatory molecules that impair insulin signaling — feeding the insulin resistance node — and promotes the conditions in which hepatic fat accumulates — feeding the MASLD node [A3].

VA6 — THE METABOLIC WEB

VISCERAL ADIPOSITY (shared upstream driver — feeds both nodes simultaneously)

↓↓

INSULIN RESISTANCE ⇔ MASLD

(bidirectional — each condition contributes to the other)

Outputs from both nodes: Systemic inflammatory state · Liver progression risk · Cardiovascular risk association

These outputs reflect population-level associations. They do not predict individual outcomes.

Understanding this cycle does not require a clinical diagnosis to be relevant. The metabolic environment it describes is what the rest of this book addresses. Chapter 5 begins that work.

CHAPTER 5

The First Lever: What You Eat

The advice is common: eat better, cut back on sugar, watch what you eat. These are not wrong recommendations — but they are not specific enough to be reliably actionable in the context of insulin resistance and MASLD, where the metabolic mechanism behind dietary change is what makes the recommendation make sense.

Why Nutrition Matters for This Specific Context

A 2026 systematic review and meta-analysis of 24 randomized controlled trials found that dietary interventions can produce measurable improvements in liver health biomarkers in individuals with MASLD [C3]. The evidence consistently identifies patterns of dietary quality associated with improved outcomes.

The Remove / Shift / Add Framework

Rather than a prescriptive meal plan, the following framework organizes dietary change into three directional movements, each with a specific metabolic rationale.

REMOVE	SHIFT	ADD

Sugar-sweetened beverages (juice, soda, sweetened drinks)	Refined carbohydrates → whole-grain, higher-fiber alternatives	Vegetables and legumes at most meals
Ultra-processed foods (refined starches, added sugars, minimal fiber)	Processed fats → whole-food fats (olive oil, nuts, oily fish)	A clear protein source at 2+ meals
Refined starch-heavy meals producing sharp glucose rises	One large protein load → distributed across 2–3 meals	Consistent meal structure

"This is a framework, not a meal plan. Use it to guide your next food decision, not to judge your last one."

The Mediterranean-Style Pattern

The pattern the evidence most consistently describes includes: high vegetable intake, legumes at multiple meals, whole-food fats (olive oil as the primary added fat), lean and protein-rich foods, moderate whole grain intake, and low ultra-processed food and sugar-sweetened beverage intake [C3] [C4].

If you have diagnosed kidney disease or reduced kidney function, discuss protein intake with your clinician before making changes.

CHAPTER 6

The Second Lever: How You Move

Many adults over 50 are already moving. They walk, use a stationary bike, take the stairs. These habits carry real value. But for many in this age group, regular walking or light cardiovascular activity is not a complete movement strategy for metabolic health — because it addresses only part of what the post-50 metabolic environment requires.

Why Muscle Is the Metabolic Target

Skeletal muscle is one of the major sites of insulin-stimulated glucose uptake in the body. When muscle takes up glucose efficiently, the metabolic demand on the liver and pancreas is reduced. Without a specific mechanical stimulus, muscle mass continues to diminish — and with it, the body's capacity for efficient insulin-mediated glucose disposal. Resistance training provides that stimulus [A2] [E1].

RESISTANCE TRAINING	AEROBIC ACTIVITY
Primary metabolic target for adults over 50	Essential — not optional. Complements resistance training.
Preserves and builds skeletal muscle mass	Supports cardiovascular function and metabolic flexibility

Directly improves insulin-mediated glucose disposal [E1] [E3]	≥150 min/week moderate-intensity recommended [G2]
2+ sessions/week (muscle-strengthening activities)	Walking, cycling, swimming — any sustained moderate effort

When aerobic and resistance training are combined, the movement framework becomes more metabolically complete than either approach alone [E2]. Sedentary time carries metabolic consequences independent of structured exercise [E2] — brief movement interruptions matter.

SAFETY NOTE

Before beginning a new exercise program, speak with your clinician first if any of the following apply: cardiovascular disease; chest pain or shortness of breath with exertion; dizziness or light-headedness; uncontrolled blood pressure; major orthopedic limitations or recent surgery; advanced liver disease; diabetes managed with medication; any prior instruction to restrict physical activity.

These guidelines are population-level recommendations, not individualized medical prescriptions. Your starting point should reflect your clinical context.

CHAPTER 7

The Third Lever: How You Recover

Sleep is the lever most often treated as optional. The evidence from controlled experiments suggests this significantly underestimates what sleep deprivation does to insulin sensitivity.

What Sleep Actually Does to Your Metabolism

Short-term sleep restriction — even across a few consecutive nights — measurably reduces insulin sensitivity in healthy adults [F2]. This is not a general wellbeing effect. Sleep restriction disrupts the insulin sensitivity pathway through mechanisms that overlap directly with the metabolic conditions described throughout this book [F1] [F2].

When sleep is restricted, cortisol follows an abnormal diurnal rhythm, growth hormone secretion is impaired, inflammatory markers rise, and the circadian system loses the timing signals that govern metabolic function [F2] [F1]. Cells become less sensitive to the insulin signal — the same pattern of impaired signaling described in Chapter 2.

The RCT Evidence

A systematic review and meta-analysis of randomized controlled trials found that sleep restriction consistently produces measurable reductions in markers of insulin sensitivity [F2]. The

causality direction is clear: restricting sleep degrades insulin function. Restoring adequate sleep supports it.

SAFETY NOTE

If you experience persistent sleep problems, excessive daytime sleepiness, or have been told you stop breathing during sleep, discuss this with your clinician — sleep disorders require clinical assessment.

VA9 — RECOVERY BASELINE CHECKLIST

This is not a clinical assessment. It is a personal baseline.

1. Average sleep duration on a typical weeknight: _____ hours

2. Sleep quality self-rating: restorative / mostly restorative / frequently disrupted / consistently poor

3. Typical bedtime consistency: consistent within 30 min / varies by 1 hour / highly variable

4. Average perceived stress level over the past month, 1–10: _____

5. One recurring weekly stressor you have some direct influence over: _____

CHAPTER 8

The Health-After-50 First-Week Protocol

The purpose of this chapter is simple: to translate everything in Chapters 1–7 into a concrete, actionable starting point. One action per day, across eight days, covering all three levers.

SAFETY NOTE

BEFORE YOU START: If you have any of the conditions listed in the Medical Disclaimer or in the Exercise Safety Box (Chapter 6), discuss lifestyle changes with your clinician before beginning this protocol. Do not use this protocol as a substitute for clinical care, medication management, or clinician guidance.

Do not stop or adjust any medication based on this protocol.

DAY 0 **ESTABLISH YOUR BASELINE**

Complete the Recovery Baseline Checklist (VA9), note your current eating pattern against the Remove/Shift/Add framework (VA7), and review the Movement Framework (VA8). This is your personal starting point. Keep a note to compare against Day 7.

DAY 1 **REMOVE ONE SUGAR-SWEETENED BEVERAGE**

Replace one sugar-sweetened beverage today with water, plain tea, or coffee without added sugar. This addresses the highest-yield removal target for the hepatic and insulin signaling pathways described in Chapter 5 [C3].

Whole fruit is not the concern here. The target is sugar in beverage form.

DAY 2 **ADD PROTEIN STRUCTURE**

Include a clear protein source at two or more meals today. Adults over 50 may benefit from protein intake above the standard dietary allowance for maintaining muscle mass [D1]. A clear protein source: eggs, fish, poultry, legumes, dairy, or plant-based protein.

Safety: If you have kidney disease or reduced kidney function, discuss protein intake with your clinician before making changes.

DAY 3 **SHIFT ONE CARBOHYDRATE SOURCE**

At one meal today, replace a refined carbohydrate with a higher-fiber alternative. White rice → legumes or brown rice. White bread → whole-grain. Processed snack → vegetables with protein. A single substitution at a single meal.

DAY 4 **ADD RESISTANCE MOVEMENT**

Perform a simple resistance-based movement session — bodyweight squats, wall push-ups, resistance band work, or equivalent. Duration and intensity should reflect your current capacity, not an idealized target. Resistance training signals muscle tissue to be maintained and developed, directly supporting insulin-mediated glucose disposal [E1].

If orthopedic limitations or cardiovascular conditions are present, speak with your clinician before adding resistance activity. Refer to the Safety Box in Chapter 6.

DAY 5 ADD AEROBIC CONTINUITY

Maintain or add comfortable aerobic activity within your physical capacity. A comfortable walk, gentle cycle, swimming, or equivalent sustained moderate-effort activity [G2]. Day 5 is not about meeting the 150-minute weekly target in a single session — it is about establishing aerobic continuity alongside Day 4's muscle-directed work.

DAY 6 INTERRUPT EXTENDED SITTING

Notice prolonged sitting and introduce brief movement interruptions where practical. Sedentary time carries metabolic consequences independent of structured exercise [E2]. Identify one extended sitting period and insert a 2–5 minute standing or walking break.

DAY 7 ADDRESS SLEEP CONSISTENCY

Establish a consistent bedtime tonight. Bedtime consistency supports the circadian signals that govern cortisol rhythm, growth hormone secretion, and insulin sensitivity [F2]. Choose a bedtime that is realistic to maintain — not ideal, realistic.

VA10 — FIRST-WEEK PROTOCOL SUMMARY CARD

Day 0: Establish baseline (VA9 checklist, VA7 framework, VA8 movement check)

Day 1: Remove one sugar-sweetened beverage

Day 2: Add protein to 2+ meals

Day 3: Shift one carbohydrate to a higher-fiber alternative

Day 4: Add a resistance movement session

Day 5: Add or maintain aerobic activity

Day 6: Interrupt extended sitting

Day 7: Establish consistent bedtime

One action per day. Stack each day onto the previous. Repeat what is repeatable.

Closing Note from Dr Max

You came to this book with a question most adults over 50 have asked at some point: why has my body changed, and what can I actually do about it?

By the time you reach this page, you have the map. The metabolic shift after 50 is real and biological — driven by gradual changes in muscle mass, visceral adiposity, insulin sensitivity, and hepatic fat accumulation. The two conditions most commonly developing within that shift — insulin resistance and MASLD — are connected, self-reinforcing, and largely asymptomatic in their earlier stages.

And the three levers that address that cycle: what enters the system (Chapter 5), what the body can do with it (Chapter 6), and whether the system can adapt (Chapter 7). With one concrete starting point in Chapter 8.

Use this education as a foundation for better clinical conversations — not a substitute for them. This book does not diagnose. It does not prescribe. It does not promise a specific outcome. What it offers is the mechanism behind the advice you have probably already received.

Dr Max

Clinical Physical Therapist | Health Educator | MAXACADEMY

How to Continue After the First Week

• Review your Day 0 baseline against what you actually did across the week

• Identify the one or two actions that felt most repeatable — keep those

• Let go of what wasn't realistic in its current form

• Do not interpret short-term results as failure — metabolic adaptation occurs across weeks and months, not days

• If clinical questions arise, bring them to your clinician before making independent adjustments

Questions to Bring to Your Clinician

• Can we look at my metabolic markers together — not just fasting glucose, but also HbA1c, fasting triglycerides, and HDL?

• Would it be appropriate to check fasting insulin or calculate HOMA-IR in my context?

• Has an abdominal ultrasound ever been performed to assess for hepatic steatosis?

• What level of resistance and aerobic activity is appropriate given my clinical picture?

• Are there any medication considerations I should be aware of as I make lifestyle changes?

Personal Tracking Notes

What I noticed this week:

_____

What felt repeatable: _____

_____

What felt unrealistic or difficult to sustain:

_____

One nutrition action to keep:

_____

One movement action to keep:

_____

One recovery action to keep:

_____

Questions for my clinician:

_____

Source Note

Throughout this book, key claims are linked to the underlying evidence using source markers — for example, [C1], [E1], and [F2]. These markers correspond to the reference list below. They represent a selected evidence base, not a comprehensive literature review.

All clinical decisions — including any decisions about diet, exercise, medications, or lifestyle — should be made with and under the guidance of qualified healthcare professionals.

References

[A2]
Karakelides H, Nair KS. Sarcopenia of Aging and Its Metabolic Impact. Current Topics in Developmental Biology. 2005;68:123–148. DOI: 10.1016/S0070-2153(05)68005-2

[A3]
Shetty S, et al. Visceral Adiposity and Cardiometabolic Risk: Clinical Insights and Assessment. Cardiology in Review. Published online July 7, 2025. DOI: 10.1097/CRD.0000000000000984

[B1]
Ballena-Caicedo J, et al. Global Prevalence of Insulin Resistance in the Adult Population: A Systematic Review and Meta-Analysis. Frontiers in Endocrinology. 2025. DOI: 10.3389/fendo.2025.1646258

[B2]
National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK). Insulin Resistance and Prediabetes. NIH/NIDDK. 2023. niddk.nih.gov

[C1] / [G3]
Tacke F, et al. EASL–EASD–EASO Clinical Practice Guidelines on the Management of MASLD. Journal of Hepatology. 2024;81:492–542. DOI: 10.1016/j.jhep.2024.04.031

[C2] / [G1]
Rinella ME, et al. AASLD Practice Guidance on the Clinical Assessment and Management of Nonalcoholic Fatty Liver Disease. Hepatology. 2023;77(5):1797–1835. DOI: 10.1097/HEP.0000000000000323

[C3]
Stern UM, et al. The Impact of Dietary Interventions on Liver Health Biomarkers in Individuals with MASLD: A Systematic Literature Review and Meta-Analysis. European Journal of Nutrition. Published February 14, 2026. DOI: 10.1007/s00394-025-03870-z

[C4]
Sualeheen A, et al. Mediterranean Diet for the Management of MASLD in Non-Mediterranean, Western Countries. Nutrition Bulletin. 2024;49(4):444–462. DOI: 10.1111/nbu.12707

[D1]
Campbell WW, et al. Nutritional Interventions: Dietary Protein Needs and Influences on Skeletal Muscle of Older Adults. Journals of Gerontology Series A. 2023;78(Suppl 1):67–72. DOI: 10.1093/gerona/glad038

[D2]
Ishaq I, et al. Role of Protein Intake in Maintaining Muscle Mass Composition Among Elderly Females Suffering from Sarcopenia. Frontiers in Nutrition. 2025;12:1547325. DOI: 10.3389/fnut.2025.1547325

[E1]
Jiahao L, et al. Effects of Resistance Training on Insulin Sensitivity in the Elderly: A Meta-Analysis of Randomized Controlled Trials. Journal of Exercise Science & Fitness. 2021;19(4):241–251. DOI: 10.1016/j.jesf.2021.08.002

[E2]
Zhang Q, et al. Effects of Aerobic, Resistance, Interval, and Combined Training on Glucose Metabolism in Older Adults. Frontiers in Physiology. 2025;16:1702669. DOI: 10.3389/fphys.2025.1702669

[E3]
Boyer WR, et al. The Role of Resistance Training in Influencing Insulin Resistance Among Adults Living with Obesity/Overweight Without Diabetes. Obesity Research & Clinical Practice. 2023;17(4):279–287. DOI: 10.1016/j.orcp.2023.06.002

[F1]
Singh T, et al. Does Insufficient Sleep Increase the Risk of Developing Insulin Resistance: A Systematic Review. Cureus. 2022. DOI: 10.7759/cureus.23501

[F2]
Sondrup N, et al. Effects of Sleep Manipulation on Markers of Insulin Sensitivity: A Systematic Review and Meta-Analysis of Randomized Controlled Trials. Sleep Medicine Reviews. 2022;62:101594. DOI: 10.1016/j.smrv.2022.101594

[G2]
U.S. Department of Health and Human Services. Physical Activity Guidelines for Americans, 2nd Edition. 2018. health.gov/sites/default/files/2019-09/Physical_Activity_Guidelines_2nd_edition.pdf

MAXACADEMY

HEALTH-AFTER-50 RESOURCES

For printable checklists, future Health-After-50 resources, and updates from Dr Max, visit:

MAXACADEMY.com

Follow Dr Max for weekly Health-After-50 content, metabolic health insights, and evidence-based education.