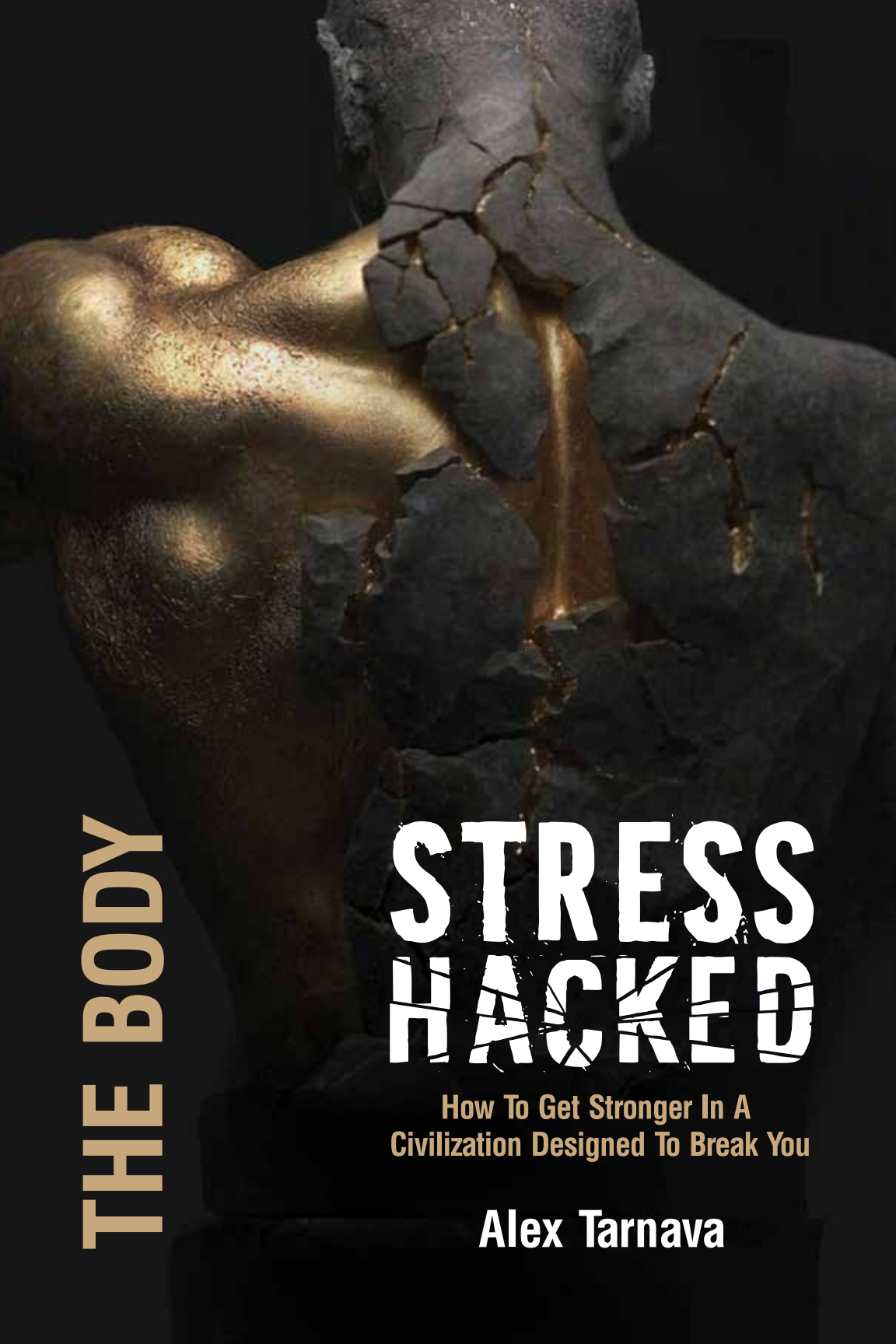
THE BODY
STRESS
HACKED
How To Get Stronger In A
Civilization Designed To Break You
Alex Tarnava

STRESS HACKED

How to Get Stronger in a Civilization Designed to Break You

Alex Tarnava

THE BODY

This book isn't short. It's not supposed to be.

Strength is not a hack. It's a system.

And systems can't be learned in soundbites.

Stress-Hacked
How to Get Stronger in a Civilization Designed to Break You
The Body

Dedication

I'm dedicating this book to my fiancée, Anastasia Astrakhantseva (soon to be Anastasia Tarnava, which she will be by the time you read this). This work took a tremendous amount of time, and she patiently allowed me to pursue it, sometimes at the sacrifice of our own quality time. Having the support and understanding of loved ones is critical in any mission to pursue what is challenging and difficult, and she offered this in ample amounts.

Acknowledgements

I want to thank those that have contributed to this work in valuable ways. Tyler W. LeBaron, M.Sc., Ph.D. (Southern Utah University, Molecular Hydrogen Institute), has been a friend for almost a decade. Much of the knowledge I have synthesized across The Body was greatly assisted by his input, feedback, and our countless hours of conversations. Tyler co-authored the chapter on molecular hydrogen, read through the book prior to release, which allowed him to write the foreword, one which is much kinder than I had hoped for.

My various editors are more than their titles give them credit in many ways. In contracting (separately) Vadim Gershteyn, M.P.H., Ph.D. (University of Illinois-Chicago; Alma Mater Europaea University) and Ljubomir Stevanovic, M.A. (University of Belgrade), my work exceeded my initial expectations. Dr. Gershteyn provided invaluable insights, owing largely to his multidisciplinary education—holding a Master's in Public Health and a Ph.D. in Humanities—and lifelong pursuit of truth and information. Dr. Gershteyn helped provide additional information I was not originally aware of, which strengthened my argument and understanding. Additionally, he assisted immensely in helping to polish my writing and thoughts to be more coherent. As I have pointed out, language is not my strength, so these efforts were greatly appreciated. Additionally, Mr. Stevanovic assisted tremendously by applying his expertise in Analytical Philosophy, approaching the work skeptically, and methodically fact-checking all of my statements, and citations, for accuracy. This work is essential, as the goal is truth and accuracy. When confronted with evidence that contradicts my narrative, it is imperative to adjust my narrative—not ignore the evidence. Mr. Stevanovic strengthened my work through his meticulous, as he termed it, pedantic need for accuracy.

In fact, our debates—almost always greatly exceeding the content written—between myself, Vadim and Ljubomir, spurred a new book, just completing, which will follow as an extension to StressHacked. Vadim and Ljubomir, in this way, were far more than just editors, they propelled true thought, inspiration, and the right amount of friction to create something entirely new.

I also want to acknowledge two other editors who provided great assistance throughout the project. One editor is Jovana Vukovic, Ph.D. (Faculty of Medicine, University of Belgrade) for her support in scientific editing and reference validation. Jovana was particularly helpful in the regenerative protocols section in The Body, where she was able to impart her own research experience on the topics.

Another is Solomon English: M.Sc., Chemistry (University of Cambridge), Ph.D. (University of Oxford, early-stage vaccine development). Solomon brought his passion for science, which motivates him elsewhere to mentor high school students interested in pursuing a science education, to the final read-throughs of

the book. Solomon meticulously reviewed, offering thoughts and feedback that I and the other editors had missed—a valuable final review to assist the rest of us, who were, by that time, "too close" to the project.

In terms of design, I would like to acknowledge Andrew Domachowski, a longtime friend who took on the role of my artist for the book's covers. I'm not a visual person, nor am I skilled at explaining the visuals I see in my mind's eye. Andrew patiently worked through my poor directions to deliver a fantastic end-product. Finally, I appreciate the work of Katherine Getta, my book designer, for helping me create a beautiful product and putting up with my team's lackadaisical process at times.

Finally, I want to acknowledge my trainer, Joey Foy, who humored me to record one of our sparring sessions to add photos within the book—and who humors me several times a week to work through unorthodox movements and combinations.

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Foreword

When Alex Tarnava first approached me to review and contribute to *StressHacked: The Body*, I appreciated not only his openness but his insistence on soliciting expert feedback to improve the scientific rigor and conceptual clarity of his message. My primary involvement was with the chapter on molecular hydrogen (Chapter 8), but our scientific and philosophical dialogues span nearly a decade. Reading the book brought back memories, as over the years I have observed Alex take ideas from his life experiences, some of which he narrates in this book, and immediately seek to contextualize them through a scientific lens. He would often share these events with me in real time, present his hypothesis, and invite me to critically analyze it.

What I've valued in our discussions is Alex's desire to be challenged, to test assumptions, and to engage with ideas that may contradict his own. As a scientist trained in critical thinking, I instinctively try to disprove hypotheses, and Alex has consistently welcomed this process as a means of refinement. Moreover, Alex has also played a unique and catalytic role in the hydrogen field through his willingness to engage rigorously, support research, and remain intellectually adaptable.

This approach resonates deeply with my own scientific career. As a researcher, educator, and Executive Director of the Molecular Hydrogen Institute (MHI), a science-based nonprofit, my mission has been to advance research, education, and public understanding of hydrogen as a potential therapeutic medical gas. From the beginning of Alex's work with open-cup hydrogen tablets, he has aligned his development efforts with the best available science.

I recall when he first shared with me the concentrations of hydrogen that he claimed the tablets could produce, I dismissed it at the time, noting that it contradicted Henry's Law. However, without hesitation, he sent samples to see if I could replicate or refute his findings. It turned out that the observed hydrogen concentrations didn't break the laws of chemistry; rather, they leveraged other principles, such as gas suspension and nanobubble dynamics.

I've participated in multiple research projects utilizing Alex's H_2-generating tablets and have seen firsthand his commitment to transparency and scientific integrity. In my experience, as well as that of other researchers whom I have spoken with, Alex has never attempted to inappropriately influence data reporting or interpretation, even when the findings were less favorable than hoped. Through product donations, subsidized research, and public advocacy, Alex and his companies have advanced awareness and research of hydrogen in ways that are often underrecognized. This commitment is part of what led MHI to select Alex as the inaugural chairperson of our public research committee, which enables independent contributors, including those outside of academia, to support MHI's scientific mission.

That same ethos of scientific alignment informed our collaboration on the InhaleH$_2$ product, a hydrogen inhalation device designed to deliver precise, non-flammable, therapeutic concentrations of molecular hydrogen. I initially served as a consultant, motivated by its potential in research settings. But as I witnessed Alex's continued dedication to scientific accuracy, I agreed to serve as the company's Chief Scientific Officer. This marks the first time in my 16 years in the field that I have publicly endorsed a hydrogen product as my own. I believe this innovation will have a transformative impact, both in applied therapy and in accelerating robust clinical and mechanistic research.

This book also reflects another mission of mine: to improve scientific literacy and critical thinking among the general public. That's why MHI has developed educational certification programs, offered both directly and through various accredited institutions. Alex represents the kind of open-minded learner we hope to empower, one who embraces scientific skepticism while exploring ambitious ideas. Though his personal health experiments and supplemental protocols (notably in Chapter 11) may be more adventurous than my own conservative approach, they are undertaken with a desire to ground hypotheses in physiology and science.

The writing in *StressHacked* is deeply personal, unflinchingly raw, and distinctly philosophical, blending memoir with incisive critical commentary on contemporary health paradigms. It is a text steeped in the concepts of anti-fragility and hormesis, methodically building a compelling argument around the virtues of resilience and the strategic rejection of pervasive modern tendencies toward fragility, overmedicalization, and comfort-driven stagnation. The narrative, candid and introspective, navigates the contours of Alex's own physical and psychological collapse and his subsequent deliberate reclamation of health through systematic exposure to controlled stressors and disciplined self-mastery. Rather than presenting a singularly sanitized or traditionally academic voice, the work is characterized by a deliberate and calculatedly polemical approach, provocative in its rhetoric, yet rigorously rooted in philosophical traditions, particularly Stoicism and Nietzschean thought.

Accordingly, it is important to clarify what this book is not. Despite the contemporary allure of the term "hack," *StressHacked* is not intended as a self-help guide offering quick fixes or easy solutions. Instead, it promotes an understanding of hormesis through the lens of systems thinking, emphasizing the necessity of personal discipline, meticulous observation, and careful application of stress for true adaptation and growth. It challenges the reader to employ critical thinking and discernment, encouraging nuanced consideration of how various modalities might fit uniquely into one's individual health and lifestyle strategies based on a personal algorithm of cost-benefit analysis.

As someone who has competed at elite levels, from running the Boston Marathon while in high school to national and international competitions in strength sports, I relate intimately to Alex's advocacy for purposeful discomfort and adaptive stress. The principles of resilience and hormesis he advocates are scientifically robust and resonate deeply with my personal experiences.

To be clear, *StressHacked* is not without controversy. Some of its rhetorical intensity may not appeal universally, and I myself do not fully align with every assertion or interpretative leap. Yet, the book's central tenets regarding the utility of hormesis, the biological importance of adaptive stress, and the necessity of resisting passive deterioration are strongly supported by scientific evidence. Its most valuable contribution lies in its capacity to provoke critical thought, challenging readers to reassess deeply ingrained assumptions and cultivate intellectual curiosity.

Readers are encouraged to engage with the work critically but openly. The blunt, confrontational language serves a purpose: behind this provocative approach lies a consistent call to personal accountability, physical autonomy, and thoughtful experimentation. Ultimately, *StressHacked: The Body* invites us to reconsider our relationship with stress, not merely as a threat, but as a potent tool, which, when intelligently applied, fosters resilience, vitality, and a profound sense of personal agency.

Tyler W. LeBaron, PhD

Executive Director, Molecular Hydrogen Institute

PREFACE:

The War on the Body

Picture 1. Me at a Crossfit competition

The moment right before my mitochondria filed a complaint.

I used to take my physical gifts for granted. Athleticism came easily to me; it always had. The gifts we receive without effort and struggle are the ones we value the least, at least until we lose them. Before my physical collapse, my feats of athleticism and strength were impressive, albeit not world-class. These were the type of feats you could brag about in local gyms, but not with the elite of the elite. There was a time when I could bench four plates (405lbs), deadlift five and some change (500+), while simultaneously being able to clear a 54" box via a plyometric jump, and string together 15+ bar muscle-ups. I did this while maintaining the ability to spar hard rounds of kickboxing, grapple, hike, and run.

My muscles didn't just move weight; they protected me. They protected me from fear, from doubt, and from ever having to question what I was made of. They protected my health, allowing me to maintain elite-level metabolic and cardiovascular measurements despite dietary and nightlife habits that should have spelled trouble. For instance, my daytime resting heart rate, as in sitting and relaxed, not asleep, hovered in the mid-30s. Then, one of my greatest allies and sources of pride, my body, betrayed me.

The collapse wasn't gradual; it hit all at once: sudden, severe, and disorienting. One week, I was hitting explosive lifts and bounding through workouts, and the next, I couldn't jump an inch off the ground. A mystery virus triggered a systemic breakdown. CRP levels spiked to 34 mg/L—nearly a hundred times a healthy

baseline—while my nervous system short-circuited, chronic fatigue set in, and narcolepsy symptoms followed. Sleep no longer restored me, and I needed upwards of 16-18 hours of it a day. Effort no longer yielded returns; I felt my best while active, doing as much as I was able. However, days when I set out to accomplish semi-normal activities became a "write-off" following the moderate exercises I was capable of. Once my system cooled down from the exercise, I would crash and lie at home, unable to accomplish anything. When the dust settled and these acute symptoms resolved, I was left with osteoarthritis in eleven joints and a body that no longer behaved within the only reality I had ever known. There was no bounce back, just a slow, brutal recalibration of what it meant to have a body at all.

When your identity is built on strength, collapse doesn't just take your body; it erodes your will. My physical deterioration didn't stop at the joints. When my body collapsed, it dragged my mental health down with it. I buried myself in work to distract myself from my new reality, and the cost showed up everywhere else. I gained nearly 100 pounds: I ate like garbage and drank enough to put Ernest Hemingway to shame. I'd always been able to eat and drink how I pleased, provided I was training hard, but the removal of training—and the subsequent increase in daily alcohol consumption, and exclusive reliance on poor quality nutrition—was too much for my physiology to handle. I was a high-functioning wreck, burning through days with intensity and through nights with whatever numbed the anguish.

Ironically, and importantly, my natural physical strength created a weak mind, at least in terms of my will towards physical maintenance. It wasn't until my body collapsed, dragging the will of my mind down with it, that I was able to rebuild both mind and body in the vision I finally understood was required. This experience was cathartic. I had always understood that those who are given everything appreciate nothing and will ultimately fail. I was blinded to the reality of the gifts I had been given, the gifts I hadn't earned. Having some of these gifts ripped from me, as if I had been violently mugged, opened my eyes to my own shortcomings, my own blind spots, and the reasons for my own arrested development.

I'd be remiss if I allowed any reader to believe that this catharsis was what led to my resurgence, as if I hit rock bottom, realized it, and built myself back from nothing. In fact, the catharsis occurred after I was rebuilt, not before. The reality of my situation is a lot less inspirational, but profoundly more human: there wasn't a grand breakthrough that initiated my return, only vanity; a reality that helped me understand myself, and, by extension, others, with greater clarity. I hit 267 pounds and refused to buy new clothes… for probably the 4th time in short order. That small line in the sand became a pivot point. I started losing weight, but more importantly, I started reclaiming my autonomy. The process of rebuilding my body reopened the path to recovering my mind. When I looked up from that crater, I saw a world I hadn't noticed before: a world built to keep people weak.

As I clawed my way back from the wreckage, building strength one painful inch at a time, I began to see this more clearly—and resent it more viscerally. The voices preaching softness weren't offering peace; they were selling sedation. The further I got from the edge, the angrier I became: because I'd been to that edge, I'd lived the collapse. I'd felt the weakness, not just in the body, but in the spirit.

Years went by, and I had made my way out of the darkness, out of the abyss, but I had not yet reclaimed my life; I had not yet started my ascent, or begun working towards the person I knew I could, and should, be. Then I heard a song: "Povorot" (the turn) by Machina Vremeni (Time Machine). The song talks about the turns in life, and where they can take you: the abyss or ascent, the whirlpool or the fjord. It talks about how everyone is afraid of change, but not to be afraid because we are still strong.

As I dissected the lyrics, my thoughts wandered into a direction that isn't the meaning of the song. Life's turn had taken me into the abyss, had I come out from it? Was I back on an ascent? The short answer was both yes and no. I had crawled to the top of the abyss and stagnated. I had improved some aspects of my body and advanced my businesses and financial well-being, but I had abandoned my intellectual pursuits. Moreover, I was avoiding some of the uncomfortable questions regarding my emotional behavior and stability.

So, I became resigned to force my way forward, not from the depths, which I had already escaped, but from the stagnant plain I'd reached after the climb: "iz proposti na vzlyot." At first, I got the grammar wrong, but my fiancée eventually corrected me. The proper translation wasn't the point here; the point was the mindset. Life may bring us change, and that change may drive us into an abyss, into turmoil, or guide us into ascension and stability. We cannot control every change that is thrust upon us, but we must never allow ourselves to be condemned or defined by these changes…these turns. Within us exists the strength to claw our way back, to turn misfortune into strength. So this became my mantra: I would not only pull myself from the abyss, but I would ensure my ascent trajectory was more explosive, guided, and relentless than ever before.

As I finally began my ascent, I increasingly became angrier and angrier about society and the culture of weakness. Every voice that told me to stay soft, stay broken, stay down, I took personally, because I knew exactly who it was trying to keep there.

We live in a society that actively shames physical excellence, where strength is "toxic," discipline is oppressive, and the pursuit of fitness is increasingly equated with extremism, where men are encouraged to be soft, androgynous, and compliant, where women are told that pushing their bodies is a form of internalized misogyny. Where the words "self-improvement" are now seen as acts of violence against others who choose to deteriorate. This isn't neutrality, it's sabotage.

We've created a culture where being fat is brave, being weak is virtuous, and being sick is inevitable, where criticizing obesity is "hate speech," but promoting it as beautiful and healthy is somehow empowering. We've reached a point where not wanting diabetes is considered a political stance. We are told to love ourselves as we are, but what if we hate what we've become? Then it is we who are broken, not the system. The same system that keeps us medicated, malnourished, chronically inflamed, and infertile. That tells us our low energy, brain fog, mood instability, hormonal collapse, and dependency on external validation are just "who we are." That we need another label, another prescription, another safe space—never a barbell, a cold plunge, or a fasted walk.

This inversion has become so complete that major media outlets—*The Guardian*, *TIME*, and others—have run pieces suggesting that fitness is a pipeline to fascism, complete with a connection to Nazi Germany (Townsend, 2022; Waxman, 2022). They imply, if not outright state, that strength, self-discipline, and physical sovereignty are somehow coded expressions of far-right ideology. As if creating a fitness regimen is one step away from organizing another Kristallnacht. As if wanting to walk uphill without pain makes you not just politically suspect, but dangerous. Strength is dangerous, but not to society, to those seeking total dominion over all others.

We are told to be kind to ourselves. Kindness without accountability is not love, it's decay. When I was first writing this book, I was sent a quote from Audre Lorde, which I was told captured my thoughts; surprisingly, since she was a radical feminist and self-proclaimed Marxist. Obviously, this is not my worldview, but her words, in this instance, ring true: *"Caring for myself is not self-indulgence, it is self-preservation, and that is an act of political warfare"* (Newman-Bremang, 2021). She was right. In today's culture of institutionalized fragility, every rep, every plunge, every fasted walk becomes a declaration: *I will not be managed*. This section of the book is not kind in the modern sense of the word. It is not gentle, and it is not affirming. It is a call to arms against the slow suicide of softness. Let's be clear: this isn't about aesthetics. Strength is not defined by visible abs. Some of the greatest athletes in history prove that.

Fedor Emelianenko—my favorite fighter—dominated heavyweight MMA with calm precision and terrifying power, and never once had a six-pack. Tyson Fury, despite never being lean, outclassed a generation of more "shredded" fighters before finally being bested by Oleksandr Usyk in back-to-back matches, choosing to say his farewell and stroll into the sunset of retirement. Shaquille O'Neal bulldozed his way through the NBA without ever fitting the modern fitness aesthetic. Frank Thomas, Cecil Fielder, and Prince Fielder all had Hall-of-Fame-level baseball careers while carrying significant mass. Babe Ruth—arguably the most iconic athlete in American history—was built more like George Costanza than the image of a sports legend we've been trained to envision, yet he changed the sport forever.

The idea that strength must look a certain way is modern marketing nonsense. A bit of fat, if your metabolic health is intact, is not a problem. Chasing hyper-leanness at the expense of performance, hormones, or sanity is just another form of dysfunction. The goal is adaptability, not Instagram validation. To put it another way, your body was not made for dopamine drips and Uber Eats, it was made for heat, cold, hunger, intensity, challenge, and movement. If you deprive it of stress, you deprive it of purpose. Furthermore, if you think a pill, a padded chair, and a 1,900-calorie meal plan are going to save you from aging, dysfunction, and despair, you've already lost. This is about integrity—it's about the will to remain human in a culture that increasingly rewards regression. Discomfort isn't the enemy, stagnation is, and every chapter that follows is a blueprint to reclaim the body the modern world has tried to erase.

I've periodically shared this parsed-together Nietzsche quote for the last 13 years. It's the beginning and end of a longer passage in *On the Genealogy of Morals*—the middle section cut to sharpen the focus. As time goes on, this quote becomes more relevant, not less. Western society is in the middle of a civil war. This is not a war between races or classes, but between those who adapt and those who deteriorate. Those who are weak in mind, body, and spirit have been organized and deployed—or more accurately, weaponized—by those who benefit from dysfunction. This is done not by helping the weak become strong, but by glorifying weakness itself.

The most tragic irony is that Nietzsche originally aimed this critique at his contemporaries of a Christian persuasion, and rightly so. However, today, it's often the modern Christians (Christians in name and identity, but not always in compatibility of philosophy) who are leading the charge against the new religion of fragility.[1] This book is a response to the world Nietzsche warned us about: a world where the strong begin to feel guilty for being strong, and the sick demand not just compassion, but complete and unchecked control, necessitating the elimination of the healthy and strong, as the healthy and strong are both a site of resistance and a source of ressentiment for the weak and sick.

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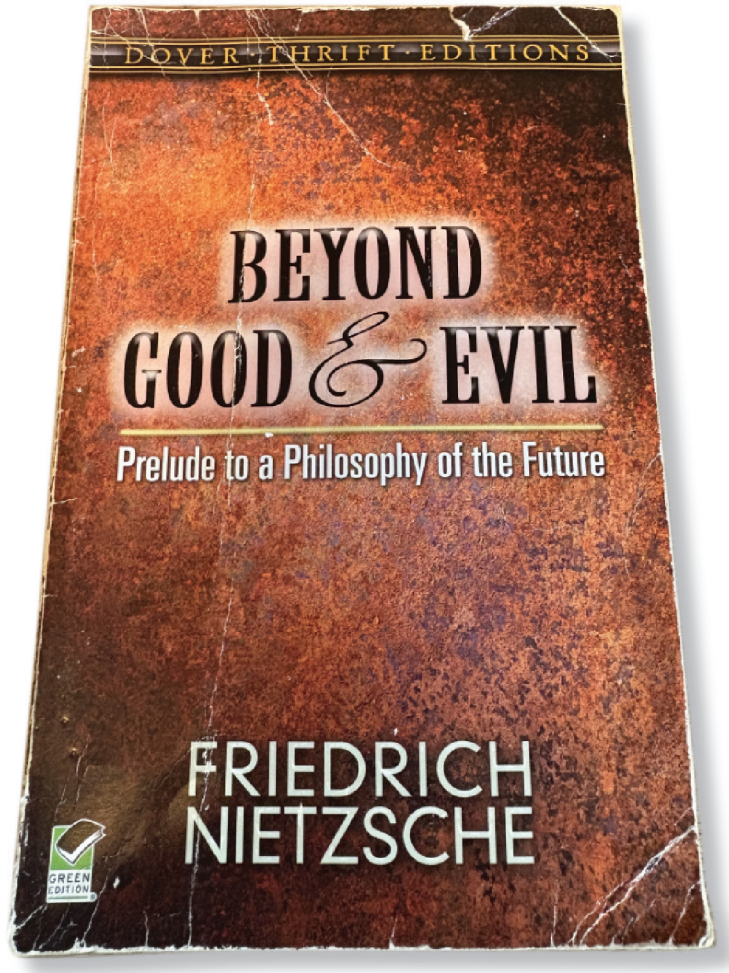
[1] Nietzsche's critique of Christianity targets what he calls "slave morality": a morality born of weakness, resentment (ressentiment), and a reversal of classical values. He argued that Christians of his era elevated humility, meekness, and suffering not out of inherent virtue, but as a psychological compensation for their powerlessness under Roman rule. These values, Nietzsche claimed, masked envy and weakness as moral superiority. His polemic was largely directed at the Christian moral sensibilities of 19th-century Europe, which were closely intertwined with liberalism, pacifism, and egalitarianism. Ironically, in today's political climate, many who still identify as Christians, particularly in the West, tend to align with conservative values and often stand in opposition to secular progressive ideologies that Nietzsche might have viewed as new incarnations of ressentiment, albeit divorced from religious theology.

Picture 2. A prized possession



Worn on the outside. Still dangerous on the inside.

They have taken a lease of virtue absolutely for themselves, have these weaklings and wretched invalids, there is no doubt of it; "We alone are the good, the righteous", so do they speak, "We alone are the men of good will." They stalk about in our midst as living reproaches, as warning to us- as though health, fitness, strength, pride, the sensation of power, were really vicious things in themselves, for which one would have some day to do penance, bitter penance. Oh, how they themselves are ready in their hearts to exact penance, how they thirst after being hangmen!...

At that time, doubtless, when they succeed in pushing their own misery, in fact, all misery, into the consciousness of the happy; so that the latter begin one day to be ashamed of their happiness, and perchance say to themselves when they meet "it is a shame to be happy, there is too much misery"...

But there could not possibly be a greater and more fatal misunderstanding than that of the happy, the fit, the strong in body and soul, beginning in this way to doubt their right to happiness. Away with this "perverse world"! Away with this shameful soddenness of sentiment! Preventing the sick making the healthy sick, this ought to be our supreme object in the world.

—FRIEDRICH NIETZSCHE

INTRODUCTION:

Physical Hormesis

Picture 3. My colleague Dr. Tyler LeBaron and I at a fitness show

Hormesis: because life will hand you ridiculous weights whether you train for them or not.

When I was younger, I believed that true strength was the ability to endure anything and that the strong ought to seek out challenges for enjoyment. I echoed thoughtless quotes such as "sleep is for the weak," and stubbornly demonstrated, while loudly exclaiming to all who weren't put off by my obnoxiousness, that I didn't believe in rest days—for exercise, work, or anything else. I even parroted statements to friends, family, and especially sales staff, such as, "*There are only two E's in life: excuses or enthusiasm.*" Of course, we all grow up, and with us, our opinions tend to mature, too. I'll get to how my views changed later.

First, though, a story: when an unusually cold winter hit Vancouver, which is not known for its frigid weather, the young me pounced on the mantra of enduring too much just to show off. Despite my best efforts to instill excitement into my team, my salesmen, who were mostly high school students, were less than enthusiastic: they

were cold, they complained, and our sales suffered. Many of them tried to argue that a bit of snow necessitated cancelling shifts. In Vancouver, it rarely snowed, at least back then.

As I watched the weather forecast, realizing it was a week of freezing temperatures, I started hatching a plan. The next evening, since it was an after-school job, I showed up to pick up my reps in a muscle shirt: just a muscle shirt, no jacket, and no gloves, meaning no protection from the elements. My reps were all bundled up in winter jackets, toques, and gloves—the full Canadian winter armor. I even bought them hand and foot warmers to put inside their gloves and boots.

As for my conditions in the van? The windows were wide open, and the air conditioning ran on full blast. I operated like this for hours every day that week, from the first sales rep I picked up around 4 PM to the last one I dropped off at home around 9:30 PM. This proved the point I wanted: that their excuses were absurd. If I could withstand far worse conditions than they, while doing my job and keeping them motivated and excited, then they, too, could withstand the short walks between houses, bundled up and receiving frequent respite in the warm homes of concerned customers. Their excuses vanished, and our sales surged. I behaved like this day one as a performance, but the rest of the week was fueled solely by spite.

To recap, through hormesis, the complaining stopped, and my reps started selling. Customers, seeing these kids braving the "cold," were more likely to invite them in and to hear them out. Some of my reps made more money in that week than most students earn in a month. By the end of that cold spell, no one flinched; they showed up ready. Sometimes resilience isn't built through careful pep talks; it's built through friction and discomfort. Resilience is built through the realization that the environment isn't the problem; your response to it is, as a certain philosopher named Epictetus argued long ago (Sofield, 2017).

Looking back, it was aggressive, perhaps too aggressive. I was 20 or 21, and I thought everyone needed to be hardened. I've grown since then to recognize that weakness is a choice people have the right to make. Though I also know this: comfort didn't bring those kids results, controlled stress did.

That story might sound extreme, but stress is supposed to feel extreme. That's what makes it powerful. When stress is introduced in a calculated, deliberate dose, in the right context, and with a specific aim, it doesn't just sharpen performance, it changes people. In other words, stress isn't optional. It comes for all of us—through physical

effort, environmental extremes, or metabolic challenge. Instead of preparing for it, we've been trained to run from it, to seek comfort. We idolize comfort and pathologize strain. Perhaps it shouldn't be surprising how often we wonder why we're falling apart.

Hormesis is the reversal of this existential gridlock. Let's go over how this principle works: you apply stress—precisely, not recklessly—and the body adapts. You grow and you fortify as a result, you build something that can't be faked, where no shortcuts exist. Adaptive stress helps you grow in ways that are not immediately connected to the stress, in ways that may at first seem impossible. In fact, hormesis may be the only way to improve your life in certain aspects, a perspective that I will put forward in this book.

Many people are looking for a miracle cure for whatever ails them. I often quip that most people want to go to bed broke and wake up rich, not having done any work to get there. If there were a magic elixir that restored youth, increased IQ, resolved all health problems—basically, an elixir to help you get rich, healthy, and beautiful—most wouldn't drink it if it weren't easily accessible, and didn't taste like Coca-Cola. Although, thankfully, I think people are becoming increasingly skeptical of such miracle cures, or rather, those offering such miracles that never pan out. I am not offering hormesis as anything like that; it is a principle, and a very powerful one. It takes work, as this book will show, but it's the only method that reliably produces something like flourishing in the long run.

Along these lines, there is something important to get out of the way: no treatment currently exists—no supplement, no stack, and no miracle protocol—that's been proven to extend human lifespan in any meaningful way. It's not even close, despite what the peddlers of life extension hype may state. That doesn't make hormetic tools worthless; we just need to be honest, seeking to understand what makes them effective, and what that means for our health and longevity. For me, the goal of hormesis isn't immortality, it's capacity: a capacity for a greater healthspan, and a body that works for as long as you live in it.

Practicing hormesis means rejecting magical thinking, a facet of skepticism that I explore both in this book and in The Final Thought War. Some experimental tools, like psychedelic microdosing (Anderson et al., 2019), or immunological training (Netea et al., 2020; Netea, 2016), may hold promise in improving people's lives in

both meaningful and measurable ways. Others, like giving allergens to infants,[2] can be outright dangerous in the wrong hands. When a life is on the line, or risk runs high, you don't take advice from a podcast, or even this book. You find the expert. Anyone suggesting a one-size-fits-all approach regarding any hormetic stress, and especially protocols stacking numerous hormetic stressors, is not an expert. Many are well-intentioned; said protocol has worked for them. That doesn't mean it will work for everyone, always.

Most tools aren't high-risk. In fact, most of them are hidden in plain sight. I discuss many throughout this book, including cold, heat, movement, and fasting. All the hormetic principles and examples I discuss in this book are based on cycles of strain and recovery, which means that what matters is how you use them and when you stop.

That's the map this book lays out: the science, the strategy, and the sharp edges of hormetic stress as a tool for physical resilience and biological performance. You'll learn how to push the system without breaking it, and why most people fail by doing either too little or, conversely, too much. This volume is about the body: *physical stress, environmental challenge, and physiological adaptation*. The companion book explores the mind, examining cognitive, social, and emotional pressures as a form of psychological training. You need both because one without the other is incomplete. If there is one lesson I think you should take away from *StressHacked*, it's that. If you take away a second lesson, it's that the dose makes the medicine—or the poison.

Hormetic stress is a double-edged sword for this reason: it recognizes that the very thing that heals you can wreck you. So, if you get the dose wrong, or apply it at the wrong time, or fail to recover, this poses a problem for your flourishing. Think of just a few hormetic principles: cold, heat, fasting, or lifting heavy things—these are all pharmaka.[3] Each one is both a remedy and a toxin, depending on the application of it. There's no neutral and no sideline to hormesis; you either build from the stress or you break from it.

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[2] Controlled introduction of allergens in infancy has shown real promise in reducing allergy risk, particularly with peanuts and other common triggers (e.g., Chan et al., 2018; Du Toit et al., 2016). Clinical protocols exist for gradual exposure under expert supervision. My criticism is not of the research itself, but of reckless, unsupervised application. Exposing infants to allergens without medical oversight—based on podcasts, influencer posts, or Internet hearsay—is not resilience-building but stupidity with potentially serious consequences.

[3] The ancient Greek word pharmakon, explored by Plato in Phaedrus, means both cure and poison (Reames & Sloey, 2021). Socrates uses it to describe writing: something that can help us remember or make us forget. It's a reminder that the same force can heal or harm depending on context, dose, and timing.

That's the difference between calculated exposure and chaos, or between a fighter who trains to take a punch and the one who walks in swinging without a guard. *The stressor doesn't care about your mindset*; it either forces adaptation or exploits your weakness. That outcome depends on precision.

Stress isn't the enemy; careless dosing is. As I mentioned, too little hormesis and you decay, too much and you snap. Don't let this scare you away—with the knowledge and understanding you gain from this book, you should be able to devise a plan on how to dose your stressors, how much recovery is needed for your specific physiology and total stress load, and what measurements you should be taking. Don't think of this book as a protocol, because it isn't. It's a guide to understand the principles behind these interventions, so you can create a protocol that works for you, specifically, so long as you commit to learning and to remaining honest with yourself. I begin this journey with cold, not because it's the most powerful tool, but because it's one of the most available, and one that remains dearest to me. It's the simplest, it's the hardest to excuse, and there's no gym membership needed for it. There's no supplement stack, either. There's just your body, the cold, and the discipline to step into discomfort.

Power starts with a shiver. Let's begin.

Picture 4. In Switzerland

Peak cold exposure: scenic views included, feeling in fingers not guaranteed.

CHAPTER 1:

Sharpened By Cold

Cold has never frightened me. If anything, it's where I've always felt most alive, where the world sharpens and all pretenses vanish. For most, cold is something to be avoided. For me, cold has been a tool, a challenge, and occasionally, a crucible.

The first time I realized cold could be an asset, not just something to endure, but something to wield, I was 18 and working as a team leader for a door-to-door sales operation in Calgary. The Calgary winter is much worse than the one in Vancouver, where I had subjected my entire team to cold hormesis a couple of years later, temperatures dropping to -20°C, even -30°C on some days. Most reps layered up like arctic explorers, and I wore a t-shirt.

I already discussed why this works in the Introduction: it wasn't a stunt, but a highly successful operation, because the confusion of seeing a kid in the cold stripped people of their sales resistance. When most people open the door and see a kid standing outside in -20°C weather, instinct kicks in. Their suspicion and resistance are subdued, and concern takes over. If that kid is in a t-shirt, the instinct is amplified. Additionally, this instinct served the homeowner, as inviting us into their home prevented the cold from entering. So, they'd invite me in virtually 100% of the time. Almost always, the proverbial "ice" would be broken as they would be incredulous as to why I was out in the cold in a t-shirt, and if I was okay. I'd smile and let them know that since it was so cold, all of their neighbors were inviting me in. Bundled up in a winter jacket and gloves, I was getting too hot and sweaty in everyone's warm homes, so I gave my winter attire to my manager. If I needed it back, he was a quick call away.

This served a dual purpose, as my response, equal parts truth and sleight of hand, allowed them to relax and directed them to feel part of something good. It also implied that all of their neighbors were on board with what I was doing, further lowering resistance. So, once this initial dance was out of the way, I would deliver my pitch with calm confidence and clarity, drastically different from the robotic salespeople they were likely used to. With the offer we had from the newspaper, the deal sold itself: the challenge was reducing the resistance and guiding customers to a place to listen from a neutral state of mind. My strategy accomplished this, and my daily sales numbers skyrocketed, remaining consistently high.

The cold didn't stop at sales; my love for this ancient form of conditioning was only starting. Years later, in Amsterdam, I stayed in a -20°C subzero bar for the full duration—if memory serves, an hour. I had no coat, no gloves, wearing just jeans and a t-shirt, while everyone else shivered in layers and venue-issued

Picture 5. In Switzerland Cont'd

Nothing like a glacier to remind you you're not the main character.

parkas. My fiancée, Russian by birth and tougher than most, wore two jackets. I stayed for the thrill: for the grin that creeps in when your skin protests and your blood pulls tight and your mind clears like frost vanishing from a glass.

In Russia, I met the cold again on its own terms—in a traditional banya, booked as a gift from research collaborators. The heat in their sauna was brutal, the worst I have ever endured. I must admit that I've always handled cold better than heat; my physiology throws red flags quickly when temperatures climb. Veniks thrashed across my skin, which only intensified the burn. Even with the staff dumping ice water on my head as my body deteriorated into heat-driven agony, I could barely endure; in fact, I was the first to leave the sauna every round. It wasn't until the plunge that I found what I'd come for: the shock, the stillness, and then the clarity and calm. I begged in Russian to extend my stay in the ice bath. I told them I was a white bear, that this was my terrain. They laughed, then relented—five minutes, then ten. Everyone else entered the cold for mere moments, forcing themselves to submerge

Picture 6. My Fiancée and I In a Subzero Bar In Amsterdam

Love is patient. Love is kind. Love is freezing solid in an Amsterdam ice bar.

Picture 7. Me In a Traditional Russian Banya

Somewhere between toughness and madness lies the banya.

once, then rushing to escape at a speed resembling panic. For me, even the 10 minutes was not enough, not after the heat I had endured, but it was better than nothing. The heat became harder to bear each time, while the cold grew easier. My system knew where it belonged.

Even the familiar can betray you if you grow arrogant. A few months later, south of Cancun, on what was meant to be a working retreat for me, and a "baby moon" for my fiancée, I made a mistake: alternating sauna and cold plunge like usual, I took a call in the plunge. At first, I was focused, calm, and then… I began to realize I had been in the cold for too long. Rather than feeling alive and energized, my brain had started to slow. This was my third round in the cold plunge, escalating from 8 minutes to 15 minutes, and then over 25 minutes on this final rotation. This is the first, and only time in my adult memory, when the cold was not invigorating, but rather, depleting. My core chilled, my limbs were sluggish, and my energy vanished: I had overshot the dose. The same stress that once made me sharp now made me slow.

That's the thing about cold: like any great teacher, it punishes complacency and rewards presence. It doesn't care about your track record or your tolerance. It cares about precision, about listening, about knowing when the test becomes the threat. I don't romanticize it, but I do respect it, because cold doesn't lie: it tells you exactly where you stand. If you listen closely, it tells you what still needs work: after all, not all cold is created equal. There's the kind that punches your nervous system awake: ice bath, cryochamber, wind-cutting sprint through a winter dawn. It hits fast, and it steals your breath. If you're ready, it gives you something back: clarity, focus, and energy. That's the *hormetic window*—the short, sharp stress that signals your body to upgrade.

Then there's the other kind of cold: the slow, creeping chill that seeps into your bones and refuses to leave. This is a cold that lingers, not for minutes, but hours. The cold that you can't quite shake even after you've dried off and changed. That's not hormesis, it's *degradation*.

The science backs the distinction. Acute cold exposure, which is brief, controlled, and high-intensity, spikes thermogenesis, activates brown fat, and improves metabolic flexibility (Blondin et al., 2015; Ouellet et al., 2012). A study noted that the most dramatic physiological changes occurred within the first thirty minutes of exposure, when participants reported less discomfort (Acosta et al., 2018). You read that right: the most powerful adaptations happened not at the peak of suffering, but while the body still had agency to respond. This is before survival mechanisms kicked in and before systems started shutting down to conserve heat.

Contrast that with chronic cold. A study on piglets compared short bursts of near-freezing temperatures in intermittent bursts against lower-level but extended cold. The first group of piglets adapted; they had their white fat convert to brown, while thermogenesis ramped and metabolism sharpened. The second group simply added fat; white adipose tissue increased, which is a defensive response (Gao et al., 2018). Instead of building capacity, they built *insulation*. It's the body's way of saying, "If we're stuck here, we'd better soften up."

It is important to consider what likely accompanied the observed increase in white adipose tissue (WAT) from chronic cold. As confirmed in other models, including human retinal pigment epithelial cells, WAT is not just inert insulation. WAT actively releases pro-inflammatory cytokines, such as TNF-α and IL-1β, which increase reactive oxygen species (ROS) through mitochondrial and NADPH oxidase pathways (Yang et al., 2007). In other words, excess WAT doesn't just sit there; it contributes to systemic oxidative stress and inflammation, potentially reinforcing the maladaptive loop that chronic cold appears to trigger. It is also important to note that, even though cold exposure is often linked to mitochondrial activation, Yun Gao and colleagues (2018) found the opposite. After chronic cold stress, mitochondrial numbers in white fat actually dropped by about 30%. Gene expression related to thermogenesis didn't go up, either (Gao et al., 2018). In short, instead of building more mitochondria, the system backed off. Chronic cold didn't trigger adaptation; it signaled the body to downshift.

Herein lies the golden rule of every hormetic stressor: *it must be intermittent*. First, you spike, and then you retreat. It doesn't work any other way: it's not enough to feel cold, you have to return to warmth after. That contrast is what forges adaptation, and without it, the body digs in, hoards resources, and plays defense.

As easy as it would be to pretend we all handle stress the same way, it simply isn't true. Tolerance to hormetic stressors is not universal, and as such, protocols cannot be, either. Importantly, we must realize that just as different people may respond to a stress in wildly different ways, how any person responded to physiological stressors in the past, especially with different stressors, is not necessarily an indication of how they'll respond to the next addition, or repetition of a stress. It's tempting to believe that because we are strong in one domain, we will be resistant and strong in all.

Perhaps we are metabolically fit, capable of fasting for long periods, and resistant to heat stress. We assume we will also be resistant to the cold or any other stress we have yet to build tolerance towards, but this is a false assumption. Each stress targets different parts of our physiology; it is only our body's response that has overlap. Each stressor needs to be approached with caution, respect, and careful exploration. For some of these stressors, such as cold, this respect is critical; the line between powerful adaptation and serious harm, even death, is a lot narrower than many realize.

How Does Cold Exposure Work?

Cold isn't just a shock to the system, it's a physiological catalyst. You've probably heard about *irisin*, dubbed the "exercise hormone" (I talk about irisin in greater detail in Chapter 3). Irisin is released during physical exertion and plays a role in converting white fat to metabolically active brown fat. It's also been linked to mitochondrial health, glucose regulation, and even neuroprotection. Additionally, some research suggests it may help reduce risk factors for Alzheimer's (Lourenco, 2024; Kim et al., 2023).

Here's where it may get interesting: it's been reported that cold exposure can trigger irisin release more efficiently than exercise (in Chapter 3, I argue that while irisin has been hyped as a key "exercise hormone" linked to fat conversion, bone health, and neuroprotection, the evidence for its effects in humans remains speculative due to unreliable detection methods and overinterpretation of early animal studies. However, human data are limited, and the methods for quantifying irisin are still under active discussion). One study by Paul Lee and colleagues (2014) found that just ten minutes of shivering can burn as many calories as a full hour of steady-state cardio, a metabolic response that isn't accidental or abnormal, but the body's precise and purposeful reaction to cold stress. The rapid, involuntary muscle contraction and relaxation pattern that defines shivering evolved to rapidly increase core body temperature after a significant drop, thereby maintaining homeostasis. Shiver-induced thermogenesis is driven by irisin and FGF21, both of which ramp up your metabolic firepower and energy output (Lee et al., 2014).

This is why cold exposure is sometimes referred to as an "exercise mimetic." In short, it doesn't replace training, but it activates many of the same pathways, particularly those related to mitochondrial biogenesis (Patrick, 2023). What this means is that your cells build more engines, burn cleaner fuel, and produce more power. This isn't just about calorie burn, rather, it's about increasing total metabolic capacity.

Now, while the biohacking crowd has jumped on these cold-induced benefits, including faster recovery, enhanced cognition, and better body composition, most people still reach for the ice for one primary reason: to control inflammation and

reduce pain. From trainers to moms to ER nurses, cold has been a go-to for generations. You twist your ankle, what do you do? *RICE*: rest, ice, compress, elevate. On top of this, it's common practice to use cold packs for bruises and ice baths after tough workouts. By now, it has become folk medicine, but common use doesn't necessarily mean definitively effective. Research on cold therapy for injury recovery is mixed. Some studies show benefits (Malanga, Yang, & Stark, 2014; Block, 2010); others show neutral or even negative effects on long-term adaptation and recovery (Wang & Ni, 2021). One likely reason is that the protocols are all over the place: duration, temperature, and methods are rarely standardized. A cold shower isn't a cryochamber, and a bag of frozen peas isn't a submersion tank.

Cold reduces inflammation, yes, but it also restricts blood plasma flow, and that matters. When tissue is exposed to low temperatures, the body constricts blood vessels to preserve core temperature. That vasoconstriction reduces plasma flow to the skin and extremities, which means fewer nutrients, less oxygen, and slower removal of cellular waste (Young, Sawka, & Pandolff, 1996). In simple terms: healing slows down. That doesn't make cold exposure useless—it just makes timing and dosage critical. Blunting inflammation too early, especially after training, can interfere with the adaptive process you're trying to provoke. Not all soreness needs soothing, sometimes it needs circulation.

This understanding works to pour cold water on the excitement surrounding cryotherapy for athletes wanting to recover faster and gain an edge. As mentioned, the evidence—and our understanding of the physiological response to cold—doesn't always back this claim up. Some protocols might reduce soreness, but others may blunt the very inflammation needed for muscle growth and strength adaptation. This is the tradeoff: cold can accelerate recovery, but it may also mute the signal your body needs to get stronger, defeating the very purpose of faster recovery for many athletes. It's not about "does it work?" It's about when, how, and why you use it. Because even the best tools become counterproductive if you don't know what you're building.

Neurohormesis: What Cold May Do To Your Brain

Like most forms of hormesis, cold exposure has started to catch the attention of mental health researchers. The interest has resulted from possible benefits for mood, stress tolerance, and even depression. Let's be clear: the science is early. Nothing you're about to read should be taken as a replacement for legitimate psychiatric care or prescribed treatments. This is not medical advice, it is cautious exploration. That said, there could be something here. People who consistently winter swim report an elevated mood, improved stress resilience, and even reductions in chronic pain. In one small study, winter swimmers showed improved well-being and pain reduction—though it wasn't blinded, nor could it be, as participants were obviously aware they were freezing themselves on purpose (Huttunen, Kokko, & Ylijukuri, 2004).

Whole-body cryotherapy has shown similar anecdotal benefits in limited trials (Holmes & Willoughby, 2016; Costello et al., 2015), but again, sample sizes are small, controls are loose, and placebo effects are impossible to eliminate. One interesting hypothesis paper proposed that cold showers might help mitigate symptoms of depression via norepinephrine release and peripheral nerve stimulation (Shevchuk, 2008). This is not tested, but it fits what many practitioners report: cold exposure creates a physiological reset. Something about the jolt shifts mood, clears fog, and triggers focus. I've consistently felt this myself. After a brutal plunge, my system doesn't just feel awake, it feels aligned, I feel happy, and importantly, energized and motivated. I have experienced this on days I was feeling down, stressed, and on days I felt like giving up. At times, the effects of a cold shock have felt nothing short of remarkable for me. I don't mistake that for conclusive evidence, but I don't dismiss it either.

Published single-subject case studies have shown promising results—one published in *Biology* documented mood improvements in a patient using cold immersion as part of a self-directed protocol (Yankouskaya et al., 2023). Again, not proof, but another reference point to the potential effects.

Zooming out, hydrotherapy in general has been studied in various forms. The evidence, according to recent reviews, remains likewise inconclusive. Some methods show promise while others don't, and results vary wildly depending on temperature, duration, frequency, and individual baseline (An, Lee, & Yi, 2019; Corvillo et al., 2017; Naumann & Sadaghiani, 2014). Animal studies mirror this complexity, with one experiment showing that acute cold exposure followed by rewarming enhanced spatial learning and memory in rats, alongside activation of key MAPK signaling pathways in the brain (Zheng et al., 2008). Others suggest cold exposure increases norepinephrine—a neurotransmitter tied to focus, alertness, and attention—in both rodents and humans. That seems consistent, but it gets messy quickly.

Take serotonin, for example. It's commonly labeled the "happiness" neurotransmitter, but bump it up artificially in fruit flies, and you get more aggression, not joy (Alekseyenko, Lee, & Kravitz, 2010). Even in humans, meta-analyses show the relationship between serotonin and mood, particularly aggression, is far less clear than most pop-psych would have you believe. One major review summed it up bluntly:

"*Contradictory findings, unreliable measurement, and a high degree of complexity leave our overall finding of a small inverse correlation between serotonin and human aggression open to multiple, equally plausible interpretations*" (Duke et al., 2013).

Here's the takeaway: cold exposure might help with mental clarity, mood regulation, or emotional resilience, but the mechanism isn't fully understood, and the effects aren't universal. What works for one person might do nothing, or even

harm, another. If it helps you feel better, and it doesn't interfere with your recovery or medical treatment, use it, but don't worship it. Cold might sharpen the mind, but it's not a cure.

Body composition & Metabolism

Some of my first and early interest in cold exposure was in its connection to brown adipose tissue conversion or "BAT," also referred to as "Beige Adipose Tissue," which is the process when white adipose tissue, resistant to being burned off for

Picture 8. My fiancée enjoying a hot cup of coffee on a cold day in Switzerland

Some fight the cold with willpower. Some fight it with espresso. No judgement.

energy production, has been converted to a brown-like adipose tissue which easily converts to heat energy. BAT tissue is very easily converted to heat energy via thermogenesis—imagine little droplets of fat in a sack that can slowly convert to heat and be released. White adipose tissue, on the other hand, is like having a giant block of fat in the same sack. The entire block becomes resistant to change. Picture if it was ice: 100 small ice cubes will melt much faster than 1 giant block of ice.

While the mechanisms are very different, the results are comparable enough to use this as a simple analogy. As we get older, we accumulate more and more white adipose tissue, or WAT. This tissue is resistant to 'melting away' into heat energy, and in fact, when substantial WAT is present, often, muscle wastes away first. This leads to the condition often referred to as 'skinny fat,' which is hazardous to our health. We can consider the slow deterioration in body composition from two angles: as we age, we tend to accumulate more white adipose tissue (WAT) and lose brown adipose tissue (BAT), meaning our fat cells become less responsive to being utilized for energy production.

Simultaneously, after age ~30, we tend to lose ~1% of our muscle mass each year, even if we maintain the same routine. This is due to the unfortunate physiological transformation whereby our anabolic potential slowly decreases, while our rate of muscle catabolism slowly increases, leading to a situation where we are slowly losing muscle, even when desperately trying to add it on. At this point, our slow physiological decline is a losing battle, and the best we can do is slow down this slow deterioration as much as possible. In Isaac Asimov's "The Last Question," regarding whether entropy could ever be reversed, or if the universe would eventually die, Multivac repeatedly responded with "There is as of yet insufficient data for a meaningful answer" (Asimov, 1994). Perhaps reversing the deterioration of our own physiology will prove to be a riddle we can solve, however, for now entropy always prevails.

Back to fat accumulation, cold exposure has, in fact, shown to increase BAT activation and non-shivering thermogenesis. In fact, as previously mentioned, it may work by the same methods that exercise does, namely irisin and FGF21 (Pyrżak, Demkow, & Kucharska, 2015), but I will caution the evidence is much better in preclinical models than in human clinical trials. This isn't surprising, as we have 'cured' rodents of obesity more times than I can count. There are literally dozens of molecules that have been shown to dramatically activate thermogenesis in mice or rats. This is compounded by the fact that they (rodents) are resilient enough to deal with the rise in internal temperature from ramped-up thermogenesis.

In listening to Dr. Rhonda Patrick's podcast on cold exposure (and don't get me wrong, I enjoy listening to her show and appreciate her platform as a resource), I was left with a rebuttal to her comments on BAT and UCP1. Namely, humans have dramatically lower levels of BAT than rodents, and uncoupling proteins are far less

relevant to our physiology. On top of that, molecules that drive this response often have high toxicity, and are not tolerable for chronic, or possibly even acute, consumption.

While a 'magic pill' to cure obesity is attractive and exciting, the closest we have come to this breakthrough is a now-banned illegal intervention called 2,4-Dinitrophenol or 'DNP.' Let's get this straight: *DNP works*. You will drop loads of weight. Life will become harder and harder while taking DNP, as your energy expenditure will become very inefficient. Getting out of bed will be a task, and when you do, you will find that you have possibly ruined your mattress from sweat. This is just the tip of the iceberg. It was used clinically in the 1930s, then pulled after it killed enough people to make the point stick. It uncouples mitochondrial respiration so efficiently that your body becomes a heat engine with no brakes (Grundlingh et al., 2011). The same mechanism that burns fat also melts your insides. That's not fat loss, it's self-immolation.

Long-term ingestion of DNP has been linked to the development of cataracts, skin damage, and adverse impacts on the bone marrow, brain, and heart (U.S. Environmental Protection Agency, 2000). That isn't even the worst of it: DNP will lead to death even in low doses, and it is not from the toxicity. It is from the rise in body temperature created by the very uncoupling and thermogenesis that allows it to be so effective. What ultimately restricts the continual increase of DNP dosage isn't insufficient ATP generation, but the dangerous elevation in body temperature caused by the excess heat released during the uncoupling process. As a result, an overdose of DNP can lead to deadly hyperthermia, with body temperatures reportedly reaching up to 43.1 °C (109.6 °F) just before death. Because of this risk, when DNP was used in medical settings, doses were carefully adjusted over time based on individual tolerance, which differs significantly from person to person.

Wow, what a kick in the teeth for anyone wanting a miracle diet pill leveraging thermogenesis, huh? Even if we find one that works in this manner, as it becomes more effective, it will kill you. We are fragile beings, and we need to take this into consideration.

There are peptides now in development—some promising uncouplers with a higher safety ceiling—but we're not there yet. Nothing is both powerful and perfectly safe. Every lever you pull has a cost. Concerning molecules with a direct pharmacological effect, often the more effective the intervention, the higher the likelihood of unwanted side effects.

So no, cold exposure is not a miracle, but it's a tool. For some, it's free, and for all, it can be accomplished affordably. It's accessible, and when used correctly, it contributes to the thermogenic machinery that keeps your engine running clean. I've experimented with dozens of methods, from protocol-driven ice plunges to

experimental dosing with uncouplers. I've read the papers, talked with researchers, and done the n=1 work on my own body.

Here's what I've found: cold exposure works best as a complement, not as a crutch. It doesn't undo bad decisions, it simply amplifies the good ones. It builds capacity when paired with nutrition, strength training, and recovery. It wakes you up—literally and metabolically—and reminds your cells that you are not a passenger in your own body. You are a driver, and the cold can increase your acceleration towards metabolic fitness.

Try it, but don't expect magic. Expect clarity, expect resilience, and expect just enough stress to keep you human.

Conclusion: Cold Showers, Hot Takes

Cold therapy isn't a miracle. It works, as in it drives a physiological response, just not always in the ways it's sold to you. Regardless, cold can become a powerful tool in your toolbox, helping you improve your mental and physical health in the ways that matter, but only if you know what you're looking to improve and understand how to wield the tool.

As I described in this chapter, cold exposure has been shown to activate brown adipose tissue (BAT), stimulate thermogenesis, and trigger the release of FGF21 and irisin, which are two metabolic hormones also associated with exercise (Lee et al., 2014). Most of the data, as usual, comes from rodents and pigs, but there are some interesting, albeit limited, human studies. Like most emerging research in this space, the potential benefits are preliminary, fragmented, and often oversold, as I also mentioned, especially when it comes to claims about fat loss or muscle recovery.

Here's what I've seen in myself: cold doesn't strip fat, and it doesn't cure soreness, but it charges me with energy and focus, like few other interventions are capable. The energy hit I get from a cold plunge, in a way, is stronger than a mega dose of caffeine, but wildly different. After cold exposure, I feel recharged, focused, and have an optimistic positivity about myself that is, at times, rare. It wakes my mind up in ways stimulants are incapable of.

Importantly, cold exposure can be accomplished very inexpensively, even for free. While many prefer to indulge at spas or in expensive plunges, they aren't necessary. I've seen some biohackers modify old chest freezers. Ice blocks in the bathtub are cheap and easy, too. If you live somewhere that experiences cold winters, then walking out your door can accomplish the task. This is why I recommend cold exposure to everyone, provided they understand the dangers and limitations.

CHAPTER 2:

Heat: When the Air Tries to Break You

Ayub Khan, the first military president of Pakistan, once remarked that democracy is incompatible with hot climates, stating

> *You need a really cool and phlegmatic temperament, which only people living in cold climates seem to have. Also it requires a long period of probation. The British took 600 years, and even France, which gave birth to liberal philosophy, has not been able to work it. So don't let us kid ourselves and cling to cliches and assume that we are ready to work such a refined system.* (TIME, 1962)

He was talking about political structure, but anyone who's spent a week in Houston during a heat wave might see his point. Democracy takes work, because when the air itself feels adversarial, even basic cooperation becomes effortful. Thought slows, tempers spike, and willpower melts. The heat doesn't just challenge your body—it taxes your mind.

I don't necessarily agree with Khan's politics, but I understand the sentiment. Heat strips you bare. It reveals how fragile focus and discipline become when your core temperature rises and your brain begins to ration clarity. It's not just sweat, it's friction in every system. While cold sharpens, heat sedates.

Picture 9. Me Hiking at the Grand Canyon

Grand Canyon, spring sun, no stunt doubles.

Picture 10. Me gardening on a nice day

Day 1: Man vs. Jungle.

Day 10: Man 1, Jungle 0

I plan for it now. When I travel to hot cities for work, I don't pretend I'll power through; I build in time to return to the hotel throughout the day to take a cold shower, cool off, and recalibrate. Sometimes I need this respite and recalibration from the heat up to three times a day. It's not indulgent, it's tactical: I've learned that for me, pushing through heat without a plan is more stupid than heroic.

I had to learn that the hard way. Years ago, I was helping construct an R&D facility, intended for my own use in refining the hydrogen tablet technology, on property owned by one of my shareholders. I was installing shingles myself on a black rooftop—under brutal heat, with temperatures north of 40°C (over 104°F). Wildfires in the region made the air thick, the sky dull, and the sun a haze-filtered spotlight that never let up. I was careless and ran out of water, stubbornly refusing to get off the roof until I had accomplished a predetermined, and arbitrary, amount

of the work. This is a personality trait of mine that has caused me issues in other domains of life, which I will discuss in *The Mind* companion of this volume. I was alone, and I got sloppy, dizzy, and weak. Wobbling on a rooftop with no margin for error, it took everything I had just to make it down the ladder in one piece.

I poured cold water, as cold as was possible from the hose on a hot summer's day, over my head for two straight minutes before I could even think, then sat in my car with the A/C blasting for another thirty. When I finally felt okay to drive—one block to a corner store—I bought all the cold water and juice I could carry. That's how close I came to heatstroke, and I'm someone who lifts, who fasts, and who thrives under pressure. None of this matters; heat is its own beast, and it doesn't care about your résumé.

I know the edge, because I've crossed it before. Years ago, on a trip to Cabo, I spent four hours kayaking under the sun, out on the open ocean. There was no shade, and no way to escape for a break. By the time I got back to shore, I felt drained and dizzy. It wasn't until I got back to the hotel that the real issues began. I was vomiting and unable to regulate my body temperature. At one point, I passed blood in my stool. I didn't move from bed for the next four days. That wasn't just dehydration, that was full-blown heatstroke. It was a total *system crash*.

Sometimes people think the line between resilience and recklessness is clear, this is a mistake, and a dangerous one at that. You only know where the line is once you've gone too far, and by then, it's too late. So no, this isn't going to be another glorified chapter on how "hot yoga changed my life." Heat, like any stressor, has to be respected. It can condition you, however, it can also kill you. The difference is never just about exposure; it's about awareness, dosage, recovery, and adaptation. Let's get into it.

My Experiences

As I've mentioned, to the same extent that I've become highly tolerant to cold exposure, I've learned that I'm unusually vulnerable to heat. For a long time, I assumed that was just a quirk, an inconvenience, maybe. The deeper I leaned into understanding hormesis, the clearer it became: *the level of stress a system can tolerate isn't just about overall resilience*, it's also about individual sensitivity to each independent source of stress. If cold barely affects me, perhaps the adaptive components of its hormetic benefit are reduced. Likewise, if heat knocks me sideways, it might offer more opportunity, but only if I approach it wisely.

My history with heat hasn't been mild, as you've seen. My most frightening acute health experiences have come from heat stroke: not after marathons or in extreme environments, but just after a few hours in hot weather, followed by days of confusion, dehydration, and physical shutdown. The memory of those episodes isn't distant, it's visceral.

Yet, at other points, heat has been a powerful ally. In my mid-twenties, I was living in Toronto, training in martial arts—Muay Thai and Brazilian Jiu Jitsu—up to eight hours a day of exercise when including weights, cardio, and drilling for each martial art. Initially, my flexibility and mobility were decent, but incomplete. I could palm the floor, but my hips were tight, especially for the kinds of rotation, dexterity, and control most martial arts demand.

My condo building had both a steam room and a dry sauna. The steam room was a non-starter; I'd get lightheaded within minutes. The dry sauna, though, offered promise, even though it didn't go smoothly at first. While stretching inside, I had one of my earliest blackouts. Five minutes in, my vision blurred to black. I got out in time, but it left a mark. I started setting timers religiously, and once, when I couldn't muster the strength to stand after overambitiously increasing my intended heat exposure, I had to wait for the temperature to fall before crawling to the cold shower. Even so, I kept at it.

There was something undeniable happening: my hips opened up, and my range of motion expanded. Movements that once felt stuck became fluid. I started bringing four-liter jugs of ice water into the sauna, extending sessions to 30 minutes, using the water to stay hydrated and prevent disastrous overheating, while continuing light stretching. A cold rinse afterward left me alert and recharged. My flexibility didn't just improve, it skyrocketed. For me, there seemed to be real, measurable benefits.

Years later, after moving back to Vancouver, I gave hot yoga a try. I assumed I'd be fine; I'd used the sauna regularly, I brought my ice water, I was in shape… I was not fine. The heat of the room hit differently; it was a full-body strain, rather than a passive soak. Within 30 minutes, my water was gone, my vision was tunneling, and I was lying flat on my mat, unable to move. I didn't say anything, I didn't want to, and even if I did, I couldn't. When class ended, I stayed on the floor. Eventually, I made it to the sink, ran my head under cold water, and left. I barely made it a few blocks before I had to pull over and sleep in my car, windows down, head spinning.

Still, I gave it another shot; I am nothing if not stubborn. A friend had found a smaller studio that kept the temperature a few degrees cooler than the chains. I tried ten classes. I made it through all of them, but every single one left me depleted, aching, and stiff. My recovery took a full day. The worst part was that my mobility began to decrease, my joints felt stiffer, and movements took more energy to accomplish. I was at the peak of my fitness, and it still broke me down.

This is the truth about hormesis that often gets lost: the dose matters. Cold, heat, fasting, and exertion—none of them are universally beneficial. What's therapeutic for one person can be destabilizing for another, and your own tolerance may shift over time, even day to day. I talk a lot about resilience and grit, about showing up and pushing through. Resilience isn't about tolerating the maximum stress, it's about finding the right stress: the one that signals adaptation, not collapse. Heat has taught me that lesson over and over, not abstractly but viscerally.

The body doesn't lie, and it remembers. For me, heat therapy requires precision. Cold, I can joke through, but heat, I have to negotiate. More than this, I've learned to listen. Because the cost of not listening isn't just discomfort—it's collapse—and no hormetic protocol intended to drive benefit is worth that.

The Science of Heat Therapy

Heat therapy, what doctors call *thermotherapy*, has been a staple of pain management for generations. This is for good reason: from a heating pad on a tight back to a scalding bath after a hard workout, few things soothe the body as quickly or as reliably, but we're only now beginning to understand that its effects run deeper than temporary relief.

It's not just about comfort, it's about chemistry. Whether it's delivered through dry saunas, steam rooms, infrared panels, or hot yoga studios, heat therapy triggers profound physiological responses. Blood vessels dilate, circulation improves, and muscles relax. Meanwhile, inflammatory markers drop (Brunt & Minson, 2021). Beneath all that, there is a quiet cascade of cellular adaptation: heat shock proteins, mitochondrial shifts, and even hormonal responses that prime the body for recovery and resilience. It's easy, inexpensive, and remarkably safe when used with common sense.

Heat Shock Proteins

Among the many fascinating systems the body uses to respond to stress, heat shock proteins (HSPs) stand out as some of the most vital, and also, the most misunderstood. As the name implies, these proteins were first identified in response to heat stress, but scientists have since learned they react to a wide range of physical challenges: exercise, infection, inflammation, cold exposure, fasting, and more (Singh et al., 2024; Hagymasi, Dempsey, & Srivastava, 2022).

That's because HSPs are fundamentally part of the body's adaptation toolkit. They help us cope with temporary stress by maintaining cell stability and facilitating repair. Like almost everything in biology, though, their effects are not universally good or bad. The same proteins that repair damage can, in the wrong context or at the wrong dose, contribute to dysfunction.

One of the primary roles of HSPs is in protein folding. Proteins don't work straight out of the gate, but rather, they begin as long, floppy chains of amino acids that have to fold into complex 3D shapes to function. Think of them like ropes that need to be coiled and twisted in a precise pattern to become useful tools. When proteins fold incorrectly—whether due to stress, genetics, or poor regulation—they can become useless or, worse, toxic. Misfolded proteins are implicated in a wide range of illnesses, including neurodegenerative diseases like Alzheimer's, certain cancers, and autoimmune and allergic responses (Melikov & Novák, 2024; Hu et al., 2022; Bagola & Sommer, 2008).

Because HSPs help guide proper protein folding, we assume that more of them is usually a good thing. In many cases, this appears to be true: observational studies have shown that individuals with regular exposure to mild physical stressors, including heat, tend to have better outcomes across several health domains (Shan et al., 2020; Nishizawa & Nishizawa, 2018; Tóth, Gombos, & Sántha, 2015).

One of the most compelling pieces of evidence comes from a longitudinal study in Finland, where researchers followed thousands of participants and found that those who used saunas frequently had a significantly lower risk of Alzheimer's and dementia (Laukkanen et al., 2017). Relatedly, a 30-minute Finnish sauna session in a cohort of 100 participants with cardiovascular risk factors significantly reduced systolic and diastolic blood pressure, with effects persisting after 30 minutes of recovery. These responses resemble some of the hemodynamic changes seen during moderate exercise (Laukkanen & Kunutsor, 2024). In other words, heat exposure doesn't just make you sweat; it activates systems of renewal, recalibration, and repair.

This is why heat therapy is being explored not just for cognitive decline, but also for cancer treatment. In medical settings, localized or whole-body hyperthermia is sometimes used as a secondary therapy intended to make tumors more susceptible to chemotherapy or radiation (Kok et al., 2020). While the research is still in development and should not be interpreted as a standalone solution, it represents a fascinating and encouraging frontier.

The results with recovery and injury, however, are more mixed. In one small but interesting study, researchers found that daily heat therapy helped preserve mitochondrial function and reduce muscle atrophy in participants who had been immobilized—an important finding for recovery scenarios (Hafen et al., 2019). Yet other research, including studies on elderly rats, failed to show similar benefits (Kataoka et al., 2017). Some early studies on hot-cold alternation protocols for stroke recovery reported contradictory results or were never published at all, as evidenced by registered clinical trials that yielded no published findings. This is the pattern we consistently observe: heat is a stressor, and like all stressors, its effect is heavily dependent on the context.

There's another layer to the HSP story that deserves attention, because it reminds us that even beneficial processes can have a dark side. Some forms of HSPs, particularly HSP90, have been linked to increased risk of cancer, possibly by contributing to DNA damage or enabling the survival of damaged cells (Calderwood & Gong, 2016). In fact, pharmaceutical companies are now developing HSP inhibitors. HSP inhibitors are drugs and vaccines designed to reduce the activity of these proteins as part of cancer treatment strategies (Karapanagiotou, Syrigos, & Saif, 2009).

Yes, the same proteins your body releases during a relaxing sauna session are also being targeted by anti-cancer therapies. So what are we supposed to do with that

contradiction? The answer isn't to fear heat or abandon stress-based therapies. It's to understand what's happening under the hood. Hormesis, at its core, is about controlled stress for adaptive benefit, but "controlled" is the key word. When a system is pushed too hard, for too long, or without recovery, the same stress that once sparked resilience can begin to drive dysfunction.

For most people, sauna use or heat therapy is overwhelmingly beneficial when practiced in moderation and guided by personal response. Like with all tools, however, the difference between medicine and poison is often the dose. Listen to your body and respect the signal. Perhaps most importantly, don't assume that more is always better.

What Does the Science Say on Muscle Recovery, Pain, and Stiff Joints?

The Promises—and Gaps—of Heat Therapy

When it comes to heat therapy, there's no shortage of bold claims: improved cardiovascular health, reduced all-cause mortality, faster muscle recovery, less soreness, fewer aches. The list goes on. Heat sounds like the ultimate wellness cheat code. That said, once we move past the more grounded cellular mechanisms such as heat shock proteins and their role in stress adaptation, the evidence starts to thin out. That's not to say it's without value. The idea that regular sauna use could reduce cardiovascular disease, or even all-cause mortality, as some large-scale observational studies suggest, is compelling (Laukkanen et al., 2018; Laukkanen et al., 2015). However, we need to acknowledge what these studies are and what they aren't. Most are observational, which means they show correlation, not causation. People who regularly use saunas may also be more active, have better access to resources to maintain their health, or they may simply be more attentive to their health in general. The heat might help, but so might the overall lifestyle.

When we look at rheumatoid arthritis (RA), for instance, the results get even more nuanced. A review of seven clinical studies found that heat therapy didn't produce statistically significant improvements in inflammatory markers, joint pain, or x-ray-assessed joint damage. In a small randomized crossover trial on patients with significant knee involvement, each participant received both ice pack and hot pack treatments on alternate knees, separated by a washout period. Of the 14 participants, 7 preferred cold therapy, 5 preferred heat, and 2 reported no preference, suggesting that most found some form of thermotherapy preferable to none at all, even though objective assessments showed no significant difference between the two modalities (Kirk & Kersley, 1968). These findings highlight how subjective relief and individual preference may matter just as much as statistical significance, especially when it comes to chronic inflammatory conditions like RA.

This tells us something important: just because something doesn't move the needle on lab values doesn't mean it doesn't impact the lived experience. Sometimes,

subjective relief matters, and this is especially true for chronic conditions where patients seek any form of comfort.

Pain is a very real physiological response, but it is also a subjective experience. If patients believe they are better off with a therapy that isn't actually moving the needle on their biomarkers, they may still be able to reduce the intake of harmful anti-inflammatory drugs or illicit substances used to dull the pain. That is not a small win; it is a significant victory that could lead to long-term implications in improved health.

Heat's reputation as a recovery tool for athletes and regular exercisers is also up for debate. Some studies suggest it's more effective than cold therapy for reducing delayed onset muscle soreness (DOMS) and enhancing blood flow and tissue flexibility (Malanga, Yan, & Stark, 2014). That's certainly been my experience, especially when stretching in the sauna post-training. The difference in mobility and perceived tightness has been obvious. When broader reviews are conducted, the tone shifts. The data, so far, isn't strong enough to confirm heat therapy as a consistent or superior approach to post-exercise recovery. The evidence base is simply too sparse. Promising, yes, but far from definitive.

What does that mean in practice? It means exactly what much of this book argues: use your body's feedback as your first metric while you wait for the science to catch up. In the meantime, if you're finding value—real, repeatable benefit—from heat, that's a data point worth keeping. Just don't confuse possibility with proof, and don't assume what works for you will work the same for everyone. We're still learning. That doesn't mean we shouldn't act, but it does mean we should act with awareness.

Conclusion: Heat, Held Lightly

Heat therapy is a powerful but often misunderstood tool. It's not a miracle cure, but when used intentionally, it can contribute meaningfully to a well-rounded health regimen. For athletes, whether elite or recreational, it offers a low-barrier method to enhance flexibility, support recovery, and potentially improve performance. The key, as always with hormesis, is dose. Mild stress can stimulate growth. Push it too far, and the benefits reverse. What starts as helpful can become harmful.

Heat therapy, like any intervention, works best when it's part of a broader system, not a silver bullet, but one arrow in the quiver. Used wisely, it can help the body adapt, repair, and even relax. Used recklessly, it becomes just another stressor tipping the scale.

So if you're going to embrace the heat, you need to do it with awareness and respect your limits. Watch for warning signs, start small, track how you feel afterward, and always remember that the goal isn't to prove your toughness. It's to build resilience that you can carry into the rest of your life.

CHAPTER 3:
Exercise As Stress: A Controlled Burn

Martial arts training looks much different for me these days. In the past, I'd enter the gym and feed on the electricity. I'd see dozens of bodies, testosterone permeating the air, hear the crack of strikes on pads, or on people themselves, the cheers and laughter—all of it, creating an energy that any martial artist can immediately visualize. What it didn't create, however, was exactly what I realized I needed: calm and clarity.

As my body aged and threatened to break down, I learned that I could no longer rely on my physical gifts. Day by day, I needed to lean on my mind, my creativity, and my instincts. The question became: how do I intentionally develop instincts which defy expectations—mine or anyone else's? The creativity that existed within me was born from chaos; it was forged over a decade of hard sparring, built by shots that I would find in frantic exchanges, which were improvised but wholly effective. Hard sparring with younger, fitter, and more durable training partners becomes less and less appealing as you approach and get into your 40s; therefore, to expand my strength and creativity, I needed a creative solution. My coach and I devised a plan, which we now refer to as theory days. Theory days start slowly, primarily with exploration.

When we first started this practice, my coach, a seasoned southpaw, would lead the dance. Now, I often lead, but not because I am the better striker, simply because I am the better visualizer: I'd run through exchanges in my head—exchanges that he and I, or myself and others, frequently get into—exchanges which I get the worst of. In slow motion, we'd run through these exchanges, and I'd make changes until something worked. I would run through my pattern, and then the next time he would change and run through his counter-pattern, devising a plan against whatever strategy I had concocted, thinking about how it could be baited, then exploited. Once we found something that maximizes my ability to surprise and land cleanly, minimizing the potential for damage, we began drilling.

When the combinations get weird and unorthodox, even figuring out how to flow through the padwork takes some thought. Holding the pads at the right angle, moving in a manner to match the combination, and, as the striker, ensuring you aren't missing and hurting your partner are all critical considerations. This is all necessary, as my strikes don't always have names; they don't follow expectations, bending trajectory, breaking rhythm, and cutting from odd angles. They're hybrids that I've stitched together from instinct and experiment. Sometimes I switch stances mid-combination, sometimes even two or three times in a row. I don't do this to imitate high-level pros; I'm forced to do this to cut angles, alter distance, and to achieve this within the confines of having one working arm to throw punches with.

Importantly, when I realized that this forced style could work, I began to use it to disrupt. As stated, changing stances can change the dynamics substantially, maybe allowing you to take an unexpected angle or close the distance faster than expected. Sometimes I fake a stance switch—just enough to sell it—then snap back to my original position without ever fully shifting my weight, exploding into a strike before my opponent can register the reset. The big question is always: *what happens when you break the blueprint and keep moving*?

When you start speeding up theory into real-time exchanges against a game opponent, it becomes something else entirely, a kind of embodied game theory, like a physical version of the Prisoner's Dilemma: do you cooperate, or do you defect? Do you play safe, or betray early? Every feint, shift, or misstep is a micro-decision under uncertainty. In sparring, just like in life, you don't know exactly what your opponent will do. You only know the incentives you've created and the pressure they feel. Theory days were all about designing traps—building decision trees in real time, with your body instead of a pencil, starting in slow motion and building to throw my full speed and power by the end of the session.

This was all about subverting rhythm, but it involved cementing a form that was open to experimentation. Good striking has structure: clean, simple, even beautiful in its fundamentals. Great striking, on the other hand, is more like *jazz*. It disrupts expectation, it plays with timing, hides intention, and throws from angles no one's ready for: a strange strike here and a stuttered tempo there. It's calculated deception, you learn the rules so you can break them just enough to stay unpredictable. If a new idea didn't hold up in motion, my coach would shut it down quickly, exposing its weakness and watching it collapse under pressure. Sometimes my ideas worked: sometimes a move no gym would ever teach became a weapon,[4] because we'd built it, step by step, in that quiet room where ideas hit just as hard as fists.

This wasn't just fighting but *creativity* in motion. I was using the same brain that could build a strategy for scaling a business or breaking down a scientific paper, now rerouted through muscle memory and instinct. Imagination wasn't suspended in the gym. It was amplified; the mind *had* to be there, fully engaged, inventing possibilities in real time.

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[4] One gym might be the exception. A new team out of São Paulo, the Fighting Nerds, has been making waves by doing exactly this kind of offbeat, high-IQ fight innovation. Founded in 2014 by Caio Borralho and Pablo Sucupira, they rewrote what fight culture could be. Instead of pretending to be street tough, they embraced being nerds: mathematics tutors, ex-copywriters, thinkers first, brawlers second (King & Subhan, 2024). Their fighters wear taped-up glasses into the cage (with UFC approval), not as a gimmick but as a badge: a symbol of mental agility in a sport that too often glorifies brute force. Their whole philosophy is that fighting is a puzzle to be solved. They build gameplans like engineers, and they're starting to prove that weird works. As of 2025, they're expanding into the U.S., which means the secret's out.

That's the part people miss when they think about physical training: they treat the body like a brute machine, though the best athletes, the ones who evolve, don't just push their limits, they think with their fists. *They problem-solve in sweat*. Finally, they blend aggression with analysis, and it's in that blend, that synthesis, where real growth lives.

Exercise is arguably the most powerful tool we have for long-term health. If you're physically able, in any capacity, you should be doing some sort of physical exercise. It doesn't matter if it's weights, hiking, swimming, martial arts, or pushing a sled in your backyard. The exact routine is less important than the fact that you're training your system to handle stress, because that's what builds resilience.

Here are some topics to avoid if you want to keep the peace: religion, politics, and, in today's day and age, you can add on "healthiest diet" and "best form of exercise." While I have no problem wading into any of these debates, my thoughts on which form of exercise is best are likely to disappoint most and truly enrage none. Many forms of exercise have their purpose, but none are the best for everyone, *all the time*. That goes for CrossFit, Marathon running, bodybuilding, or any other form imaginable. What I'm interested in is how exercise functions as a hormetic stressor: how different types and intensities of movement trigger different adaptive responses across the body, especially hormonally.

Doug McGuff and John Little nailed it in *Body by Science* when they said: "*Fitness is a state that lacks a precise definition*" (McGuff & Little, 2009) They're right: everyone talks about "getting fit," but no one agrees on what that actually means; not the fitness industry, not the medical world, and definitely not the average person scrolling through Instagram. You won't find my advice on which workout is best, nor will you find any specific protocols to accomplish certain goals. What you will find is the science behind exercise, and some important considerations for when you are developing your own fitness routine.

PGC-1α and Irisin: Signals of Adaptation

Let's talk about two players often brought up in the conversation around exercise and cellular adaptation: irisin and PGC-1α. The theory goes like this: when you exercise, PGC-1α (a regulatory protein involved in energy metabolism) ramps up in your muscle tissue. That ramp-up supposedly triggers the release of irisin, a myokine some have dubbed the "exercise hormone" (Chen et al., 2016; Boström et al., 2012).

Myokines, if you're unfamiliar, are signaling proteins released during muscle contraction. They play a role in how your body communicates stress and triggers an adaptive response. That said, there is an important consideration: the implications of irisin on our physiology, despite all the hype, remain on shaky scientific ground. What erupted into the scientific consciousness with excitement quickly turned to questions, uncertainty, and skepticism. When his team discovered irisin in 2012, and

named it after Iris, the Greek goddess that sends messages between gods and humans, Bruce Spiegelman foresaw the role of irisin as a 'messenger,' stating, "*There has been a feeling in the field that exercise 'talks to' various tissues in the body. But the question has been, how?*" (Boström et al., 2012). Spiegelman believed that irisin was an important first step in understanding the biological mechanisms that translate physical exercise into beneficial changes throughout the body, both in healthy people and in preventing or treating disease.

While not declaring elevated irisin as a panacea, as some fitness influencers would have you believe, the team hypothesized that when you train, your body releases irisin, and that irisin tells other tissues, such as fat, bone, and brain, how to adapt (Boström et al., 2012). It was an elegant theory, and to be fair, several studies conducted since then have demonstrated potential links between irisin and positive outcomes, including fat conversion, bone health, and even brain resilience (Fessl, 2021; Colaianni et al., 2015). Let's be clear, however: most of this research is either in mice, cell cultures, or using detection methods we still don't fully trust.

It was thought that one proposed benefit of irisin is that it may help convert white fat (as detailed in Chapter 1, the stuff most people want to lose) into brown fat (which burns energy) (Boström et al., 2012). That could be useful for weight regulation, and it's been a selling point in a lot of pop-science circles, but I'm cautious, much more than most anti-aging types hyping it up on podcasts. I've also seen a 2018 study showing that irisin binds to receptors on osteocytes, the bone-maintaining cells, hinting at possible applications for osteoporosis (Kim et al., 2016). Again, these reports are based on mouse models, making them promising at best but not yet clinically actionable, at least not for these precise circumstances.

Then there's the neuroprotective angle. A 2019 *Nature Medicine* study found that irisin might protect against synapse failure and memory loss in an Alzheimer's model, but again, the research was in rodents (Lourenco et al., 2019). It makes sense mechanistically, since irisin is downstream of PGC-1α, and PGC-1α is tied to mitochondrial biogenesis, which is critical for brain health. Here's the catch, however: correlation doesn't necessarily mean causation, and we still lack a clear line of evidence connecting irisin to outcomes in actual humans. That hasn't stopped media outlets and influencers from selling it like a miracle molecule. To their credit, *The New York Times* handled it with more caution than most.

Instead of framing irisin as a miracle cure, *The New York Times* emphasized that the findings were preliminary, limited to mice, and part of a broader scientific inquiry into how exercise supports brain health (Reynolds, 2019). It acknowledged the allure of a molecule like irisin while resisting the urge to overpromise. That kind of restraint—highlighting the promise without discarding scientific humility—is what made the piece stand out for me amid a sea of hype-driven coverage.

That said, the cautious optimism around irisin hasn't stopped researchers from probing deeper into what triggers its release, and what they've found is starting to map a clearer picture. Some studies suggest that circulating irisin levels go up during low-intensity aerobic exercise (Jandova et al., 2021). However, other research shows more robust increases following high-intensity workouts, particularly HIIT and resistance training, when compared to non-exercising controls (Haghighi et al., 2022; Huh et al., 2012). Direct comparisons also support this distinction: high-intensity exercise produces a greater irisin response than low-intensity activity when energy expenditure is matched (Tsuchiya et al., 2016), and resistance training appears to elevate circulating irisin more effectively than aerobic training in overweight or obese adults (Kim et al., 2015). In head-to-head comparisons, strength training and high-intensity protocols appear to have an edge over steady-state cardio in terms of irisin (Ma et al., 2021).

In 2021, a systematic review was published that increased skepticism about all of these findings. The researchers elucidated on why our methods for detecting irisin are questionable at best. The researchers basically said: we can't confidently say irisin goes up with exercise, and we also can't say it doesn't (Jandova et al., 2021). The evidence just isn't strong enough, mostly because the tools for measuring it aren't up to par. It's important to note that shortly after they made their discovery, Spiegelman's team claimed they *could* quantify irisin in blood samples (Jedrychowski et al., 2015). That was several years before the 2021 review came out demonstrating the tools to do so are not robust enough; so the debate isn't settled—far from it—but you wouldn't know that if your only source was one of those pop-science dopamine farms like "I Fucking Love Science," who love to turn soft hypotheses into clickbait certainties.

Here's the bottom line: irisin *might* go up with exercise. I think it probably does, but either way, the effect has been wildly overstated by bloggers, influencers, and even a few well-meaning but overconfident doctors in the anti-aging space. Until we have better testing and more consistent data, it's speculation dressed up as science. Don't chase irisin, chase adaptation, and the rest will sort itself out.

What Does Increased Expression of PGC-1α Mean?

PGC-1α gets a lot of attention, and for good reason. It's often called the "master regulator" of mitochondrial biogenesis, which is a fancy way of saying it helps your cells build more mitochondria. That matters because mitochondria are where your body makes energy. More mitochondria generally means better energy metabolism, better endurance, and better resilience under physical stress (Halling & Pilegaard, 2020).

Mitochondrial decline is strongly linked to aging and age-related diseases such as heart disease, diabetes, and neurodegeneration (Srivastava, 2017). So naturally, people assume that ramping up mitochondrial production must be the key to

reversing aging. Maybe, but let's not pretend it's that simple: while boosting PGC-1α seems promising, we don't have strong evidence that doing so on its own radically extends lifespan or even health span. It's likely one important piece of the puzzle, but not the whole picture.

Myokines and the Interesting Role of IL-6 as an Anti-Inflammatory

Like irisin and PGC-1α, another myokine called IL-6 is a signaling molecule released during muscle contraction. It's a fascinating one because it flips the script on what we normally think of as inflammation. In disease models, IL-6 is a red flag. Chronic elevation is associated with a range of conditions, including cardiovascular disease, frailty, and accelerated aging (Mehta, DeGoma, & Shapiro, 2025). It's one of the biomarkers most anti-aging researchers want to keep low.

As always with our physiology, context is everything: when IL-6 is released acutely from muscle during exercise, it doesn't act like a typical pro-inflammatory cytokine. It actually triggers a cascade of anti-inflammatory effects, including the activation of other anti-inflammatory cytokines, such as IL-10. Some studies show serum IL-6 levels can spike more than **100-fold** after exercise, and instead of causing damage, this surge promotes repair and resilience (Pedersen & Febbraio, 2008; Fischer, 2006).

The key is *where* and *why* IL-6 is being released. If it's coming from muscle tissue in response to a short, controlled bout of physical stress, it signals adaptation. Yet, if it's coming from immune cells, such as macrophages, as a result of your body being under chronic stress, it contributes to long-term inflammation and degeneration. In short, it's the same molecule, with completely different outcomes depending on the context.

Exercise, Vasodilation and Nitric Oxide and VO_2 Max

Nitric oxide (NO) is one of those molecules that was misunderstood for decades, and for a very specific reason. It's a free radical, which means it has an unpaired electron and is highly reactive; free radicals lead to cellular and tissue damage. For years, knowledge about the deleterious effects of free radicals has led to firm and uncompromising messaging: *free radicals are always bad, and the best practice is to eliminate them from our bodies*. Uniform messaging regarding unambiguous harm in the 1980s and 90s created a singular villain: oxidative and nitrosative stress were the main culprits in cancer, cardiovascular disease, and even aging itself. As such, anything that contributed to that stress, like free radicals, was branded as harmful (Sies, 1997).

However, NO broke that mold; it turns out this so-called troublemaker was doing vital work in the body, relaxing blood vessels, enhancing oxygen delivery, modulating neurotransmission, and even supporting immune function (Moncada & Higgs, 1993). The early bias came from a chemical truth taken out of a physiological context. Yes, NO is reactive—but in the right dose and in the right tissues, that reactivity becomes beneficial signaling.

The initial misunderstanding gave rise to an entire industry built on antioxidant hype, which has clung on to relevance long after the science has been largely refuted, stubbornly continuing to grow and expand. The misunderstanding fails to understand the nuance of NO, because, like most things in biology, the truth is complicated. NO is reactive, but it's also essential; it is, in a word, *hormetic*. In recognition of the revelation of the hormetic nature of NO, the 1998 Nobel Prize in Physiology or Medicine was awarded to the team that discovered its critical role in cardiovascular signaling (Nobel Prize Outreach, 2025).

Exercise reliably increases NO production. That's one of the ways it improves circulation, lowers blood pressure, and boosts performance, especially in untrained or moderately trained individuals. That's also why supplements like citrulline, arginine, and beet-derived nitrates have taken off in the athletic world. Here's the catch, though: if you're already highly trained, these supplements have less of an effect, as your system's already close to optimized (Arefirad et al., 2022; Jones, 2019). You may not get the same bang for your buck, but that isn't to say supplementation is wholly useless. Even small changes can have downstream trickle effects that improve your overall vitality.

To understand why exercise, and to a lesser extent supplementation, can have such a powerful impact, you have to zoom in on nitric oxide's central role in vascular function. NO is a key driver of vasodilation, which is the widening of blood vessels that facilitates more efficient blood flow throughout the body. When NO production is impaired, your vessels stay constricted, your blood pressure climbs, and your cardiovascular risk increases. Exercise, predictably, helps with this. In fact, it's as effective as many first-line drugs when it comes to lowering blood pressure (Naci et al., 2019). That alone should tell you how potent movement is as a therapeutic intervention.

VO_2 Max: Capacity Is Not Just About the Lungs

On to VO_2 max, which is essentially your body's maximum oxygen utilization during intense exercise. The higher your VO_2 max, the more aerobic work your system can sustain. We know that regular training, especially at higher intensities, can increase VO_2 max, particularly in people starting from a lower baseline. The increase in VO_2 max does level off quickly, and intensity is the lever that matters most. Casual activity helps, but it won't push VO_2 max much past your genetic set point without challenging effort (Scribbans et al., 2016).

What's interesting is that while VO_2 max declines with age, the decline doesn't seem to come from your lungs, or your ability to *take in* oxygen, it comes from the muscles' ability to *use* it. That's why muscle loss (not aerobic capacity) is often the limiting factor in aging athletes. In fact, master athletes maintain their VO_2 max at nearly double the rate of sedentary people their age. While nitric oxide can enhance oxygen delivery, an increase in VO_2 max doesn't necessarily boost NO levels

(Shannon et al., 2022; Betik & Hepple, 2008). As with most things in physiology, harmony, not overload or suppression, is the goal. More isn't always better, and neither is less. Maintaining homeostatic function through movement? That's everything.

The Case for Caution on IGF-1

IGF-1 is a hot topic in the biohacking world. You'll hear it mentioned on podcasts, in Reddit threads, and by plenty of anti-aging doctors as either the holy grail or the hidden villain, but here's the honest answer: we don't really know what to do with it yet. There are two camps: one thinks boosting IGF-1 is the key to growth, regeneration, and long-term vitality, while the other sees elevated IGF-1 as a fast track to cancer and metabolic disease. Both make strong claims, and both have data to back them up, but none of the data is consistent. The more we study it, the more contradictions emerge. That tells me we're dealing with something far more complex than a simple "up is good" or "down is better" scenario.

Personally, I'm not convinced that chronic manipulation of IGF-1 in either direction is wise. There are real conditions, on both ends of the spectrum, linked to its dysregulation. Until we have clearer data from large-scale human trials, nobody should encourage people to experiment with it directly. We don't even have enough clarity to *debate* the pros and cons intelligently yet. Even once we have this large-scale data, individual needs may differ drastically from the averages. We are simply too far from understanding this subject to offer guidance.

What we do know is this: IGF-1 tends to decline with age. That may be a factor in aging, but it may also be just a symptom. We also know that many hormetic stressors, such as fasting, cold exposure, and resistance training, seem to influence IGF-1 levels, up or down, depending on the context. We also know that attempts to raise it through direct supplementation or administration in clinical trials haven't panned out. Results have been inconsistent, and the benefits, if any, have not been reliably demonstrated in humans.

So, where does that leave us? Probably in a similar place to inflammation or reactive oxygen species (ROS). These aren't enemies; they're functional signals that're necessary, even protective, in the right amounts and contexts. IGF-1 might be the same. Until we know more, chasing it directly isn't smart, but if your training or hormetic practice modulates it as a side effect? That's fine. Let it regulate itself through process, not force.

Heat Shock Proteins and the Biology of Stress

As I mentioned in the last chapter, despite the name, heat shock proteins (HSPs) aren't just a response to heat. They're triggered by a wide range of stressors: exercise, cold, toxins, and fasting, just to name a few. They show up in almost every major

topic we cover in this book (Ghosh et al., 2018). Think of them as your cells' emergency response team: they kick in when things get unstable.

As I also mentioned, like most adaptive mechanisms, HSPs aren't inherently "good" or "bad." That kind of black-and-white thinking doesn't hold up in biology. Nearly every major process in the body, such as oxidation, inflammation, and immune signaling, has dual roles depending on the dose, timing, and context. HSPs are no different: sometimes they help, sometimes they hurt. Since we still don't fully understand all their downstream effects, the best we can do is examine overall health outcomes and make informed decisions based on what the data *actually* supports, not what we wish it said.

Exercise, being a stressor, naturally boosts HSP production, and here's where it gets interesting: the same stress that raises HSPs also reduces risk for diseases associated with misfolded proteins. Regular training, especially involving leg strength and resistance, has been linked to a lower risk of neurodegeneration, sarcopenia, and even some forms of cancer. It promotes neurogenesis, improves post-stroke recovery, and is often recommended during cancer treatment (Feng et al., 2024; Tan, Tan, & Chung, 2024; Veldema & Jansen, 2020; Pinho, Aguiar, & Radák, 2019). Particularly, resistance training was associated with lower risk of bladder and kidney cancers (Rezende et al., 2020). Aerobic movement can literally help your brain rewire after trauma.

So what should you do with all this? Continue training, but avoid overtraining. Exercise is one of the best things you can do for your long-term health, but it's still a stressor. Like anything hormetic, dose and recovery matter; too much of a good thing can flip the script and turn a signal into damage.

When Exercise Backfires

Exercise is a significant physiological stressor, and that's what makes it effective for so many health-related indications, but it's also what makes it dangerous when misused. Like any hormetic input, there's a threshold. Push too far past it, too often, and the same thing that builds resilience starts tearing the system down. I'm not saying exercise is as destructive as alcohol or drug abuse, but if you overdo it long enough, the outcomes can look surprisingly similar, especially in a culture that glorifies exhaustion, pain, and "no excuses" grind as virtue.

Overtraining, whether chronic or from an acute blast of intensity in someone unprepared, can lead to a cascade of problems: chronic inflammation, fatigue, hormonal disruption, sleep loss, and ironically, reduced performance (Cadegiani & Kater, 2017; Eichner, 1995). This happens in women who suddenly lose their menstrual cycles after ramping up training too fast. This happens to men who pride themselves on never missing a workout, even when their recovery is compromised and their injuries accumulate. When you push past the beneficial adaptation into

chronic stress, your routine is not discipline anymore—it's compulsion. I've been guilty of this compulsion myself, too many times to count, in the past.

The most serious version of this is rhabdomyolysis. It's rare, but it's real, and it's rising. It happens when muscle tissue starts breaking down so fast it floods the bloodstream with cellular debris, stressing the kidneys and risking organ failure. It used to be a condition only seen in car crash victims or soldiers carrying loads through heatstroke. Now it's showing up in fitness enthusiasts who can't stop pushing (Hopkins et al., 2019), and I've personally come close myself.

In my late 20s, I entered a local two-day CrossFit competition just days after getting over food poisoning. That acute stress, stacked on top of chronic overtraining (4–6 hours a day, five to six days a week), wrecked me. I couldn't walk properly for days, and developed gastroenteritis. Two months later, I had an autoimmune-like reaction to a virus that changed the course of my life.

On some level, that breakdown is what led me here. If I hadn't gone through it, I probably never would've started researching molecular hydrogen, I never would have created the hydrogen tablets, and I never would have written this book. It would be easy to believe that this catastrophe was the best thing that ever happened to me, and to look down on those who have let their collapses permanently break them. Let's be honest: most people who get knocked down like that *don't* bounce back, and my trajectory was as much luck as it was any innate characteristics of my being. Most people who face catastrophe and collapse don't invent something; they just get hurt, permanently. To romanticize that kind of collapse as "necessary" is delusional. It's the phoenix myth, and it's mostly fiction.

I used to believe in the phoenix myth; that if you push hard enough, train until you collapse, live at the edge of breakdown, you'll rise again, stronger and transformed, worthy, even. In reality, that's not what happens. At least, not always, and not usually.

What usually happens is: you get sick, your immune system gives out, you injure your gut, your joints, and your brain. You lose things, sometimes permanently, sometimes irreversibly, and for what? For a story: a story that collapse is part of becoming, that the flame that wrecks you also refines you. That's the lie.

It's also a beautiful one. Edna St. Vincent Millay captured it in four perfect lines:

> *My candle burns at both ends;*
>
> *It will not last the night;*
>
> *But ah, my foes, and oh, my friends—*
>
> *It gives a lovely light.* (Millay, 1920)

She's saying: yes, I'll burn out, fast and gloriously, and everyone will watch. That's the trap, because the candle may give a lovely light—for everyone else—but you're the one left in the dark when it's gone.

The fitness industry needs to reckon with this. When rhabdo starts showing up in weekend warriors, something's gone wrong. The goal isn't to survive your workouts, it's to benefit from them. You're not supposed to break yourself to get healthy. "Hardcore" doesn't mean "smarter"; most of the time, it just means uninformed.

Conclusion: Train to Adapt, Not to Break

Exercise might be the single most important thing you can do for your long-term health. There's no pill, no supplement, and no surgical shortcut that comes close to what consistent, moderate physical training can offer for your lifespan and healthspan. Though that doesn't mean more is always better: exercise is still a stress, a controlled assault. You break yourself down in the hope that your body builds back stronger.

Push too far, too often, and the benefits reverse: the signal turns into noise and your repair system gets overwhelmed. That's why the obsession with grinding harder, chasing arbitrary metrics, and never missing a workout isn't discipline, it's dysfunction. The goal isn't to punish yourself, it's to train in a way you can sustain for decades.

So move: lift, sprint, stretch, do what you enjoy, but stay honest about what serves you long-term. Recovery is part of the work, rest is part of the plan. Adaptation doesn't happen when you train—it happens when you recover from training. So don't just train hard, train smart.

Picture 11. Me eating a giant pizza

Fast hard, feast harder.

CHAPTER 4:

Fasting and Time-Restricted Eating

Introduction: Fasting as the Art of Refusal

Confidence is commonly viewed as the absence of self-doubt and insecurity. Logic follows that if one is confident, they must be certain of their abilities, and of all topics they venture to speak on. Following the train of thought of this logical fallacy quickly spirals into the presence of confidence and certainty being falsely attributed to competence. The reality is that the first two "C's" rarely lead to the latter: namely, unwavering confidence and certainty almost never are signs of demonstrable competence. What is falsely labeled as competence is better described as *delusion*: an inability to reflect on one's own shortcomings. This lack of introspection and self-awareness will almost certainly inhibit growth and impede the quest to flourish.

The thing about growth, when it comes to the substance of who we are, is that it needs that introspection, that doubt, and that insecurity to be initiated—and then propelled forward into progress and achievement. We may deceive ourselves and others about our true motivations, but growth is only achieved through a healthy dose of doubt, with a healthier dose of motivation, completed by the knowledge and will to act.

The confidence with which I write about fasting comes from the ironic, but expected, insecurity that led me to the knowledge I possess. After my health crisis, which I described in the Preface, I buried myself in my work. I clocked over 100 hours of working a week, split between travel for a former business, and obsessive R&D on the hydrogen tablets. This left little room for exercise and meal prep; so my waistline grew, my health markers plummeted, and my confidence in myself, and my appearance, hit rock bottom.

First, I tried to go on a diet and start an exercise program. I slowly decreased my calories until they were below 2000 a day. This strategy may have further harmed my goals, as the low calorie intake led to decreased energy. The previous years of abuse—late-night pizza delivery as my only meal of the day, washed down with red wine—had certainly had a deleterious effect on my metabolism.

I kept struggling, and I kept gaining weight. I was deep into metabolic syndrome, and my clothes began to tighten—for the 4th time post-health scare. Tipping the scales at 267 pounds didn't trigger me to make a change, but facing down the possibility of admitting defeat and buying a new wardrobe for the 4th time in 2 or 3 years did. Moving from a medium to a large to an XL and finally an XXL all hurt my ego, but the jump to a XXXL, and likely needing to shop at stores for large men, was too big of a pill for me to swallow.

I was already wearing the weight: everyone I worked with could see that I was in metabolic distress. I knew this and was deeply insecure about it. It made me cautious to discuss my knowledge on the research of various health and metabolic fields; after all, how could someone like me, with the health decline I was on, have the audacity to tell others how to improve their health? Another size up wouldn't change this dilemma, but it did aggravate and inflame my impostor syndrome—as relating to my perceived place in the industry—to the forefront of my consciousness. This situational insecurity was the catalyst I needed in order to motivate the change I needed.

My vanity and insecurity required a poetic solution, one which could improve my image while simultaneously improving my health. (Quick digression; I do not have the slightest concern about what people think of me *if* their disparagements are inaccurate, or based on actions or behavior I intended to execute, for calculated purpose. I do care, deeply, if I agree with the criticisms. Regarding my health and girth, I cared, and agreed).

I found a solution in fasting. When I started my fasting protocol, it was just beginning to emerge as a popular tool in the biohacking community. This allowed me to talk about what I was doing, become excited about it, and publicly state I was doing it. This public declaration, no matter how small the audience I was speaking to at the time, forced me to commit. So, commit I did, and the results were better than expected (as I detail shortly).

Nothing that follows is meant to evangelize fasting, or even suggest it will work for any given reader at any given moment in time. We're surrounded by dietary evangelists these days, and I have exactly zero aspirations to be one myself, or advocate for any of them. Scroll through social media, glance at a headline, and wander past the supplement aisle, and you'll find no shortage of protocols: each one confident, and most of them contradictory. Everyone's got the answer, and everyone's a guru. Unfortunately, the ideal diet isn't universal, and there is no one-size-fits-all approach. The best diet for you will depend on a number of factors, including your lifestyle and activity level, genetics, and microbiome. What worked for you yesterday may not work for you tomorrow. Fasting worked for me, for a time. I'm simply sharing my experience, along with what the science says it may assist with, what the limitations are, and the trade-offs I experienced in my protocol.

There is some genuinely interesting research behind many of these trends, particularly around calorie restriction, fasting, and the so-called "fasting mimicking diet." However, I wouldn't confuse scattered studies with settled science. The benefits you'll hear breathlessly touted, such as longevity, autophagy, mental clarity, and near-magical fat loss, can and do quickly cross into fantasy, and while the scientific foundation isn't entirely made of sand, it's definitely still under construction. Much of the data is preliminary. Some of it, frankly, is questionable.

Let's make something clear up front: this is not a chapter about the ketogenic diet, which was all the rage a few years ago and still has its proponents today. This is a chapter about *metabolic flexibility*—the ability to shift smoothly between different fuel sources depending on what your body needs. Metabolic flexibility is one of the most important hallmarks of metabolic health, and one of the most neglected. When you're metabolically flexible, you can burn fat when food is scarce, use glucose when performance demands it, tap into glycogen stores during a sprint, or produce ketones during a fast. This is less about being in any one state and more about the ability to move between states.

Ketones will come up, I'd be remiss not to mention them in any discussion of fasting. That said, I'm not here to sing the praises of permanent ketosis. There is evidence that short-term ketosis might confer metabolic and cognitive benefits for certain individuals under certain conditions (Altayyar et al., 2022; Walsh et al., 2021), but living in a chronic ketogenic state? That's another matter entirely. Here's what we know from the lens of hormesis: permanent stress isn't adaptive, it's corrosive, even if that stress comes with coconut oil and MCTs. Studies have shown that while a high-fat ketogenic diet can offer metabolic improvements in the short run, those benefits often plateau—or worse, reverse—when it becomes a long-term lifestyle (Gallop et al., 2024). What starts as a therapeutic edge can dull into dysfunction.

Instead, what I advocate—strongly—is metabolic flexibility: the ability to shift, adapt, and cycle through macronutrient profiles, to move between caloric restriction, maintenance, and yes, the occasional surplus (because, let's face it, life happens). Metabolic flexibility isn't sexy, it's not a brand or a buzzword, but it's what your body is designed to do. It's what we lost, and what we're trying to get back.

3-5-Day Fasts

I've been watching the fasting craze evolve for nearly a decade now. It started with curiosity, it turned into discipline, and eventually, it turned into data.

The first time I attempted a true water fast was six years before I wrote this book, in the spring of 2019. I documented the entire thing—not just what I did, but how it felt. Here's what I wrote at the time:

> *I just completed a 3-day water fast, kickstarting it with my last meal being a high fat, moderate protein intake following a soccer game. I worked out every day during the fast to maximize benefits. My experiences were very different from what I read regarding expectations, and I have considered some of the reasons behind this. During the fast, I consumed only water, lots of it, a multi-vitamin/mineral, and twice daily high dose hydrogen water generated by my patented hydrogen tablets. I will note that the hydrogen water intake led to acute, and substantial, increases in physical and mental energy, but only for an*

hour or two. During this fast I learned to "save" my hydrogen water dosages for when I was experiencing great difficulty.

First off, it was not one of the hardest things I have ever done. I don't think it would make the top 100. This was perhaps the biggest surprise to me. I woke up the first morning hungry, and then subjected myself to a hard workout early morning day one, and after that, the 16-24-hour mark was by far the hardest. I considered giving up during this eight-hour window, but never actually came close as my thoughts and cravings were easily pushed down. By the time I woke up for day two, my hunger pangs had subsided, and it became much easier. I even went to a movie the night of day two, standing in line staring at the treats and smelling the popcorn and other foods, trying to buy a water, and didn't consider folding. Perhaps the temporariness of it, with a known end date, made it psychologically easier for me, but I found a three day fast much easier to stick to than even something as seemingly easy as a "no dessert or junk food for a month" protocol.

I experienced mental clarity, but no enlightened thought. What I did experience was overwhelming stupidity. My mind rarely goes blank. I cannot stop thinking, except when I initiate guided meditation or relaxation hypnosis. I also usually have no issues focusing. During the fast, I was almost always not thinking or struggling to remember what I was doing. Sometimes I would be completing a task and I would forget what my task was. Or I would finish, and my mind would go blank, spaced out into nothing.

I think what others experience is the high I felt. I presume as ketones are burning, releasing energy erratically and not constantly, waves of energy flow through the body. I would be lying down, as if stuck in concrete, and suddenly, my whole body would warm and start tingling. My head would tingle, euphoric. I want to say this is the "mental clarity" others have spoken to, but no advanced thought, no revelations or creativity accompanied it. I was nothing short of intellectually useless for the last two days of the fast. Two days later, and I am still "slower" than usual, my mind struggling to get back on track.

My workouts were arduous, and putting myself through them was perhaps the hardest part of the fast. My recovery from the pathetic training sessions has been slow, as workouts where I would usually barely break a sweat, when fasted, have my muscles feeling like they are "falling off the bone." Between the fast and the workouts, I have been able to solidly sleep two nights in a row of over nine hours a night. Great timing, as it is rounding out the Easter weekend (2019) as I write this. I would consider another fast, and likely will.

Since writing this, I have fasted many, many times, often up to 5 days at a time. I have even tracked my health markers across 15 months of consistent fasting. To summarize what I've discovered over this time, I regularly engaged in extended water fasts—initially fasting once per week for 42–48 hours (with a 72-hour fast every fourth week), and later shifted to two 42–48 hour fasts per week. When I made this shift, I stopped the planned 72-hour fast every 4th week and opted instead to extend the odd fast to 66–72 hours, although not on any set schedule. Throughout, I adhered to and completed a single full 5-day fast annually.

During this period, I lost 47 pounds, dropping from 267 lbs to 220 lbs—the lightest I had been in nearly a decade—while eating intuitively on non-fasting days, often without dietary restraint. Cravings all but disappeared, and fasting eventually became psychologically effortless. I maintained my regular training schedule during fasts and only observed performance decline beyond the 48-hour mark, with sharp drop-offs in strength and recovery around hour 67 and beyond.

However, not all effects were positive: over time, I noticed diminishing returns. A separate retrospective analysis of a full year's worth of Oura Ring data showed that, despite the weight loss, my sleep quality had declined slightly across several metrics: total sleep time, REM duration, deep sleep, and resting heart rate. Comparing sleep data during 24-, 48-, and 72-hour fasts against non-fasting nights also showed subtle but consistent reductions in sleep duration and REM, with elevated respiratory rates.

In the end, I concluded that fasting was a highly effective tool for resetting metabolic control, simplifying my relationship with food, and producing short-term anti-inflammatory effects. That said, longer fasts had trade-offs, particularly in terms of sleep, physical performance, and most importantly, mental performance. For me, the ideal fasting frequency and duration needed to be reevaluated, not based on ideology, but based on long-term data and personal response.

As far as my physical performance, as mentioned, it held steady through the 48-hour mark, but past 66 hours, it plummeted, and it plummeted fast. My strength, exercise capacity, and ability to recover all flatlined, maybe even declined, so I made the conscious decision to make changes. These days, I fast less often, not because I've lost faith in the method—it works—but because I simply can't afford the cognitive hit (as I noted in my story about my first fast, what some call mental clarity I experience as vacuity). Perhaps this experience is enjoyable for some, but for me, it is an unmitigated disaster. My days now demand intellectual horsepower, and I don't have room to spend 48 hours lost in the void.

Instead, I've transitioned to intermittent caloric deficits—less intense than fasting, but more sustainable for my current life. My calorie intake on these days is somewhere above the fasting mimicking diet and below your standard sustained weight-loss

deficit. I opt for roughly a 1,000-calorie daily deficit, achieved by locking in protein, cutting carbs and fats, and increasing low-intensity movement. Think long nature walks, cleaning, cooking, as in, something that I can jar and freeze, not eat in the moment—all the background rhythm of daily life dialed up with purpose. Fasting gave me a reset. It helped me reclaim my metabolic health, but like all tools, its value depends on the context. Use it wisely and don't get religious about it.

mTOR

mTOR, short for "the mammalian target of rapamycin," is exploding on the research scene, due to the fact that very early research suggests inhibition of its action has implications in aging. Here's what we know so far: mTOR is a protein kinase that helps regulate cell growth, protein synthesis, and metabolism. It's vital for building and repairing tissue—which is great when you're growing, recovering from a workout, or healing an injury (Wei, Luo, & Chen, 2019), though when mTOR stays activated too long, or too often, things start to go sideways.

What's been observed is that chronic activation of mTOR is correlated with cancer progression and inhibition of autophagy (Zoncu, Efeyan, & Sabatini, 2011), and it is also tied closely to some of the pathologies of Alzheimer's disease, such as neurofibrillary tangles, which are made up of tau protein (Tang et al., 2013; Caccamo et al., 2013) and beta-amyloid accumulation (Cai et al., 2015). That said, it's still unclear if increased mTOR signalling leads to increased beta-amyloid accumulation or whether beta-amyloid secretion leads to increased mTOR activity (Zoncu, Efeyan, & Sabatini, 2011).

The prospect of inhibiting mTOR, and the potential longevity implications from such inhibition, transformed from a whisper, a dream, to a potential reality with the discovery of rapamycin. Rapamycin, which inhibits mTOR activity, is being heavily studied in all of these disease models, with human trials underway for cancer, Alzheimer's, and even lifespan extension. Originally discovered in a soil sample from Easter Island (Rapa Nui), rapamycin has been shown to extend life in mouse studies, even when given to elderly mice who are already deep into their natural lifespan (Apelo & Lamming, 2016; Lamming, 2016).

That's a big deal, since most anti-aging interventions work best when started early—this one seems to work even when you initiate it late in life. In one study, researchers found that although rapamycin and dietary restriction both affect some of the same aging-related markers, they don't overlap completely. Some of the changes caused by caloric restriction didn't show up in the rapamycin group (Unnikrishnan et al., 2020). So it's not a perfect substitute, and we shouldn't assume that it is.

Here's why this matters to *StressHacked*: mTOR suppression leads to the activation of autophagy, which is the key reason rapamycin is gaining excitement. Fasting and dietary restriction both seem to promote autophagy, also, in part by inhibiting

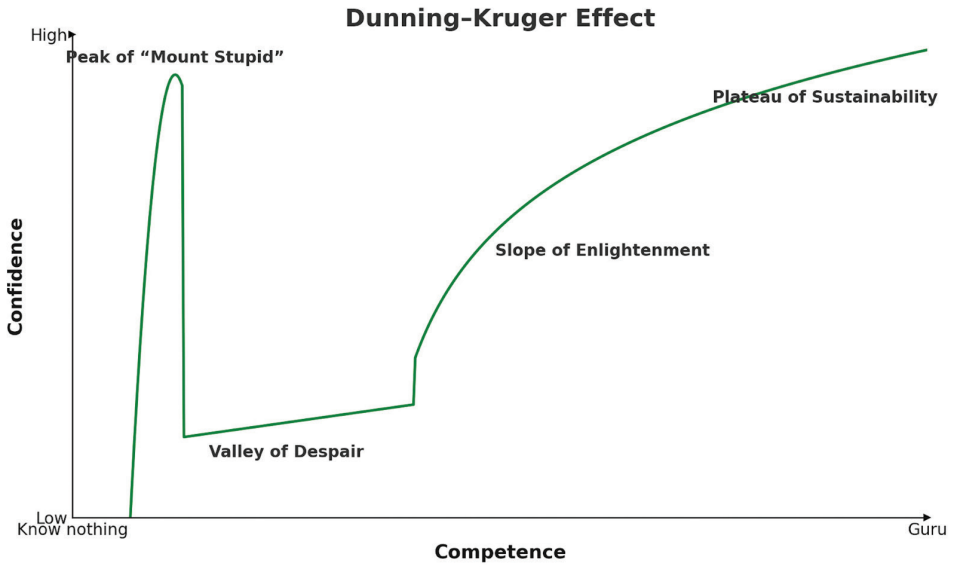
mTOR. When you fast, you're giving your body a signal: hold off on growth for now, and shift into maintenance mode. That signal helps clean out damaged cells and misfolded proteins, and it's likely part of why fasting shows such promise in everything from metabolic health to cognitive function. I'll explore autophagy more deeply shortly, but for now, think of mTOR as a growth switch. You want it on sometimes, especially when you're building or recovering, but you also want to know how to turn it off, at least temporarily. Fasting is one of the simplest, cheapest, and most accessible ways to do that. Unlike rapamycin, it doesn't require a prescription, or commitment to trusting a black market source, though both require optimism regarding the potential implications from early research.

Autophagy

Autophagy is the natural process of cellular breakdown. It works by removing damaged and improperly functioning cells and recycling the "parts." Yoshinori Ohsumi earned the 2016 Nobel Prize in Physiology or Medicine for his work and discoveries on the mechanisms of autophagy. The conceptual implications on the benefits in activating autophagy, namely breaking down "broken machinery" in our body and using the scrap parts to create new, functioning "machinery" are very intuitive—so much so I believe I peaked in confidence, reaching "Mount Stupid" on the Dunning-Kruger scale, within half a dozen hours of reading on the subject.

Figure 1. Dunning-Kruger Effect

Note. From these authors.



In trying to follow along during Dr. Ohsumi's recorded lectures, and reading significant portions of the literature, I spent the subsequent 20-30 hours attempting to increase my understanding, which was a straight nose-dive into the valley of despair. Some of the blame for said nose-dive can be attributed to the new technical terminology I had to become acquainted with in this new field, such as specific pathways and mechanisms I was not yet educated on. That said, a considerable portion of my confusion was due to the substantial complexity and deeply nuanced cause and effect ramifications for activation and suppression of this critical physiological mechanism.

So, what are the causes for confusion? For starters, autophagy is implicated in both cell survival and cell death (Baehrecke, 2005; Debnath, Baehrecke, & Kroemer, 2005). Further confounding my understanding of the overall implications is the notion that autophagy both suppresses and promotes cellular senescence (Kwon et al., 2017). Considering that when I dove into the research on autophagy I had already "known for years" that senescent cells are so-called unequivocally deleterious, being one of the largest drivers in disease states and accelerated age-related degeneration (Childs et al, 2015; McHugh & Gill, 2018; Sikora, Bielak-Zmijewska, & Mosieniak, 2014); my initial confusion was substantial—*how could a function that supports the process of senescence be good*?

This question necessitated me to dive back into the science behind cellular senescence. I learned that senescence can both be a protective barrier to cancer (D'Ambrosio & Gil, 2023), and dysfunction of cellular senescence can promote cancer via contributing to a chronic inflammatory state (Davalos et al., 2010). In a similar paradox, autophagy seems to prolong the survival of cancers when apoptosis (programmed cell death) is impaired, even leading to cancers believed to be eradicated re-emerging, while impaired autophagy increases the rate of tumor growth. Evidence also suggests that autophagy is protective in limiting tumour necrosis (dead cells within the cancer that are particularly nasty) and inflammation, while also mitigating genomic damage in tumour cells in response to metabolic stress (Poillet-Perez & White, 2019; Mathew et al., 2007).

Regardless, experts who are far more knowledgeable on the subject understand the implications of autophagic activation more clearly than I do, and there is significant excitement within this community of experts. Importantly, deactivating *autophagy is seemingly more harmful to more conditions as compared to activation* (Levine & Kroemer, 2008), and additionally proper activation is a potent protector against cellular stress (Kapuy et al., 2021).

I am going out on a limb, but perhaps autophagy is neither good nor bad, but simply a system that needs homeostatic function, such as our inflammatory defenses or our redox status, or virtually every molecule that plays a physiological role in our body, from proteins to neurotransmitters to hormones to nutrients. Senescence may even fit

into this, to an extent. The default in most models could be under activation of autophagy, so the knowledge that short-term and intermittent fasting has been shown to promote autophagy is particularly relevant (Antunes et al., 2018; Alirezaei et al., 2010).

AMPK: Your Metabolic Switchboard

AMPK activation is another interesting area of research that has been implicated as a mediator, and necessary component, of exercise-induced mitochondrial biogenesis via activation of PGC-1α. PGC-1α is a key player in the activation of autophagy, and a regulator of our endogenous antioxidant defense systems (Jeon, 2016). AMPK activation, or rather the activation of the equivalent AAK-2 found in *Caenorhabditis elegans* (worms), has also been shown to be necessary in the extension of life in glucose-restricted *C. elegans* (Schulz et al., 2007; Curtis, O'Connor, & DiStefano, 2006; Apfeld et al., 2004).

What this means is that when these worms are starved of sugar, they live longer, but only if their AMPK analog is working properly (Jeong et al., 2023). In humans, most of the research connecting AMPK to real-world health benefits has focused on exercise—which remains the most potent and consistent activator we know of—but fasting, and particularly nutrient and glucose deprivation, is starting to show promise as another route to turn on this energy-sensing switch. There's a catch, though—actually, several: first, much of the fasting-AMPK data comes from rodent studies, and mice are metabolically hyperactive little machines. They burn through energy about seven times faster than we do (per gram of body weight), so a 24-hour fast for a mouse is not even close to the same metabolic stress as 24 hours without food for you or me (Carper et al., 2020; Perlman, 2016; Speakman, 2013).

To put it another way, energy requirements in mice are significantly higher per gram of body weight—seven times higher, in fact, than humans (Demetrius, 2005)—so we cannot directly convert fasting durations from mice to men and expect the same altered expressions to be consistent. A direct multiplication would imply that a seven-day fast is necessary to see the same results as in mice, which may be excessive. A better standard could be the determination of glucose starvation by the measurement of blood or urinary ketones.

Personally, when I conducted my first 72-hour fast, I measured my ketones throughout. My ketone measurements via urine were roughly 1.5 mmol/L by hour 46, 4 mmol/L by hour 52, and in excess of 8 mmol/L when I broke my fast at the end of the third day. I did "kickstart" the fast with a high-fat meal after a soccer game, then a hard workout the next morning, and daily workouts and long walks throughout day two and three, that said.

Think of AMPK as your body's built-in energy conservation mode, like the setting your computer offers you to switch to when the battery runs low. When cellular

energy dips during exercise, fasting, or caloric restriction, AMPK kicks in, rerouting processes toward efficiency and survival. It doesn't just slow things down, it reprograms your metabolism. Sugar burning gives way to fat oxidation, growth pathways pause, and cleanup and repair ramp up. In short, AMPK activation triggers a metabolic shift that favors long-term resilience and healthspan (Cantó & Auwerx, 2011; Hardie & Carling, 1997).

Ketones

There was a point where ketone bodies were a hot topic in mainstream media and discussion, primarily due to the then-popularity of the ketogenic diet, controversy over its claims, and the ensuing release of countless products purporting to raise ketone levels. These days are long gone, with the ketogenic diet largely declining in popularity. The data on ketones and their role, both beneficial and harmful, in different disease states, such as cancer, should nonetheless warrant caution. Ketones have been shown to increase the rate of tumour growth and metastasis (the development of secondary growths), with ketone suppressants being a proposed drug candidate for cancer research (Martinez-Outschoorn et al., 2012). Another study has shown that while initial beneficial signalling for anti-tumour effects occurred on a high-fat ketogenic diet, prolonged exposure to this state ultimately led to increased tumour development and faster growth (Liśkiewicz et al., 2016).

Conversely, a pair of rodent studies from the 1980s showed inhibited tumor growth from a low-carbohydrate, high-fat ketogenic, diet (Beck & Tisdale, 1989; Tisdale, Brennan, & Fearon, 1987), and more recent studies using human cancer cells in mice on a calorie-restricted ketogenic diet have been favorable in slowing tumor formation (Ho et al., 2011; Seyfried et al., 2009; Otto et al., 2008; Zhou et al., 2007b). It is still unknown if the cancer-inhibiting benefits come from the caloric restriction or the high-fat, low-carbohydrate diet. Evidence on the latter is contradictory in its conclusions, while evidence regarding potential mechanisms of action, such as mTOR inhibition and autophagy activation, is stronger in calorie-restricted states. Considering this, I would err on the side of the former, as I tend to err on the side of what we do know rather than what we don't when making recommendations for others, or where evidence of safety is inconsistent or absent. (If something is nascent but likely harmless and with potential benefits, I jump into it, cost permitting. My views on finances are quite different from the norm, so a cost analysis comes into play less than it would for the average person, or even someone with similar financial circumstances to me. Not everyone's risk assessment will be the same, so I try not to be presumptuous).

There are a few small, early human studies on a high-fat ketogenic diet and tumour growth in cancer patients (Schmidt et al., 2011; Nebeling et al., 1995; Nebeling & Lerner, 1995). These studies generally have short durations, typically ranging from a few weeks to a few months, and their results are heterogeneous and inconclusive regarding anti-tumor efficacy (Römer, Dörfler, & Huebner, 2021). More studies are

currently underway, along with studies in fasting and the fasting mimicking diet. Perhaps ketone bodies are an important piece of the puzzle. That said, the evidence is currently much stronger in promoting acute exposure than maintaining an extended or permanent state. High-fat fed mice do not typically have high rates of health and survival over a lifetime, which is an important consideration, as well (Muller et al., 2013). In fact, most metabolic disruption models in mice utilize a high-fat diet to induce obesity, cardiovascular stress, and more (Johnson et al., 2023; Che et al., 2018). Luckily, there are several methods available where we can experiment with short exposures to ketosis, such as fasting.

5:2, 1:1, and the Problem of Evidence

Two "fasting-inspired" diets have made waves over the past decade: the 5:2 and alternate-day fasting models. Both promote intermittent caloric restriction, but in different formats. While they've gained popularity—and headlines—I have some concerns: chiefly, long-term adherence, impact on physical performance, and, as is so often the case, the quality of the evidence.

The 5:2 diet is simple in concept: eat normally five days a week, then restrict intake to about 25% of your usual calories on two non-consecutive days. The idea is to create a net weekly deficit without daily deprivation, but there's a problem: data is limited. I could only find two published studies on the 5:2 diet, both from the same research group. While they showed benefits, the results weren't any better for weight loss than traditional caloric restriction evenly spread across the week (Harvie et al., 2013; Harvie et al., 2011).

One interesting note: the 5:2 group showed a greater increase in ketone levels and improved insulin sensitivity compared to the daily restriction group. That's encouraging, but it's worth pointing out that the study used *consecutive* fasting days, not the "non-consecutive" model promoted in the original diet. That's a pretty big deviation. Additionally, despite the short timeframe—just three months—the 5:2 group still had a 23% dropout rate. This is not unusual for diet trials, but telling. If nearly a quarter of participants couldn't stick with it for 90 days, we have to question the long-term practicality for the general public.

Alternate-day fasting (ADF), or the "1:1" model, has amassed a bit more evidence, though even here, most studies lack control groups or comparisons to traditional calorie restriction protocols (Sutton et al., 2018; Heilbronn et al., 2005). One trial used a randomized crossover design, which is a methodological upgrade—but again, no comparison to a daily restriction control group (Horne et al., 2013).

A few hybrid approaches, variations between 1:1 and 5:2 have been investigated, but the evidence remains inconclusive. Nearly half of the studies lacked any control group, making it impossible to attribute outcomes specifically to the fasting regimen (Hoddy et al., 2016; Eshghinia & Mohammadzadeh, 2013; Varady et al., 2009; Johnson et al., 2007). In another case, both the fasting and comparison groups

consumed identical calories on non-fasting days, obscuring the effects of the fasting periods themselves (Williams et al., 1998). Some investigations used participants' habitual diets as a "control," which provides no active benchmark against standard calorie restriction (Bhutani et al., 2013; Varady et al., 2013). Even when fasting was paired with exercise, the superior results over doing nothing tell us little about how alternate-day fasting alone compares to conventional energy-limited diets (Ezpeleta et al., 2023). Overall, methodological inconsistencies and inadequate controls prevent clear conclusions about the true benefits of these hybrid fasting approaches.

The bottom line is that we can't make any strong claims based on the current human data. From a practical perspective, especially for those of us who care about performance, there are real limitations. Training hard while in a significant caloric deficit is difficult. Recovery suffers and muscle protein synthesis slows. Over time, this can blunt progress in both strength and endurance training. I suspect that both ADF and 5:2 protocols could hinder consistent, high-quality exercise performance and recovery, particularly for those pushing their limits.

That said, if you're already practicing one of these methods, feel great, and can train effectively, carry on. There's a difference between poor data and poor results. If you've found something that works for *you*—physically, psychologically, and socially—don't let a lack of perfect literature talk you out of it. Now, from where I stand, the jury's still out. If you're looking to optimize both body composition and performance over the long haul, there may be more sustainable, flexible approaches available.

Time Restricted Eating: When You Eat vs. What You Eat

Time-restricted eating (TRE) is a strategy that is interesting, easy, and has a small amount of research supporting its efficacy. Much of the research on TRE revolves around our circadian rhythm. Mice restricted to eating during their normal feeding hours lower their risk of metabolic issues compared to mice fed *ad libitum* at an equivalent caloric intake. This is true even for mice on a high-fat diet, which are used for the express purpose of promoting metabolic disruption and obesity (Chaix et al., 2018). Interestingly, according to one study, when individuals with type 2 diabetes skip breakfast but consume the same amount of calories at lunch as those who eat breakfast, blood glucose levels following a meal worsen, with higher blood glucose, free fatty acid, and glucagon levels, as well as impaired insulin and GLP-1 responses, despite identical caloric intake later in the day. Thus, skipping breakfast does not improve metabolic markers, has been associated with weight gain, and may be detrimental to glucose regulation (Jakubowicz et al., 2015). Additionally, nighttime eating restrictions have shown improvements in human models (LeCheminant et al., 2013).

There are a pair of studies on the use of reduction of meal frequency to maximize the benefits of TRE (Carlson et al., 2007; Stote et al., 2007). While both showed weight

loss and improvements in metabolic outcomes, I remain skeptical of the overall benefit. As I will detail shortly, I eat almost in reverse of this recommendation, and the very reason I began delaying my first meal was the lethargy I feel after a large meal, no matter how nutritious and balanced the meal. If we are to accept the circadian rhythm research, logic would suggest having the large meal earlier in the day or midday. Eating a day's worth of calories in a single meal in the middle of the day would render me incapable of functioning, as I imagine it would for many.

When I first heard about TRE, it was in casual conversation, explained to me in no more than a few sentences. I was elated, and thought to myself, "*I am already doing this*." You see, for years I had naturally gravitated to eating in a short window, typically consuming my first calorie in the afternoon, and then eating through the evening. When I heard that eating within a restricted window conferred benefits, I naturally wanted this to be true, considering it was something I was already practicing, or so I thought.

Then I dove into the research, intent on arming myself with information to validate my actions—not consciously, of course, but subconsciously, my drive towards confirmation bias was underway. Soon after, I began realizing I was "doing it all wrong." The research was suggesting that I should be front-loading my meals in the morning, to breakfast and lunch, and cutting out the feeding window in the evenings. In fact, the way I was doing it may have deleterious effects, as I could be disrupting my natural circadian rhythm.

Intent on correcting my errors, I worked to switch my eating habits towards that which the TRE guidance recommends. I began indulging in breakfast and lunch. What followed was a reminder of why I had developed the eating habits I previously had, namely, skipping both of them: eating, particularly early in the morning, slows me down. I get substantial brain fog, fatigue, and my motivation deteriorates. If I manage to go to the gym after eating a full meal, my performance isn't just subpar, it's downright atrocious. Determined to "solve my problem," I spent an inordinate amount of time adjusting the macros in my diet in every possible way, focused on making TRE, as described in the literature, work for me.

Finally, I abandoned the pursuit. TRE, as advised, does not work for me. My own modified TRE, however, *does*. I tend to begin snacking on fruit around lunch time, between 11 AM-12 PM each day, typically waking up around ~6 AM. This gives me ample time to digest and work through my small mountain of supplements, which I begin taking shortly after waking, many in beverage form. I exercise at around 1 PM, the fruit supports glucose levels, and I have my first meal following training. Typically, I will consume ~600-1000 calories in this first meal, consisting of anywhere from 40-70g of protein. I then eat my second, and final meal, around 7 PM, five hours later. The only change I have made is that I now do my best to stop snacking, finishing all food no later than 8 PM.

For my performance, mental and physical, an empty stomach works the best, up to a point. At 40 years old, my metabolic markers are optimal, and my sleep data—the marker which TRE proponents claim is negatively affected by late feeding—is strong enough to put many of the most devoted biohackers to shame. Namely, my sleep latency is on point, and my combined deep and REM sleep tends to be 50% or more of my total sleep. I dream vividly, and I wake up refreshed. If feeding later in the day affects people on a population-wide basis, then I am an exception. This is an important lesson, as no matter how intriguing the data is, and no matter how much you want the data to be true, for many of these lifestyle interventions, the best advice is still, unfortunately (because this is difficult and subjective), to listen to your body and follow what works.

Fasting vs. Calorie Restriction: What's Really Driving the Results?

In recent years, a series of reviews have begun to slow the hype on the fasting research, adding a healthy dose of doubt and skepticism, by suggesting that all these trendy new protocols (whether it's intermittent fasting, time-restricted eating, or alternate-day strategies) are just different delivery vehicles for an old concept: *caloric restriction*. According to these analyses, the benefits people experience from fasting (such as weight loss, improved insulin sensitivity, reduced inflammation, etc.) are due not to the fasting state, but simply because people end up eating less (Hamsho et al., 2025; Črešnovar et al., 2023). On paper, that argument holds weight and validates the "calorie in, calorie out" mantra. If your overall calorie intake drops, your body responds. Whether you skip breakfast every day, don't eat Tuesdays and Thursdays, or just stop eating at 7 PM, if you're taking in fewer calories, you'll likely see results, at least for a while.

Yet this reductive framing misses some important nuance: first, not all forms of calorie restriction are equal in how they affect behavior, compliance, hormonal adaptation, or metabolic flexibility. Anyone who has tried to "just eat less" every day for months on end—or forever—can tell you it's psychologically taxing and unsustainable. Fasting, by contrast, offers structure. It sets clear and binary boundaries, which, for many people, makes it easier to follow than a constant low-grade restriction that demands decision fatigue at every meal. Second, some protocols, particularly longer fasts and alternate-day approaches, appear to activate unique cellular pathways (like autophagy and AMPK) that aren't as robustly triggered by mere caloric deficit alone. While the science is still emerging and not universally agreed upon, it's possible that *how* you restrict matters as much as *how much* you restrict. Third, the timing of food intake, meaning whether it aligns or conflicts with circadian biology, may influence outcomes independent of calorie count. As I've already discussed, the evidence, albeit limited and questionable, suggests that eating the same number of calories at breakfast vs. dinner can lead to different insulin and metabolic responses.

So yes, calorie restriction is likely the engine behind many of the benefits seen with fasting, but fasting may be the more efficient (and for some, more sustainable) vehicle to get there. That doesn't mean it's for everyone, and it certainly doesn't mean we should oversell its benefits or ignore its trade-offs. However, reducing all fasting protocols to mere "stealth calorie cutting" might be too simplistic as well.

Where That Leaves Me

Dietary restriction, as a broad category, continues to draw fascination and hype—not always in proportion to the strength of the evidence. There's enough preliminary research to justify curiosity, but not enough to suggest any one protocol as universally superior to the rest. Personally, if I had to pick from everything discussed so far, I'd lean toward a time-restricted eating schedule that cuts off food intake in the evening, combined with the occasional water fast or significant caloric deficit, cycled in and out as needed. I choose this not because that approach is decisively better in the literature, but because it's the one I can actually stick to without destroying my productivity or sanity (though my protocol has changed slightly). I never fast anymore. I TRE, sort of, as I already mentioned. Your needs will undoubtedly vary. Also, just to state the obvious: before jumping into any of these protocols, speak with your doctor, especially if you have any underlying medical conditions that could make fasting or caloric restriction risky.

Calorie Restriction

Introduction

It's hard to pin down exactly when humanity first started making the connection between eating less and living longer. Philosophers and physicians have been warning about gluttony since at least Ancient Greece—long before metabolic pathways had names or longevity studies had control groups, but It wasn't until the last century when the first real efforts to study the effects of dietary restriction in a systematic, measurable way finally showed up, after World War I, when researchers began examining energy efficiency in populations affected by food shortages (Benedict, 1919). Since then, the nutritional world has been flooded with theories—many of them eventually disproven, repackaged, or replaced by the next big thing. Despite the noise, we've accumulated a reasonably solid body of evidence on caloric restriction, namely, its benefits, its limitations, and where it might play a role in optimizing human lifespan. Like most things in the health and performance world, it's more complicated than "eat less, live longer," but there's enough to warrant serious attention.

The Case—and Caveats—for Calorie Restriction

Caloric restriction wasn't originally studied for its health benefits. The first major foray came in 1935, when a study on mice found that eating less extended lifespan (McCay, Crowell, & Maynard, 1935). That modest finding didn't turn the world

over, but it did plant a seed of thought. Over the following decades, various human studies, most of them incidental and conducted for other purposes, began to show that dietary intervention alone could influence key health markers like blood pressure, cholesterol, and resting heart rate (Huffman et al., 2022; Puska & Jaini, 2020; Appel et al., 1997). Eventually, the idea took hold that caloric restriction might function through hormesis (Nikolai et al., 2015; Martins, Galluzi, & Kroemer, 2011). Of course, by now you know that hormesis is the concept that small, manageable stressors can make an organism stronger.

Yet, despite nearly a century of investigation, caloric restriction and human longevity remains as much a philosophical problem as a scientific one. There's a compelling amount of evidence that reducing caloric intake improves biomarkers of aging, such as fasting glucose, cholesterol, oxidative stress, and adiposity. Additionally, the evidence suggests caloric restriction may reduce risk factors for diseases like diabetes, cancer, cardiovascular disease, and even brain atrophy (Redman et al., 2018; Holloszy & Fontana, 2007). That said, when we zoom out to longer-lived mammals, like primates, things get more complicated. Of three long-term studies on macaques, one showed no significant life extension despite the animals aging "better," while the other two showed clear longevity benefits (Mattison et al., 2012; Colman et al., 2009; Bodkin et al., 2003). The only other long-term caloric intake study in primates was conducted in mouse lemurs, a more distant cousin on the primate tree, with the study results suggesting that there's evidence of lifespan extension from caloric restriction—though notably, the same study found that CR accelerated grey matter loss across much of the cerebrum, even as cognitive performance remained unaffected (Pifferi et al., 2018).

Some researchers have tried to reconcile these discrepancies. A 2017 review suggested the "non-significant" macaque study did in fact show lifespan benefits compared to typical wild populations, and chalked up differences in results to methodological inconsistencies (Mattison et al., 2017). Then, a 2018 review took the opposite view: the longevity effects may have stemmed less from calorie restriction itself than from reducing overfeeding and metabolic overload in poorly managed control groups (Le Bourg, 2018). In short: the jury is still out. When it comes to humans, the plot again thickens. Some argue that caloric restriction could yield real, if modest, longevity gains—assuming people can actually stick with it without tanking their quality of life (Roth & Polotsky, 2012). Others believe the benefits won't scale up meaningfully in humans, and that pharmacological interventions may hold more promise (Spindler, 2010). The most cautious (and possibly wisest) voices remain neutral, emphasizing how little we really know from studies on non-obese, otherwise healthy humans. Unfortunately, this is the population mostly left out of this research to date, for the obvious reason that caloric restriction interventions tend to be geared to those who need it most: the overweight and obese.

Even aging science luminaries have hedged their bets. Aubrey de Grey, perhaps the

field's best-known optimist when it comes to extending human lifespan, estimates caloric restriction might buy you two or three extra years (de Grey, 2005). Valter Longo, another major figure in this field, has suggested that long-term caloric restriction may actually backfire as we age, leading to muscle loss and frailty (Longo & Anderson, 2022). His solution is to change the strategy later in life—add back more calories, especially protein, to preserve strength and functionality. That stance, to be fair, does clash with other studies showing that even late-life caloric restriction can improve health markers in both humans and rodents (Parikh et al., 2016; Witte et al., 2009). Longo's critics aren't necessarily disproving him, as one study noted that late-life restriction only helped because participants had weight to lose. If someone had been restricting all along, they would be unlikely to experience that benefit. The results, in other words, are highly conditional (Prehn et al., 2017).

Adding more fuel to the fire, it's often said that calorie restriction clearly extends lifespan in both yeast and rodents, but even that's not as simple as it seems. A 2014 meta-analysis of 40 yeast studies found that lifespan extension wasn't consistent, and depended heavily on strain and context (Huberts et al., 2014). In rodents, the effect varies dramatically between species: rats seem to benefit significantly, while mice, particularly wild ones, do not (Swindell, 2012). Much of the research has been done on lab-bred strains engineered for consistency, not longevity, which further skews the data.

To sum it up, caloric restriction shows promise, but its long-term efficacy in humans remains largely uncertain. The complexity of the data, combined with the practical challenges of lifelong adherence—and the questionable appeal of doing so—renders the prospect less compelling, especially when we have evidence suggesting that intermittent fasting, practiced just a few times a year, might achieve many of the same physiological benefits (Siles-Guerrero et al., 2024; Mahoney, Denny, & Seyfried, 2006). If two weeks of restriction can enhance biomarkers, and those improvements persist even after returning to normal eating, then the thought of committing to a lifetime of stress seems not just unnecessary but even masochistic, at least from my perspective. I'll choose a few carefully timed fasts over 40 years of low-grade suffering any day.

Valter Longo's Longevity Diet: Explained

Valter Longo released a book in 2018 on his proposed "Longevity Diet" and intermittent use of a "Fasting Mimicking Diet"—putting forth his proposal as an alternative for caloric restriction, and also as an alternative to extended 3-5 day (or longer) water fasts (Longo, 2018). Longo spends almost 100 pages in his book (although a more standard use of spacing, indexing, and font size could have easily reduced this to ~30 pages) weaving a story in the attempt to reframe what in essence is a pescatarian diet with specific restrictions, particularly on eggs and dairy from cows (although he does recommend hard cheese from sheep and mentions yogurt from goat's milk, etc.).

Longo discusses centenarians and the diets found in the various "blue zones": areas in the world with statistically very high rates of individuals living to and past 100, emphasizing more on their diet and lifestyle than genetics. I forced myself to think long and hard about Longo's positions. After watching several of his podcast interviews totalling hours of time listening to his positions and beliefs, and reading his book, I became aware of, and began to shed, a halo effect I had regarding Longo: Longo *is quite transparent with his biases*, criticizing pharmacology and the pharmacological approach to disease management, using statins as evidence. Criticism of statins is widespread; however, the ineffectiveness of statins for improving mortality is not evidence that other drugs do not, and cannot, work. As I show shortly, Longo contradicts himself by using his own family—and the anecdotal prevalence of obesity and heart disease in his relatives from Chicago as compared to his relatives in Italy—as evidence that diet is more important than genes.

In particular, he claims that the explanation of Emma Morano's incredible longevity, the second longest living European recorded—and longest living Italian—is an "example of good genetics, as her diet was poor." Emma consumed 3 eggs a day, which does not fit into Longo's position. Longo states, however, that his friend Salvatore Caruso, who does not make the list of the 100 longest-lived Italians, *was a perfect example of longevity from adhering to an ideal diet*, and *not genetics*. Longo is also quite noticeably proud of being Italian, and excited about areas where he had spent childhood being among those of the longest lived. His dietary recommendations tend to be skewed to the Italian side, drawing stronger conclusions on an Italian town of 2,000 inhabitants, rather than, for instance, the more logical skew, statistically speaking, towards those in Okinawa, Japan, and their 1.4 million inhabitants.

With Longo, I yo-yo'ed quite substantially. I went into reviewing his work expecting to learn, and that he was an expert, but then began to reject everything he was saying due to his biases. Sitting back and carefully considering Longo's positions, I tend to agree with much of what he says, despite my "bullshit detector" going off while listening to him quite frequently. I have long been of the position that a modified pescatarian diet was likely the best choice for health. The benefits of the diet extend into sustainability as well, with Lord Martin Rees, a renowned British cosmologist and astrophysicist, suggesting at a talk I watched live years ago that the planet could sustain 35 billion people if everyone simply lived in high rise condos, ceased to own pets, and ate a pescatarian diet. This isn't a future I want for myself, or advocate for, it is simply a commentary on the apparent math regarding sustainability.

Contributing to his position that fish protein intake should be limited to a few times a week and in small quantities, this study on caloric restriction showed that IGF-1 does not increase in humans even under caloric restriction unless protein is also reduced (Al-Regaiey, 2016). When stripping away Longo's biases and storytelling, I believe the longevity diet is prudent advice based on the best evidence we have.

That said, other factors need to be considered, such as the pursuit of athletics for enjoyment. Competing in sports typically isn't done for health and longevity, but passion and enjoyment. I cannot imagine a lifestyle in which I didn't exercise more than what is deemed "moderate and healthful." I still exercise above this recommended level, despite having permanent disabilities from structural damage to my left shoulder. My exercise is just greatly modified to avoid my permanent musculoskeletal issues.

I've attempted to eat diets closely related to Longo's longevity diet in the past, and simply could not function throughout the day while exercising to even an above moderate level. Upping my vegetables and nuts caused digestive distress, and increasing my whole grains from pasta, brown and wild rice, and whole grain breads, caused gains in adipose tissue and not muscle mass. Training above a light 30-45 minutes a day, I have found that I cannot function without the inclusion of eggs and daily meat protein intake. I may be an exception and cannot prove I am not, or perhaps with another attempt I could eventually get the protocol to work with some modification, likely an increase of both nuts and whole grains—that said I am not convinced that the suggested dietary interventions and restrictions are the answer for everyone, always. They may simply be a good guideline for some people, some of the time.

Valter Longo's Fasting-Mimicking Diet: Explained (And Tested)

Valter Longo claims he developed the fasting-mimicking diet (FMD) in the belief that acceptance and observation of a sustained three-day, or more, fast to be unlikely and difficult for most individuals. Additionally, he argued that potential health hazards would exist for a portion of the population. Based on quick math, his five-day FMD protocol is very close to the total caloric deficit of a three-day fast, based on the standard 2,000 calorie diet. Based on 2,000 calories, a three-day fast puts stress on your body to account for a 6,000 calorie deficit. During this fasting period, your body will generate energy first by exhausting glycogen stores before moving to fat burning or ketosis. Conversely, during the FMD, Longo recommends a 1,100 calorie day one, split evenly between carbohydrates from vegetables, a small amount of proteins from nuts, which also contribute to fats alongside a bit of olive oil, before moving to the same 50/50 split days two to five, but on 800 calories.

Over the five days, an expected 10,000 calories becomes a deficit of 5,700 calories (300 less than a three-day water fast), with elimination of animal proteins and a decrease in total proteins (likely) contributing to a similar decrease in IGF-1 as would be expected in a three-day fast. The FMD also comes with a very low glycemic load and low total sugar and carbohydrate intake, which I would hypothesize would lead to ketosis. To experiment on this in 2019, I subjected myself to the FMD and thoroughly detailed my experience.

I will note that, mathematically, Longo's FMD is a larger stress to me than a three-day water fast. I am larger than the average man with significantly more muscle mass than the norm. In 2019, several years after my health crisis, I was in a recovery phase. I had put on considerable fat mass over the last several years, but at the time of experimentation had dropped 16 pounds in four months, or roughly a pound a week. I managed this loss while not just maintaining, but increasing, muscle mass. During this loss I was maintaining a ~3,000 calorie a day diet—which was an increase of 50% of my caloric intake that I was previously gaining weight on just months previous—meaning the stress the FMD put on me, as compared to someone on a standard 2000/day caloric intake, had to be re-evaluated. At a 3,000 calorie daily intake, a three-day fast puts me on a ~9,000 calorie deficit, whereas Longo's FMD will put me at a deficit of ~9,700 calories.

Day 1: The Hardest Bite

To make sure I was closely matching my experience from the three-day water fast, I started the FMD with a high-protein and fat/low-carb meal right after a soccer game the night before. I tried to be active during day one, doing a short workout, then working up a heavy sweat moving around totes and bins at my R&D facility, and finally going for a 2 km walk in the evening. I had diligently mapped out my daily meals of almonds, green vegetables, and two tablespoons of olive oil. I chose to err "low" in calories in every measurement, maybe totalling ~780/day. Day one was almost as challenging as day one on a water fast. I was irritable and constantly hungry. I had serious issues falling asleep and ended up "sleeping" for 10 hours, or rather spending 10 hours in bed.

Day 2: Cognitive Fog, Fast Breaks, and Blue Cheese Temptation

I woke up to day two of my FMD with a splitting headache. My blood glucose was not altered from my typical readings at that time, at about 5.1 mmol/L or 92 mg/dl. I didn't bother to check for ketones as I was certain I was not yet in ketosis. I tried to have my "breakfast" and the food only further agitated me. My headache became worse, and my food cravings more intense. My mouth was watering constantly throughout day two. And I bit almost everyone's proverbial head off that I spoke with. I was cranky, aggressive, stupid, and managed very little productive work. I had an indoor soccer game at 7:30 pm that I did not want to show up to. Since it was my last game for my men's team before shoulder surgery, which would have me sidelined for months, I went—despite feeling awful.

I wasn't very optimistic about my ability to play, and warned my teammates before the game started. My first shift went quite poorly. I was low on energy, became tired quickly, and my ability to recover was nonexistent. I contemplated telling my team I couldn't play another shift. We were short on subs, as some of our players were late to show, so I went back on for another shift, thinking I would just coast. I was playing mid, and I figured I needed to be more defense-focused due to limited energy.

A chance break with one of my teammates, with our other forwards being caught back, had me sprint to the end of the field for support. I once timed my teammate's rebound, missing just high, then headed my rebound in. I tell this story, not to brag about my low-level rec soccer accomplishments, but because interestingly, instead of being "gassed" from a full sprint at the end of a normal shift, I felt charged and full of energy.

My skin was buzzing. I played the rest of the game at mid (I was usually a forward), running up and down the field much easier than I typically manage. I ended up scoring another goal, a one timer on a full sprint, which surprised me, as lately my coordination in a full run has been clumsy and reflexes slow, or at least much slower than my own self-perception. Aging comes for every man. As I rewrite this story 6 years older, I am keenly aware of the further decline in my fast-twitch muscles. Of note, this was my first two-goal game of the season, and I was playing mid, not forward. It was also the first game in quite a long time where I was coming off the field when I was out of breath, and not because my muscles were exhausted.

After the game, I went upstairs with some others and talked as they had wings and beer. This was by far the closest I came to giving up: *the smell of the wings had me in a frenzy*. My mouth was watering. I tried to break my stare on the wings and had myself looking at an empty spot on the table. A lid from a blue cheese dip container was in the corner of my eye, and soon I was fixed, staring at it. I'm not sure how much time went by staring at the blue cheese lid, battling myself on not picking it up, and licking the sauce. I was in my own world, fighting a battle. I've never been addicted to a drug, but this is the closest thing I could relate to. My breathing was altered, and I couldn't hear what others around me were saying. The "owner" of the blue cheese lid finished his wings and piled all the trash on his plate, freeing me from my gaze. I was upstairs with others for almost an hour and a half, but on the drive home, I could recall all of 10 minutes of conversation. I had just been "gone," and when I was part of the conversation, I was unpleasant.

My energy had crashed to rock bottom. At one point, I needed to use the restroom, finding the one upstairs in the pub closed for maintenance, necessitating me to go downstairs. I stared at the stairs and almost gave up and went back to my seat. A single flight seemed daunting. I knew I wouldn't make it home, so I pushed through and went down again, feeling a rush of energy after using the restroom and washing my face with cold water. I jogged back to the stairs and went to run up them. I made it halfway and felt dizzy, clinging to the railing. A friend and teammate started walking down as I was about to sit down on the stairs. I changed my mind on sitting down and struggled up the stairs. Immediately, I told everyone I was going home.

I made it home and could barely manage to drink some water, brush my teeth, and crawl into bed. My head was spinning, I couldn't even shower. I passed out early and slept solidly for 9 hours.

Day 3: In Ketosis, Out of Power

I woke up after 9 hours of sleep and could barely roll over in bed. I was weak and dizzy. Half an hour went by, and finally I was able to muster enough energy to climb out of bed. When I finally managed to get up, I felt "good," or at least relative to how I had felt half an hour before. I measured my ketones right away, and sure enough, trace levels were detectable.

I measured my blood glucose at 4.6 mmol/L or 84 mg/dl. I was quite behind on some emails, and spent the first hour catching up on work, then another hour writing. I didn't "want" food, but I did want to adhere to the protocol, so I forced myself to eat my breakfast. Almost immediately, my brain function became impaired; I became agitated, and all I could think of was food. About an hour went by, and I accomplished maybe 5 minutes of total productivity. Another hour passed, and I slowly started to feel OK again. I was able to start writing and responding to e-mails, and started remembering tasks that had vacated my consciousness the preceding hour.

My muscles were devastated. It was an arduous task to take the garbage out. To keep my body working and mimic my behaviour from the three-day water fast, I decided to try a round on my Thai bag. Walking out the garbage was a daunting task that proved to be laborious, but in contrast, once my heart rate elevated, I was able to get through a few rounds on the bag with no problem. I felt fine, my reflexes and speed were normal. I got cocky and tried to pick up some weights to do some bicep curls and deadlifts, and that was a mistake. Within a few reps, my muscles were exhausted on the curls, and my elbows hurt. I went to try some light deadlifts, and by rep two, my back was pinched, although minor.

The workout did increase my energy levels momentarily, and I was able to plow through more work, more tasks, and get more writing done. One thing I noticed is that hydrogen water seemed to have less of an impact during the FMD for the biggest side effect I experienced: headaches. During the water fast, the biggest downside was the lethargy. Using a hydrogen tablet would charge me up for a time, and I would be good to go. During the FMD, while I experienced a burst in energy after drinking the high-dose hydrogen water, my headache wasn't going away.

The worst part of the water fast was the waves in energy and how I would only get work done in a couple of hour spurts twice a day, immediately after drinking hydrogen water. I was getting much more done on the FMD, but the biggest side effects had no solution. Interestingly, "dinner" on day three led to an increase in energy and satiation for a couple of hours, before plummeting to the worst I had felt so far. Perhaps a combination of burning ketones with a small burst of calories allowed my body the energy it needed for a moment.

My stomach, or ability to eat, shrank dramatically. The small bowl of vegetables and almonds filled me up. I needed roughly 30 minutes to finish the bowl completely. This is anecdotally an even more aggressive temporary reduction in appetite than I experienced in my first meal following the actual complete three-day water fast. The crash in energy came suddenly, and I stopped writing mid-sentence with a spinning and throbbing headache. I ended up going to bed hours before I typically do, and actually fell asleep. That may have been a blessing, if only I could have stayed asleep. What happened instead was I shot up awake around when I usually fall asleep, my body tingling and high.

I went to the bathroom and checked my ketones. Sure enough, they were somewhere between 1.5-4 mmol/L. I spent the next two hours salivating, thinking about food and what I would eat when this was over. If I had any food in the house other than for my FMD, I may have actually cracked.

Day 4: Still Here, Still Fogged

I woke up on day four feeling weak and exhausted. I checked my ketones and they hadn't moved from the night before, with my blood glucose being 4.5 mmol/L or ~82 mg/dl. I wasn't capable of having my normal hot-then-cold shower, even starting it off close to hot, my body didn't take it well. I settled on a lukewarm shower. I had shockingly lost 9 lbs waking up at the start of day four. For comparative purposes, during my first water fast undertaken not long before, I was only down 7 lbs by the end of day three. While I was not tracking water intake, it was well above usual, and the vegetables I was consuming were high in water content. Day four struggled on, barely hanging in, feeling like day two of the water fast. Energy was up and down, with periods I felt OK, and periods I had to sit or lie down.

I did manage to get some work done during short stretches; however, at other times, my brain would shut off, and I couldn't even formulate sentences to speak. After "dinner" on day four, it felt as if I had turned a corner. I was able to do a few hours of solid work and then managed through 3x five-minute sets on the Thai bag at a reasonable pace. My body was no longer "tingling" when I experienced energy rushes, nor crashing moments later. Towards bedtime, I was exhausted, my head pounding once again, and I had intense cravings, although not as bad as days two and three.

Day 5: Shaky Start, Solid Finish

I woke up on day five feeling like a train had run me over. My muscles were sore from the 15 minutes on the bag, my head was spinning, and I lacked energy. It took me 45 minutes to get out of bed. My blood glucose and ketones were identical to day four, and I had lost another pound. I slowly got going and ate my entire daily allotment of almonds. I had a busy morning and needed to get through it. I was

conducting R&D scale-up attempts at my facility, dragging containers around, lifting buckets, barrels, and bags weighing ~50-100 lbs, and once I got into a workflow, I felt fine. I worked up a huge sweat, and the more I worked, the better I felt. I put in a solid four hours of fairly strenuous manual work before heading home to take care of emails and writing, and so on.

By the time I got home, I was getting hungry. I drank 1.5 L of water, and that took care of my hunger pangs for a while. The last bit of my fast finished up the easiest. Right before finishing, I tested my ketones and blood again. My ketones were well above 8 mmol/L, significantly darker in the urine test, but not yet at the darkness tone of the next measurement threshold of 16 mmol. My blood glucose was 3.8 mmol/L or 68 mg/dl. It was hard to draw my blood; I failed twice. My hands were shaking, and I couldn't get the drop in the sensor.

Research on FMD

There is some early research on the FMD published. For instance, a randomized trial of 100 participants found benefits in the FMD in reducing body weight, body fat, and blood pressure (Wei et al., 2017). Several studies in rodents have found benefits in the FMD for improving the microbiota and function of the gastrointestinal system (Rangan et al., 2019; Zhou et al., 2019; Wei et al., 2018), which may contribute to observations of diabetic reversal in mice (Cheng et al., 2017). Elsewhere, the FMD has been studied for its role assisting chemotherapy treatment, specifically targeting cancerous cells (Vernieri et al., 2019), and in assisting in various autoimmune diseases (Choi, Lee, & Longo, 2017; Choi et al., 2016). As per Longo, numerous teams around the world are currently utilizing the FMD in studying numerous conditions, and I (and others) are anxiously awaiting further results and data from human trials.

Conclusion: Between Two Fasts

Personally, I don't view lifetime caloric restriction as a realistic or sustainable strategy for life extension. Not only is it psychologically and socially difficult to adhere to over the long term, but in older adults, it may carry real physiological risks due to muscle mass and nutrient intake. It's not just about living longer; it's about staying functional while you do.

As for the fasting-mimicking diet, it had both pros and cons as compared to the water fast. The experience was interesting, and so is the research data. From a purely personal standpoint, I found the three-day water fast easier, at least mentally. I felt foggier and less stable during the FMD, constantly hovering on the edge of irritability. That said, I was also far more productive. The FMD didn't knock me out intellectually the way the water fast did, at least not as frequently, but there was still a point where I couldn't formulate sentences, neither speaking nor writing. For some people, that alone could be the deciding factor, and for me, it was.

If my biomarkers are any indication, and they were strikingly similar between the two, then it's reasonable to consider that the FMD and a standard water fast may be different paths to the same destination. Whether the subtle, pulsing stress of micronutrient intake in the FMD confers additional benefits over total fasting is still an open question. I've heard arguments both ways, but that doesn't mean the route is identical. The FMD offers intermittent reprieve from the total stress of nutrient deprivation, and that pulsing—brief dips into safety before diving back into stress—may itself be a form of hormesis (Boccardi et al., 2023). We know from broader hormetic research that pulsed stress often produces more adaptive responses than continuous, unrelenting exposure (Li, Yang, & Sun, 2019). While early support for the FMD relied heavily on animal data and niche applications, such as adjunctive cancer care, that's no longer the case. Over the past decade, substantial clinical evidence has emerged demonstrating that FMD can confer meaningful health benefits in humans; ranging from reductions in biological age and insulin resistance, to improvements in cardiovascular and cognitive function (Newcomb, 2024; Boccardi et al., 2023; Mishra et al., 2023). Although direct comparisons between FMD and water-only fasting are still relatively limited, FMD has shown promise as a more sustainable and safer option for many, with added advantages in compliance and nutritional sufficiency (Rodrigues, 2024; Boccardi et al., 2023; Wei et al., 2017).

Still, despite this evidence, it makes more sense to view these as individual options rather than as competitors or complements. Don't assume one is inherently superior, as the right approach is the one that works for *you*, whether that means the structure and nutritional support of the FMD or the simplicity and depth of a water-only fast, depends on your biology, your lifestyle, and your psychology. The best intervention isn't the one that sounds most impressive on paper, it's the one you can do consistently without burning out or breaking down.

Picture 12. Landscaping at my acreage in Canada, done by me

Heat adaptation, Pacific Northwest edition.

CHAPTER 5:

Harness Radiation: Sunlight, Infrared Saunas, and Red Light Therapy

Introduction: The Electromagnetic Spectrum as Hormesis

Before we begin, let's get something out of the way: this chapter is not about breatharianism. It is not about the people who claim they can live on nothing but sunlight and "prana," which—depending on your Google search history—is either a vital cosmic force or a multi-level marketing scam in yoga pants. (Despite my perhaps warranted skepticism of this metaphysical concept, one of my editors, Vadim, urged me to consider prana seriously, and so I do so at the end of the next chapter and in Chapter 20 of *The Mind*).

Jasmuheen, one of the most famous proponents of the "living on light" movement, once claimed she could survive *on a single cup of tea and sunlight*. This belief was put to the test on national television in Australia—where she visibly began to suffer symptoms of dehydration and malnutrition within a few days, despite her cosmic assurances. Her answer was that the energy in the room was too negative (Marks, 1999). Apparently, sunlight-based nutrition is *mood-dependent*.

Let's be clear: attempting to photosynthesize as a human is not biohacking. It is delusion, and while I'm generally sympathetic to unconventional ideas in this book—especially those that challenge the modern comfort-industrial complex—I draw the line at metaphysical malpractice. Still, even ridiculous ideas sometimes sprout from the cracked pavement of something true. There *is* something essential about the sun, something many of us have forgotten.

Diogenes—ancient Greece's most accomplished troll, who lived in a clay wine jar and was known to defecate in public for the sake of philosophical "demonstration," knew this. When Alexander the Great, the most powerful man in the known world, visited him and asked if there was anything he could do for him, Diogenes didn't request riches or honor or protection. He just said:

> *Move a little to the right; you are blocking my sun.* (Wichmann, 2024)

This line, more than any modern infrared marketing brochure, captures what we're trying to recover here: not mystical dependence on sunlight, not pseudoscientific denial of food, but the sense that *sunlight is a need,* that there is something *nourishing*, maybe even sacred, about direct exposure to the elements, that maybe, just maybe, modern life has blocked too much of it out. With that in mind, let's begin.

When Light Becomes Medicine

The electromagnetic spectrum represents one of our most fundamental but underappreciated sources of hormetic stress. Most people think of radiation as

something out of a disaster movie—or at best, the invisible culprit behind sunburns and aging skin. However, from ultraviolet radiation in sunlight to the longer wavelengths of infrared and red light, these forms of radiation interact with our biology in profound ways, including positive ones. While excessive exposure can damage tissues, controlled doses trigger adaptive responses that enhance cellular function, improve energy production, and strengthen resilience.

When dosed right, in moderation, and with intention, these "stressors" can trigger a cascade of beneficial adaptations: enhanced mitochondrial function, increased ATP production, reduced inflammation, improved circulation, better sleep, and faster healing. In other words, light becomes *medicine*. This principle perfectly exemplifies the hormetic paradigm explored throughout this book—measured exposure to stress creates adaptive benefits, while either insufficient or excessive exposure leads to dysfunction. With light therapies specifically, the emerging science shows extraordinary promise when properly harnessed. In short: the sun might burn, but it also teaches.

Sunlight: Ancient Stimulus, Modern Applications

Humans evolved under solar radiation. We didn't evolve in caves, or deep underground, and we likewise didn't evolve to be nocturnal. We evolved under a massive nuclear reactor 93 million miles away, and our biology is deeply intertwined with this fact. The daily and seasonal rhythms of the sun necessitated our physiology to intricately align with these expectations.

Long before we understood anything about ultraviolet wavelengths or vitamin D receptors, our species spent millennia living by the sun: rising with it, working in it, and winding down as it set. Understanding this should lead to little surprise that recent science reveals sunlight interacts with our bodies in ways far more complex than previously understood. The sun wasn't just a clock; it was a physiological stimulus of staggering complexity. It still is, today, and we need to acknowledge this and optimize for it.

Ultraviolet Benefits Beyond Vitamin D

While most people understand sunlight triggers vitamin D synthesis, this represents only one of many biological responses. UVB radiation (290-320 nanometers [nm]) mediates vitamin D production when it strikes the skin, converting 7-dehydrocholesterol to pre-vitamin D3. However, recent research indicates sunlight provides benefits independent of vitamin D pathways.

In a groundbreaking 2017 study published in *Scientific Reports*, researchers demonstrated that UV exposure triggered the release of nitric oxide (NO) from skin cells, causing significant vasodilation and blood pressure reduction independent of vitamin D synthesis (Holliman et al., 2017). This mechanism may partly explain why geographic latitude correlates so strongly with cardiovascular disease risk, even after

controlling for vitamin D levels. Further, controlled UV exposure has been shown to modulate immune function through multiple mechanisms.

One key effect is T-cell regulation: moderate UV exposure increases regulatory T-cells that help prevent autoimmune responses (Schwartz, 2005). Think of this as the immune system's version of a brakes check. In addition, UV exposure triggers systemic endorphin release, improving mood and potentially reducing inflammatory signaling. At the same time, UV light activates neuro-hormonal pathways that reduce inflammation and boost your body's ability to repair itself more effectively (Slominski et al., 2018). Feeling better and healing better may be two sides of the same sunlit coin. Finally, morning sunlight, particularly its blue wavelengths, regulates our internal clock (or synchronizes circadian rhythms), triggering the cortisol awakening response and suppressing melatonin release (Robertson-Dixon et al., 2023). It's nature's version of a shift change: signaling the night crew to clock out and the day crew to take over with new tasks, new hormones, and a fresh physiological agenda.

The Dark Side of Sunlight: Photoaging and Carcinogenesis

Despite its benefits, uncontrolled solar radiation exposure represents a significant stressor exceeding our hormetic threshold. Photoaging, which is the premature aging of skin due to UV exposure, affects millions and stands distinct from chronological aging. UVA radiation (320-400 nm) penetrates deep into the dermal layer. This exposure induces oxidative stress through the generation of reactive oxygen species (ROS), which damage key skin proteins like collagen and elastin, leading to elastosis, where damaged elastin accumulates, much like termites eating through old wood (Szychowski & Skóra, 2021). The result is wrinkling, sagging, and that classic "I never wore sunscreen" texture. A 2013 study in *Clinical, Cosmetic and Investigational Dermatology* estimated that up to 80% of visible facial aging is due to UV exposure—not age, not stress, not bad genes, just sunlight (Flament et al., 2013). The correlation is particularly strong for wrinkles, with excessive sun exposure accelerating their formation by as much as a decade compared to protected skin. The damage is cumulative, and the threshold is lower than most people realize.

More concerning is the relationship between UV exposure and skin cancer. UVB radiation directly damages cellular DNA, creating characteristic thymine dimers that, if not properly repaired, lead to genetic mutations.

Among the most critical safeguards in the body's anti-cancer arsenal are tumor suppressor genes like *p53*. These genes act as cellular sentinels, monitoring DNA integrity and halting the replication process when damage is detected (Liu & Kulesz-Martin, 2001). However, when *p53* is compromised by UV radiation, oxidative stress, or random mutation, cells lose their ability to regulate division. What follows is unchecked proliferation, and over time, this breakdown in control can manifest as skin cancer.

The consequences depend on where the malignancy originates and how aggressive it becomes. In many cases, the result is basal cell carcinoma, the most common form of skin cancer. While rarely life-threatening, it behaves more like that overly familiar relative who never quite leaves: slow-moving, nonviolent, but constantly edging further into your space. It doesn't invade dramatically, but it persists, gradually demanding more attention and resources. Basal cell carcinoma is often more of a chronic nuisance than an acute danger, but left unaddressed, it can cause significant local damage (Daitch & Lane, 2023).

Squamous cell carcinoma takes things further. This form arises from the keratinocytes of the epidermis and tends to grow more rapidly. Unlike basal cell carcinoma, squamous cell cancers have a greater potential to invade surrounding tissue and, in some cases, metastasize to distant organs (Caudill, Thomas, & Burkhart, 2023). They demand quicker action and closer monitoring.

Then there's melanoma, the most dangerous and unpredictable of the three. It originates in melanocytes, the pigment-producing cells of the skin, and is notorious for its speed and stealth. Melanoma can develop rapidly and metastasize before it's even detected, making early diagnosis critical. If caught early, outcomes are good. If not, the prognosis becomes far more serious (Thompson & Williams, 2024; De Vellis, Pietrobono, & Stecca, 2021). This spectrum—from nuisance to lethal—highlights just how high the stakes are when genetic defenses like *p53* begin to fail.

The Sunscreen Paradox: Protection Without Deprivation

Sunscreen represents our primary defense against UV damage, but when considering the hormetic approach to sunlight, anything that inhibits the effects, such as sunscreen, deserves careful consideration. Regular sunscreen use has been conclusively linked to reduced skin cancer risk, with a 2018 study published in *JAMA Dermatology* finding that Australians aged 18–40 who regularly used sunscreen during childhood had a 40% lower risk of developing melanoma compared to those who rarely used it (Watts et al., 2018).

However, questions persist regarding whether sunscreen might interfere with the beneficial aspects of UV exposure, particularly vitamin D synthesis. Current evidence indicates this concern is largely unfounded. In particular, the concern that sunscreen stops vitamin D synthesis is a classic case of theory outpacing reality. In controlled lab settings, when an SPF 30 sunscreen is properly applied in the exact correct dosage, inhibition of nearly all UVB-induced vitamin D production can occur—up to 97% inhibition (Grigalavicius, Iani, & Juzeniene, 2016; Autier et al., 2000). That said, there is a massive caveat: *nobody applies sunscreen like that*. Most people use 25–50% of the recommended amount (Petersen & Wulf, 2014). Some miss entire body regions, while others sweat or swim it off, failing to reapply at the correct intervals. In real-world conditions, sunscreen application is not an impenetrable barrier—it isn't even a Maginot line, full of glaring holes—it is more like a flimsy barricade made of cardboard, and that isn't necessarily a bad thing.

It's important to note that even when used correctly, SPF 30 still allows around 3% of UVB rays through (Autier et al., 2000; Young, 2000). In certain climates and conditions, even with this dramatically reduced exposure, it is still possible to get a sufficient dose of UVB to drive Vitamin D synthesis, and without the risk of frying your skin. The takeaway isn't to ditch the sunscreen, it's to utilize it wisely, and intentionally. Protect the parts that age and burn quickly while you let the rest absorb the sun briefly, deliberately, and intermittently. Sunlight, like heat, cold, and hypoxia, is a powerful biological input; don't deprive yourself of it entirely—but don't be reckless with it, either.

In short, the protective effect of sunscreen against melanoma shows the importance of sun protection, especially early in life, without necessarily compromising other physiological benefits of sun exposure. As I have shown in this chapter, under real-world conditions, sunscreen use does not significantly impair Vitamin D synthesis, a critical consideration in the pursuit of good health. The optimal approach towards balancing the need for proper vitamin D synthesis, against the risks of photoaging and melanoma, appears to be consistent sunscreen application to cancer-prone areas (such as the face, ears, neck, and hands) combined with strategic, time-limited unprotected exposure, duration and intensity based on skin type and UV index. This strategy allows hormetic benefits while preventing damage, which is precisely the balance we seek with all adaptive stressors.

As for me, I'm not ashamed to admit it: vanity plays a role in my use of sunscreen. I apply it only on my face, and it's not out of fear of melanoma so much as a desire to mitigate wrinkle formation and pigment inconsistency as I age. I don't bathe in sunscreen, I apply it to my face once or twice a day, condition depending. I'm also keenly aware that I don't get sufficient Vitamin D from the sun, even prior to my incorporation of sunscreen. I supplement, because I *have* to. Even in the summer, with substantial exposure working next to a window, and being active outside, my levels barely cross into the lowest end of normal for serum Vitamin D. So for me, the theoretical debate about sunscreen blocking vitamin D is a nonissue, completely irrelevant to my specific situation and physiology. My approach is pragmatic: protect the most visible, photo-sensitive areas, and let the rest of the body engage with sunlight in short, intentional bursts. That's the spirit of hormesis, after all: not maximum exposure, not total avoidance, but smart interaction with stress that yields benefit instead of breakdown.

The Skin Cancer Paradox: Northern Latitudes and Melanoma Rates

The relationship between UV exposure and skin cancer incidence presents a fascinating and counterintuitive epidemiological puzzle. One might expect a direct correlation between solar radiation exposure and skin cancer rates, with equatorial regions showing the highest incidence. However, population data reveals a more complex picture.

Australia and New Zealand exhibit the world's highest melanoma rates, which aligns with their high UV index and predominantly fair-skinned populations (Sneyd &

Cox, 2013). However, Northern European countries—particularly Norway, Denmark, Sweden, and Finland—show melanoma rates that significantly exceed those of Southern European nations despite receiving substantially less annual UV radiation. You heard that right: Norway, Sweden, Finland, and Denmark all clock melanoma rates far higher than sunnier Southern Europe. In fact, Norwegians are about three times more likely to develop melanoma than Spaniards, despite receiving half the annual UV radiation (Petersen et al., 2015; Aase & Bentham, 1994). This is the so-called "Northern Paradox." As is often the case in biology, the answer isn't simple, but it is illuminating.

The relationship between latitude, sun exposure, and skin cancer isn't just about sunlight intensity, it's about how biology, behavior, and environment collide. The further north you go in Europe, the paler the average skin tone tends to be, though with notable exceptions. Populations in Wales, for instance, may exhibit darker complexions than their latitude would suggest, while some Italians and Greeks—despite their southern geography—can be extremely fair-skinned. These nuances don't undermine the general trend, but they do complicate simplistic assumptions. Fitzpatrick Skin Types I and II, which dominate in high-latitude populations, are less protected against ultraviolet damage, not only because they contain less melanin but because they lack the built-in resilience to recover from repeated UV stress (Del Bino, 2006).

That biological vulnerability is amplified by cultural habits. In countries where winter dominates the calendar, sunlight isn't a daily companion, it's a seasonal guest. When summer finally arrives or vacations take people south, the resulting sun exposure isn't gradual; it's a spike. Long months of overcast skies and artificial lighting suddenly give way to days on the beach under intense sun, and the skin isn't ready. This kind of intermittent UV exposure, especially when it leads to burns, is significantly more carcinogenic than consistent, moderate sunlight. The skin doesn't adapt. It suffers, and it sometimes mutates (Kricker et al., 1995). What's more, the risk doesn't vanish in the north. Heat waves have become increasingly common in recent decades. Even temperate metropolitan areas like Greater Vancouver now regularly face "heat domes," with summer temperatures soaring past 40°C on the humidex, sometimes even in actuality, in some parts of the suburbs, for extended periods. These extremes were virtually unheard of just fifteen years ago, further straining skin that isn't adapted to such sudden exposure.

Layered onto this is the issue of vitamin D. High-latitude populations are disproportionately prone to deficiency, especially during the darker months. While vitamin D is essential for bone health, it also plays a vital role in immune surveillance—the body's ability to detect and eliminate early cancer cells. Without sufficient sun-driven synthesis, this first line of defense may falter (Hayes et al., 2010).

There's also a hormetic dimension to consider. Low-dose, consistent exposure to ultraviolet light may actually strengthen the skin's repair mechanisms. Regular, moderate UV can upregulate DNA repair enzymes, enabling the skin to correct minor damage before it becomes a problem. However, intense, irregular exposure overwhelms these systems. Studies suggest that controlled UV exposure prompts protective adaptations, whereas the kind of acute overexposure that leads to sunburn actually inhibits repair and increases the chance of lasting mutations (Schirrmacher, 2021; Li et al., 2019).

Finally, behavior matters. Despite decades of awareness campaigns, indoor tanning remains surprisingly popular in parts of Northern Europe, especially among young women. In Norway, for example, nearly 70% of young adult women report regular tanning bed use (Kvittingen, 2017). These beds emit a concentrated dose of artificial UV directly to the skin, bypassing the gradual ramp-up nature once provided by seasonal sunlight. For skin already prone to damage, it's a perfect storm: vulnerable biology, extreme behavior, and an increasingly unpredictable climate.

The Northern Paradox doesn't just teach us about geography or genetics, it is yet another reminder for us that *dosing matters*. Avoiding sunlight altogether is not protective, and in fact is its own kind of risk, but overdosing intermittently, without acclimatization or caution, is a shortcut to irreparable damage.

As with most hormetic stressors, the goal is not maximal exposure, nor is it total avoidance; it's regular, moderate engagement, crafted meticulously and strategically for your unique biology, and conditions. For Northern populations, that may mean daily walks during daylight hours, gentle morning light whenever possible, and strategic, non-burn-level sunlight during spring and summer months. Sunlight, like exercise, cold, heat or fasting, is a potent stressor. Just like these other hormetic agents, how you use sunlight determines whether it promotes improved health, or drives accelerated deterioration. Yet, despite the mounting evidence for measured sun exposure as a powerful hormetic input, some self-styled health gurus have turned sun avoidance into a bizarre badge of honor.

It's hard to know whether to laugh or cry at the spectacle of Brandon Miles May: a 35-year-old "biohacker" who claims to have cracked the code of eternal youth by living like a low-budget vampire. While most people associate healthspan with strength, movement, and resilience, Miles has opted for full photophobia as a lifestyle. Armed with a "sunbrella," UV sensors, blackout curtains, and the social energy of a nocturnal housecat, he patrols his home avoiding slivers of sunlight as if they were radioactive waste. He reportedly hasn't enjoyed being outside since childhood, which makes his advice on longevity feel a bit like getting swim lessons from someone who's afraid of water (Guinness, 2024).

To his credit, Miles *does* look young, but so do most people who've spent their lives avoiding any physical strain, sunlight, or social contact. It's not so much the picture

of youth as it is the look of someone who's been flash-frozen since puberty. And while he assures readers that he's not missing out on life, his idea of balance involves measuring UVA levels in his living room and reminiscing fondly about *Interview with the Vampire* as a lifestyle model.

The irony? He's not alone. A growing cadre of digital-age immortality chasers are following in his shadowy footsteps, quite literally, convinced that shielding their epidermis from photons is the key to defeating entropy. What they miss, of course, is that real longevity isn't just about wrinkle prevention, it's about adaptation, strength, flexibility, and, of course, some sun. A tan line won't kill you, but living in a permanent game of hide-and-seek with daylight is likely to kill your spirit.

Infrared Sauna: Heat Plus Photobiomodulation

Infrared saunas represent a fascinating intersection of heat hormesis (discussed in Chapter 2) with the direct photobiomodulating effects of infrared radiation. Unlike traditional saunas that heat the air, infrared saunas use radiation in the near (NIR), mid (MIR), and far (FIR) infrared spectrum to directly penetrate and warm tissues.

Cellular Mechanisms Beyond Heat

Research indicates that infrared radiation affects cellular function through more than just heat, with one key mechanism involving mitochondrial stimulation. Near-infrared wavelengths, especially in the 810 to 850 nm range, can directly activate cytochrome c oxidase, the final enzyme in the mitochondrial electron transport chain (Vatansever & Hamblin, 2012). This activation enhances the production of ATP, increasing the cell's overall energy availability.

Infrared exposure also induces heat shock proteins (HSPs), which play a central role in maintaining protein homeostasis. These protective proteins are upregulated not only in response to mild thermal stress but also via direct photonic effects. Their presence supports proper protein folding, repair, and recycling, all of which are essential for cellular resilience under stress (Wang et al., 2017).

Another crucial pathway involves nitric oxide (NO) signaling. Infrared light can dislodge NO from hemoglobin and other storage sites, increasing its bioavailability. The resulting boost in NO improves microcirculation and tissue perfusion, facilitating better oxygen and nutrient delivery at the cellular level (Lohr et al., 2009). Together, these mechanisms suggest that the benefits of infrared therapy extend well beyond simple warmth.

Clinical Evidence for Infrared Hormesis

The clinical literature on infrared sauna therapy demonstrates benefits across multiple domains.

Cardiovascular Function

A 2018 systematic review and meta-analysis in *Clinical Cardiology* analyzed nine

clinical trials and found that regular infrared sauna sessions led to short-term improvements in cardiac function among patients with heart failure, including reductions in B-type natriuretic peptide levels and cardiothoracic ratio, as well as improvements in left ventricular ejection fraction (Källström et al., 2018). Notably, these improvements paralleled those seen in aerobic exercise programs, suggesting infrared exposure acts as a form of "passive cardiovascular exercise."

Detoxification Pathways

Unlike many unfounded "detox" claims, infrared sauna-induced sweating has demonstrated elimination of certain environmental toxicants through controlled studies. In particular, multiple studies have shown that sweat contains higher concentrations of these substances than blood or urine, suggesting the body may use the skin as an active elimination channel under certain thermal conditions (Cho et al., 2023; Kuan et al., 2022). Infrared heat also appears to stimulate phase II liver detox enzymes, giving your biochemistry a subtle nudge in the right direction (Crinnion, 2011). The hormetic stress of heat also upregulates phase II detoxification enzymes, improving the body's capacity to metabolize xenobiotics.

Practical Application and Optimal Protocols

Infrared sauna therapy appears most effective when guided by specific parameters that balance therapeutic benefit with safety. Research suggests that a session duration of 25 to 45 minutes provides sufficient exposure to stimulate adaptation without overtaxing the body (Hussain & Cohen, 2018). Unlike traditional saunas, which often exceed 170°F, infrared saunas operate at significantly lower temperatures—typically between 120 and 140°F (49 to 60°C) (Lindberg, 2024). This lower heat penetrates more deeply into tissues while placing less strain on the cardiovascular system, making it more tolerable for extended sessions.

Indeed, for people who can't tolerate high heat, because of medical issues, aging, their unique physiology, or even simple preference, infrared saunas are more accessible. Personally, I'm one of those people. Traditional saunas leave me lightheaded, sometimes even on the edge of fainting. Yet with infrared saunas, I can stay in longer, sweat more, and actually feel the benefits without flirting with heat stroke. They work much, much better for me. The real draw may lie in how infrared radiation interacts with our cells beyond just making us sweaty—through potential effects on mitochondrial function, circulation, and tissue repair. This is a different kind of heat, with a different kind of impact.

Frequency also plays a key role. Engaging in infrared sauna use three to seven times per week has been shown to produce meaningful physiological adaptations, including improved circulation, enhanced detoxification, and better stress resilience (Lindberg, 2024; Patrick & Johnson, 2021; Hussein & Cohen, 2018). The timing of these sessions can further tailor the benefits: evening use—ideally four to six hours before bed—tends to improve sleep quality by promoting

parasympathetic activity, whereas morning sessions may boost alertness and cognitive performance by raising core temperature and stimulating circulation early in the day (Patrick & Johnson, 2021).

Finally, hydration is critical. Because sauna use promotes sweating and fluid loss, consuming approximately 500 milliliters of water both before and after each session helps prevent dehydration and supports the body's detoxification processes (Agarwal, 2025). Taken together, these practices optimize the impact of infrared therapy while reducing the risk of adverse effects.

These recommendations must be adjusted for individual response, monitoring variables like heart rate, perceived exertion, and recovery time between sessions.

Tracking Your Response: Finding Your Infrared Hormetic Window

Identifying your optimal infrared sauna protocol requires more than simply following general guidelines; it demands personal tracking and feedback. One of the most immediate indicators is your energy level two to three hours after a session. Rather than feeling drained, you should notice a subtle boost in vitality and mental clarity, a sign that the session has enhanced your metabolic and neurological state rather than overstressed it.

Sleep quality is another crucial marker: a well-timed infrared session, especially in the evening, often promotes deeper, more consolidated sleep that same night. If you're waking up more rested or noticing fewer nighttime disturbances, it's a strong signal that the protocol is working for you. If you're waking up feeling exhausted, and your sleep data shows impaired deep sleep, you are in an activated sympathetic stress zone, and your protocol needs to be dialed back.

Recovery from other stressors—whether from physical training, cognitive overload, or emotional strain—also offers insight. Ideally, sauna use accelerates your return to baseline, shortening the recovery window and leaving you more resilient. Finally, resting heart rate the morning after a session provides an objective physiological readout. A stable or slightly lowered heart rate suggests your nervous system is in balance and adapting well to the thermal stimulus. Elevated heart rate, on the other hand, may be a sign of overuse or insufficient recovery time. By consistently observing these markers, you can fine-tune the frequency, duration, and timing of your sessions to match your unique needs.

Red Light Therapy: Precision Photobiomodulation

Red light therapy, also called photobiomodulation therapy (PBMT), uses specific wavelengths of visible red (620-700 nm) and near-infrared light (700-1100 nm) at precise intensities to trigger cellular responses. Unlike broader-spectrum infrared saunas, red light therapy delivers targeted wavelengths with specific therapeutic outcomes.

Mechanisms of Action: The Cellular Response

The cellular effects of red light therapy, particularly in the red and near-infrared (NIR) spectrum, are driven by a set of well-characterized mechanisms that go far beyond simple illumination. At the core is the activation of cytochrome c oxidase, a key enzyme in the mitochondrial electron transport chain. When red and NIR light penetrate tissue, they dislodge inhibitory nitric oxide (NO) molecules that are temporarily bound to this enzyme. The release of NO removes a brake on mitochondrial respiration, allowing for enhanced oxygen consumption and increased production of ATP—the cell's primary energy currency.

This photodissociation of nitric oxide doesn't just benefit mitochondria. Once freed, NO resumes its role as a signaling molecule, promoting vasodilation and improving cellular communication. Tissues experience better perfusion, and oxygen and nutrient delivery are optimized in the aftermath of light exposure.

Another essential piece of the puzzle involves reactive oxygen species (ROS). Unlike the damaging oxidative bursts associated with chronic stress or pollution, the ROS produced during red light therapy are tightly regulated and short-lived. These low levels serve a signaling function, triggering the activation of transcription factors like NRF2. In turn, this upregulates the body's own antioxidant systems, effectively boosting resilience from the inside out.

Red light also interacts with ion channels—specifically a class called transient receptor potential (TRP) channels, which respond to changes in temperature, pressure, and light. Certain wavelengths activate these channels, modulating calcium flux across the cell membrane. Since calcium ions are central to processes ranging from muscle contraction to gene transcription, this mechanism helps explain red light's wide-ranging therapeutic effects. Taken together, these processes show that red light therapy isn't just energy input, it's a molecular recalibration.

These mechanisms explain why specific wavelengths, particularly those centered around 660 nm (red) and 850 nm (NIR), demonstrate notably stronger effects than other portions of the spectrum.

Tissue-Specific Benefits and Applications

Red light therapy affects various tissues differently, with substantial evidence supporting specific applications:

Skin Regeneration and Collagen Synthesis

Red light at 630-660 nm penetrates the epidermis and upper dermis, where it stimulates fibroblast proliferation and collagen production. Multiple clinical studies have confirmed its benefits: Alexander Wunsch and Karsten Matuschka (2014) observed significant improvements in skin texture, collagen density, and wrinkle reduction after 30 sessions using 611–650 nm red light; more recently, Sang Park,

Seong Park, and Jae-A Jung (2025) reported that over 69% of participants showed global aesthetic improvements after 16 weeks of red and near-infrared LED treatment using a home-based device. While variability in study design and treatment parameters persists, these findings affirm the therapeutic potential of red light for skin rejuvenation when protocols are appropriately applied. The hormetic nature is evident here: insufficient intensity produces no effect, while excessive power density causes inhibition rather than stimulation.

Muscle Recovery and Performance Enhancement

Near-infrared light in the 800–850 nm range penetrates into skeletal muscle, where controlled trials have demonstrated enhanced mitochondrial dynamics, reduced DOMS, and faster recovery (Ferraresi, Huang, & Hamblin, 2016). In competitive cyclists, applying NIR photobiomodulation before exercise has been shown to improve time-to-exhaustion by approximately 10–20% in a randomized controlled trial (Lanferdini et al., 2023). The literature supports two primary strategies: muscular pre-conditioning and post-exercise application. Pre-conditioning, especially when timed several hours before exertion, may prevent muscle damage and enhance performance. Meanwhile, immediate post-exercise exposure has been shown to accelerate recovery and support more efficient training adaptations, promoting increased muscle growth. Both approaches work, but for different ends: one primes the system, the other repairs it.

Cognitive Function and Neuroprotection

Perhaps most fascinating is emerging evidence regarding transcranial photobiomodulation, where near-infrared light (specifically 810 nm) penetrates the skull and enhances cerebral mitochondrial function. Clinical trials have demonstrated improvements in reaction time, memory retention, and executive function following transcranial NIR sessions (Zeng et al., 2024; Lee, Ding, & Chan, 2023). Animal models further suggest neuroprotective effects against traumatic brain injury and neurodegenerative conditions (Stevens et al., 2024).

Thyroid Function

Several controlled trials show significant improvements in thyroid function with specific red light protocols. One study demonstrated that 10 sessions of 830 nm light therapy reduced thyroid peroxidase antibodies by 39% and allowed 47% of participants to reduce their levothyroxine dose (Höfling et al., 2013).

Limitations and Cautions

Like all hormetic interventions, light-based therapies offer powerful benefits, but only within a narrow therapeutic window. Misapplied, they can backfire. One potential concern, though sometimes overstated, is ocular safety. With high-intensity red or near-infrared light, particularly in concentrated devices like lasers or certain medical-grade panels, prolonged direct exposure to the eyes may present a risk to sensitive

retinal tissue (Fickman, 2024). Although laboratory analyses like the study on which the Fickman article is based suggest that uninterrupted 3-minute red-light exposures may approach or exceed theoretical safety thresholds for retinal photochemical and thermal injury, real-world clinical data from over 2,300 children treated with two 3-minute RLRL sessions (separated by at least four hours) have shown no irreversible vision loss or structural damage, only transient afterimages, and isolated case reports of reversible outer-retinal changes that resolved upon discontinuation (Chen at al., 2024). Beyond this, in most consumer-grade red light therapy beds, facemasks, and stand-up pods, eye protection is rarely used and generally not required. These devices typically emit light in the 600–850nm range, which lacks the ionizing energy of ultraviolet radiation and has not been conclusively shown to cause ocular damage at typical intensities (Cleveland Clinic, 2021). Still, those with light sensitivity or pre-existing eye conditions may prefer to avoid prolonged direct exposure, and some choose to wear goggles or keep their eyes closed as a precaution.

Photosensitivity reactions remain a more consistent concern. Certain medications—such as tetracyclines, retinoids, and psoralens—as well as specific medical conditions, can heighten reactivity to light (Odorici, Monfrecola, & Bettoli, 2021; Ibbotson, 2018). In cases of sensitivity, even low-level red or UV exposure can provoke irritation or more severe phototoxic responses. Screening for these sensitivities in advance is an important part of using light therapy safely. As with any light-based exposure, ultraviolet light carries its own risks, particularly when misused. While regular, moderate sun exposure is associated with positive effects on vitamin D synthesis and immune regulation, excessive high-intensity UV, especially in fair-skinned individuals (Fitzpatrick Types I and II), increases the risk of melanoma and other skin cancers (Uçar, & Holick, 2025; Mason & Reichrath, 2012). For those populations, sunburn isn't just unpleasant, it's an accumulative, carcinogenic insult.

Certain body regions also warrant extra caution. In individuals with autoimmune thyroid conditions like Hashimoto's thyroiditis, photobiomodulation (PBM) has shown potential benefits, particularly when applied at lower fluences and combined with supportive therapies. Clinical studies report improved thyroid function, reduced antibody levels, and even decreased reliance on thyroid hormone replacement (Berisha-Muharremi et al., 2023; Ercetin et al., 2020; Höfling et al., 2018). However, for hyperthyroid conditions such as Graves' disease, published clinical trials are lacking. While there is no direct evidence of harm, theoretical concerns remain: PBM's stimulatory effects on cellular metabolism and blood flow might exacerbate hyperthyroidism by further stimulating hormone production, potentially causing additional harm to an overactive gland (Noghabaei et al., 2024). As a precaution, PBM should be applied cautiously in such cases by avoiding direct application over the thyroid and ideally under the supervision of an endocrinologist or as part of a research protocol (THOR Photomedicine Ltd, 2025). The thyroid is highly responsive to mitochondrial input, and while this can be therapeutic in some contexts, it may aggravate symptoms in others. Careful monitoring is advised.

Finally, and perhaps most critically, is the importance of understanding the dose-response curve. Red light therapy follows a biphasic pattern: low to moderate exposure can stimulate beneficial adaptation, but more is not always better. Users who don't see immediate results often assume that increasing intensity or duration will help. Instead, they may push themselves into the inhibitory range, where the stimulus overwhelms the adaptive response and undermines the benefits. Light therapy, like exercise or fasting, depends on timing, dosage, and recovery. Without those, even a good tool can work against you.

Conclusion: Light: The Oldest Signal in the Book

The evolutionary significance of our relationship with the electromagnetic spectrum cannot be overstated. From cellular respiration to circadian rhythms, light signals have shaped human physiology over millions of years. Modern technology now allows us to isolate specific aspects of this relationship, delivering precise wavelengths at controlled intensities to trigger desired adaptive responses.

When applied correctly, light-based hormetic therapies offer a uniquely elegant set of advantages. Unlike pharmacological interventions or invasive procedures, they work without breaching the skin or introducing foreign substances into the body. The mechanism is purely energetic—light photons interacting with cellular components—making the process both non-invasive and remarkably well-tolerated.

One of the standout features of light therapy is its precision. With modern devices, users can control key variables with exacting detail: wavelength, intensity, and duration can all be tailored to specific tissues and therapeutic goals. This level of control is rare in the world of hormesis, where dosage is often difficult to quantify and responses can vary dramatically. Equally important is how well light therapy complements other stress-based interventions. Whether paired with cold exposure, resistance training, or fasting, light doesn't compete, it synergizes. It supports mitochondrial efficiency, enhances circulation, and upregulates antioxidant defenses, making it a natural ally to other resilience-building practices.

Perhaps most compelling, however, is its long-term viability. Many interventions lose their potency over time as the body adapts. However, with proper cycling and individualization, light-based therapies appear to maintain their effectiveness without triggering the same diminishing returns. They offer sustainable support for cellular health—no needles, no pills, and no crash—just light, harnessed with care and precision.

As with all hormetic interventions discussed in this book, the key lies in understanding the narrow therapeutic window and implementing protocols that provide sufficient stress to trigger adaptation without overwhelming recovery capacity. When this balance is achieved, light-based therapies become powerful tools for enhancing resilience and optimizing function across multiple physiological systems.

CHAPTER 6:

Oxygen: The Breath That Builds You

I've come close to dying three times: once from losing oxygen, and twice from being in situations where losing oxygen was moments away.

The first was when I was twelve or thirteen, caught in a riptide while surfing. I was giving everything I had to fight the current pulling me toward the rocks, but I wasn't winning. My dad wasn't nearby, and his partner—a woman I lived with through high school, who would be part of our family for years—dove in to help me. She got stuck, too. We were both in full panic, swimming as hard as we could just to stand still. It wasn't until a group of surfers saw us and jumped in that we got out alive.

The second time was quieter, but in some ways more frightening. I was nineteen, living on my own and home alone, and distracted while eating a steak. A chunk got lodged in my throat, and I couldn't breathe. I was choking hard, face red, eyes bloodshot, and no one was there to help. Instinct took over—I slammed my fists into my solar plexus over and over until I finally dislodged it. I just sat there afterward, rattled. Not from pain, although I recall my throat did hurt, but from how close I'd been to blacking out, which would have meant death.

The third time, I was twenty and thought I could handle more than I actually could. A friend and I set out on a long swim, maybe a kilometer, from a dock to the opposite shore. Neither of us had ever swum that far before. Halfway through, we both realized we were in real trouble. We were too far to turn back, and too far from help. We were probably drinking or hungover, too, which didn't help. We just kept going, which was our only option, and the option that had to work. By the time we reached land, we were wrecked. We both slumped down into the dirt on the shore for what felt like an hour before either of us could walk.

Each of those moments taught me something different, but the one common thread was the absolute clarity that comes when you're deprived of oxygen. Nothing else matters; not goals, not stress, not identity—just the next breath. That's the thing about oxygen. We take it for granted until it's gone. Yet beneath every physical feat, every cognitive effort, every biological system, oxygen is the currency. It's what powers us, and when manipulated strategically, it becomes one of the most potent hormetic stressors we have access to. This chapter is about how to use it—carefully, rhythmically, and with respect.

Oxygen is non-negotiable, it's the foundation of life, and it's also one of the most potent stressors your body ever deals with. That makes it a textbook example of hormesis: too little and you die, too much and you're damaged, but just the right amount at the right time? You adapt, you get stronger, you thrive.

We can choose whether or not to fast. We can decide to plunge into the cold or push our limits in the gym. With oxygen, there is no choice. It's always there—fueling, stressing, and signaling. That's why this chapter matters. Manipulating oxygen isn't just another biohack, it's a fundamental lever for resilience, and learning how to control it might be one of the most powerful things you can do for your biology.

The Oxygen Paradox

Oxygen serves as the terminal electron acceptor in our mitochondrial respiratory chain, enabling the production of ATP, our cellular energy currency. Without it, aerobic metabolism ceases within minutes, resulting in rapid cell death. Yet oxygen is inherently reactive, constantly forming potentially damaging by-products known as reactive oxygen species (ROS) that can damage DNA, proteins, and lipids. This dual nature creates what scientists call the "oxygen paradox"; we cannot live without it, yet it slowly contributes to our demise through oxidative damage.

Our adaptive response to this paradox has been remarkable. Throughout evolution, we've developed sophisticated antioxidant defense systems, DNA repair mechanisms, and cellular stress responses that manage oxygen's harmful effects while harnessing its life-giving properties. This delicate balance represents perhaps the most fundamental hormetic relationship in our physiology—one we can strategically manipulate for enhanced resilience and performance.

Hyperoxia: Strategic Oxygen Excess

Hyperbaric Oxygen Therapy (HBOT)

Hyperbaric oxygen therapy involves breathing pure oxygen while inside a pressurized chamber, typically at 1.5-3 times normal atmospheric pressure. This dramatically increases the partial pressure of oxygen in your tissues—up to 15 times normal levels—triggering profound physiological adaptations.

The hormetic nature of HBOT is well-documented. At therapeutic doses, it initiates controlled oxidative stress that stimulates antioxidant defenses, promotes angiogenesis, which is the formation of new blood vessels, enhances mitochondrial function, and mobilizes stem cells. Yet excessive exposure leads to oxygen toxicity, seizures, and lung damage, demonstrating a classic hormetic response curve.

HBOT has demonstrated its effectiveness across a range of clinical conditions, with strong evidence supporting its use in several areas. For decompression sickness, HBOT is the gold standard, as rapid pressurization reduces inert gas bubbles, alleviating symptoms and preventing long-term neurological damage, which is supported by its essential role in treating divers (Kahle & Cooper, 2023). In diabetic foot ulcers, a 2021 meta-analysis found HBOT significantly improved healing rates and reduced major amputations. Specifically, people treated with HBOT were more likely to heal their ulcers fully and were 40% less likely to need a major amputation (Sharma et al., 2021). For radiation injuries (especially those involving soft tissues

post-cancer therapy), a Cochrane review concluded HBOT enhances angiogenesis and wound repair, reducing long-term damage in head and neck cancer patients (Lin et al., 2023). HBOT has also been used successfully in necrotizing soft tissue infections, where its ability to inhibit anaerobic bacteria and reduce mortality has been documented in retrospective studies, including one showing significantly better survival among patients who received adjunctive HBOT (Wilkinson & Doolette, 2004). Finally, though its role in treating traumatic brain injury (TBI) remains under investigation, randomized trials have reported cognitive improvements and symptom relief in patients with persistent post-concussive symptoms following HBOT, suggesting there are potential future clinical applications in this area (Weaver et al., 2025). Taken together, these findings demonstrate the value of HBOT as a versatile tool with applications in both acute and chronic care.

Research by Dr. Shai Efrati's team at the Sagol Center for Hyperbaric Medicine and Research in Israel has shown promise for age-related cognitive decline, with evidence suggesting that controlled HBOT protocols can trigger neuroplasticity even in older adults. Their 2020 study in the journal *Aging* demonstrated improvements in cerebral blood flow and cognitive performance following strategic HBOT sessions (Hadanny et al., 2020). However, it's essential to understand that while HBOT may offer benefits, these are likely dependent on the duration and intensity of exposure, since too much oxygen can quickly become harmful. A 2022 review in *Cell Biology and Toxicology* reports that prolonged hyperoxia impairs mitochondrial function and increases mortality in animal models (Alva et al., 2022). This suggests that carefully regulated dosing is necessary to prevent oxygen toxicity (Alva et al., 2022).

The therapeutic window appears to involve intermittent exposure, typically 40-60 sessions of 60-90 minutes each at pressures between 1.5–2.5 atmospheres absolute (ATA), allowing for periodic oxidative stress (Leveque et al., 2023; Balestra & Kot, 2021; Jain, 2017). This is followed by recovery periods of at least 24 hours (Efrati et al., 2013; Hardy et al., 2007). This intermittent stress pulsing pattern maximizes adaptive responses while minimizing oxygen toxicity. Most clinical protocols achieving therapeutic benefits typically range between 1.5–2.0 ATA for 60–90 minutes, with protocols for neurological conditions often requiring the full course of 40+ sessions to achieve significant results. It's important to note that reviewers have indicated that there is no additional benefit from pressures exceeding 2.5 ATA, and that pressures exceeding 2.0 ATA should be approached cautiously due to potential neurotoxicity, despite the existence of research and protocols finding benefits up to 2.8 ATA. They advise that the same benefits are likely to occur at 2.5 ATA and even 2.0 ATA, with a safer therapeutic window (Leveque et al., 2023; Efrati & Ben-Jacob, 2014). The treatment plan should be personalized, and the total number of sessions should be tailored to the individual's condition and response, rather than applying a "one size fits all" approach. In short, HBOT is not just about getting "more air," it's about turning oxygen into a high-intensity signal for adaptation.

Different conditions demand tailored approaches within this hormetic envelope as well. For idiopathic sudden sensorineural hearing loss, the most evidence-supported regimen involves 100% oxygen at 2.0–2.5 ATA for 90 minutes daily over 10 to 20 days, reaching a cumulative exposure of at least 1,200 minutes (Murphy-Lavoie et al., 2012). Notably, increasing pressure beyond 2.5 ATA does not improve hearing recovery and may increase risk (Rhee et al., 2018). For chronic wounds and soft tissue injuries, protocols commonly employ two daily sessions at 2.3–2.8 ATA for 90 minutes, though clinical outcomes vary due to protocol heterogeneity. Studies generally report 6–32 sessions (average ~12), with the Undersea and Hyperbaric Medical Society (UHMS) recommending a pressure range between 1.4 and 2.8 ATA, most often between 2.0 and 2.8 (Kwee et al., 2024; Weaver, 2014). Although mechanistic studies suggest that pressures above 2.5 ATA may not provide additional physiological benefit, clinical use often still extends into that range, likely due to legacy protocols or personalization considerations in patient populations (importantly, it is up to the practitioners' and researchers' discretion to determine if any hormetic protocol in this book is appropriate; I acknowledge those who go above 2.5 may have merit, though there's no clinical evidence that it is more effective than 2.0 ATA in many cases, and increasing it above that threshold carries the aforementioned risks).[5] In post-stroke recovery, HBOT has been applied in 7–60 sessions lasting 30–90 minutes at pressures ranging from 1.3 to 2.5 ATA, though

[5] I would like to make it clear that I observe numerous shortcomings in the literature, and the conclusions derived from the literature. Since I am not an expert in this field, I am incapable of forming nuanced opinions on each conclusion, and as such, must rely on the reviews written. For instance, there is clinical evidence exceeding the 2.5 ATA which reviewers stated no evidence exists to surpass, however, I am unaware of any direct comparisons in these conditions between 2.5 ATA and 2.8 ATA. Additionally, a mechanistic study demonstrates that the regulatory effects on redox, i.e., antioxidant activity and ROS production, demonstrate no additional benefits beyond 1.4 ATA. However, the immunomodulatory, and inflammatory regulating effects work in a dose-dependent manner up to 2.5 ATA. Since the researchers did not test beyond 2.5 ATA, it would be impossible to conclude that these dose dependent effects stop at 2.5 ATA. Perhaps they continue to 2.8 ATA, or above, or perhaps they sharply decline after 2.5 ATA. We simply do not have the data to draw this conclusion. Finally, reviewers note that above 2.0 ATA neurotoxicity risks increase, with higher pressures offering little to no benefit. Since it has been established mechanistically that immunomodulatory and inflammation regulatory effects increase in a dose-dependent manner beyond 2.0, but redox regulation does not improve beyond 1.4 ATA, it is worth questioning if 1.4 ATA is the threshold that certain conditions should not utilize beyond. Furthermore, it could be that barometric pressure itself could be the primary trigger for HBOT's therapeutic effects, while supplemental oxygen serves to boost the consistency and magnitude of those effects. In other words, moving from 1.3 ATA (ambient air) to 1.5 ATA (100% O_2) enhances outcomes, but the incremental benefit of added O_2 plateaus beyond a certain pressure threshold (Marois et al., 2024; Harch, 2022). If future studies corroborate this pattern, HBOT could shift away from an oxygen centric model toward a pressure modulation paradigm, with oxygen functioning as an adjunct rather than the principal therapeutic agent. Again, my role here is simply to question the conclusions, based on the inconsistencies, and not offer any definitive guidance.

again, caution is warranted above 2.0 ATA due to increased neurotoxicity risk (Zhai et al., 2016; Efrati & Ben-Jacob, 2014). Across all use cases, the emerging consensus is clear: more pressure is not better. Personalization, not maximization, is key.

In short, there is no one-size-fits-all HBOT protocol, and treatment parameters must be tailored to each indication and patient. A recent mechanistic study comparing 60-minute exposures at mild (1.4 ATA) and high (2.5 ATA) hyperbaric oxygen levels found that both pressures elicited similar systemic increases in ROS production and antioxidant responses. However, immunomodulatory and inflammatory responses were dose-dependent, increasing proportionally with the higher oxygen dose at 2.5 ATA compared to 1.4 ATA. These findings suggest that while lower hyperbaric pressures can achieve key therapeutic effects such as ROS-mediated signaling and antioxidant activation, higher pressures may be necessary to induce stronger immune and inflammatory responses, potentially beneficial for certain conditions (Millar et al.,2022). Therefore, careful optimization of HBOT dosing and inter-dose recovery timing is essential to maximize therapeutic benefits while minimizing oxidative stress-related risks. Extra caution is warranted in neurological indications, where pressures above 2.0 ATA carry the risk of neurotoxicity.

Oxyhydrogen Caution: When More Isn't Better

An important cautionary example of oxygen hormesis gone wrong comes from prolonged use of "Hydroxy" devices, which are portable hydrogen generators that produce a mixture of H_2 (hydrogen) and O_2 (oxygen) gas through water electrolysis. While hydrogen gas has demonstrated therapeutic potential with a remarkable safety profile (as detailed in Chapter 8), these devices inadvertently combine it with elevated oxygen concentrations approaching 30-35%, far above the 21% found in ambient air (Lin et al., 2020).

This creates a problematic scenario where users seeking hydrogen's benefits unknowingly subject their lungs to chronic hyperoxia. While short-term elevated oxygen presents minimal risk to healthy individuals, long-term exposure fundamentally alters pulmonary physiology. Multiple studies have demonstrated that prolonged exposure to elevated oxygen concentrations damages the delicate alveolar epithelium, initiates inflammatory cascades, and ultimately leads to interstitial fibrosis.

In particular, prolonged exposure to oxidative stress can inflict direct damage on the surfactant function, essential for maintaining alveolar stability and efficient gas exchange. Surfactant function can be impaired by reactive oxygen species through oxidative damage to its lipid and protein components, potentially disrupting surface tension regulation and reducing pulmonary compliance (Pace et al., 2009; Rodríguez-Capote et al., 2006). Concurrently, oxidative insults activate nuclear factor kappa B (NF-κB), a central regulator of inflammatory signaling in lung tissue. This transcription factor promotes the expression of pro-inflammatory cytokines and

adhesion molecules, amplifying local tissue damage and setting the stage for chronic inflammation (Rahman & Adcock, 2006). At the same time as this immune response, alveolar epithelial cells start changing in a way called epithelial-to-mesenchymal transition (EMT), where they lose their original features and take on a fibroblast-like appearance, marking an early and important step in the development of pulmonary fibrosis (Kim et al., 2006). Over time, these cellular and molecular changes culminate in structural remodeling of the lung parenchyma, including progressive thickening of the alveolar-capillary membrane. This thickening compromises the efficiency of oxygen diffusion across the pulmonary interface, contributing to declining respiratory function and clinical symptoms of hypoxia (Mach et al., 2011).

A 2017 study on human adult cardiac myocytes found that 72 hours of sustained hyperoxia led to significant increases in inflammation and cytotoxicity, suggesting that even relatively short-term exposure to elevated oxygen levels can provoke harmful cellular responses—well below the extremes used in many clinical and consumer-grade oxygen delivery systems (Hafner et al., 2017). Extrapolate this to months or years of daily use, and the potential for harm becomes significant.

This example perfectly illustrates why context, dosing, and recovery periods are critical for any hormetic intervention. The same oxygen that revitalizes tissues in properly administered HBOT protocols can insidiously damage lungs when chronically elevated without adequate recovery periods. For those interested in hydrogen therapy, molecular hydrogen gas generators (without oxygen enrichment) or hydrogen-dissolved water provide safer alternatives.

Hypoxia: Strategic Oxygen Limitation

While hyperoxia strategically stimulates antioxidant defenses and vascular remodeling, its antithesis—hypoxia—leverages an entirely distinct set of adaptive mechanisms that further illustrate the power of oxygen fluctuations as a hormetic stimulus. Strategic oxygen limitation has emerged as a powerful hormetic stressor at the opposite end of the spectrum. While chronic hypoxia is unequivocally harmful, intermittent hypoxic exposure triggers remarkable adaptive responses.

Altitude Training and Intermittent Hypoxic Exposure

Unless you live in the mountains or can afford to train there full-time, altitude training isn't exactly accessible. That's where intermittent hypoxic training (IHT) and intermittent hypoxic exposure (IHE) come in. Using specialized equipment, you can simulate high-altitude conditions without leaving your living room. IHT involves training while breathing air with reduced oxygen (typically around 13–16%), while IHE is done at rest, alternating between low-oxygen and normal air.

Elite endurance athletes have long recognized the benefits of training at altitude, where lower oxygen partial pressure forces physiological adaptations. These include

increased red blood cell production, enhanced capillary density, improved mitochondrial efficiency, and upregulation of hypoxia-inducible factor 1-alpha (HIF-1α)—a master regulator of oxygen homeostasis. However, altitude training presents practical challenges for most individuals. Enter intermittent hypoxic training (IHT) and intermittent hypoxic exposure (IHE)—protocols that strategically reduce oxygen availability through specialized equipment.

IHT involves exercising while breathing oxygen-reduced air (typically 13-16% oxygen), while IHE consists of sitting at rest breathing hypoxic air interspersed with normal air. Both methods have demonstrated significant benefits. A 2023 meta-analysis published in *BMC Sports Science, Medicine, and Rehabilitation*, analyzed 27 studies and found that structured intermittent hypoxic training IHT protocols significantly improved VO_2 max in trained individuals, indicating measurable gains in endurance performance (Huang et al., 2023b) Meanwhile, Hun-Young Park and Kiwon Lim (2017) conducted a systematic review analyzing the effects of normobaric hypoxia in middle- and long-distance runners and found that short exposures (less than 3 hours) significantly increased time to exhaustion. Complementing this, Anna Lukanova-Jakubowska and colleagues (2022) documented that an Olympic short-track speed skater who participated in four high-altitude training camps over two annual cycles achieved peak aerobic performance, with marked improvements in VO_2 max and anaerobic threshold.

These improvements are thought to be mediated in part by enhanced mitochondrial biogenesis and respiratory efficiency, as shown in rodent models by Yong-Kai Zhao, Wei Guo, and Bing-Hong Gao (2022), who reported upregulation of PGC-1α and other mitochondrial markers in response to hypoxic stress. Additionally, intermittent hypoxia stimulates erythropoietin (EPO) production, which in turn boosts red blood cell count and oxygen-carrying capacity—a mechanism confirmed in human trials (Wojan et al., 2021). This process is a natural, physiological adaptation to altitude, but synthetic EPO has been used illegally as a performance-enhancing drug in endurance sports to boost red blood cell mass and VO_2 max (Alberdi-Garciandia, 2025), most famously by Lance Armstrong (who was stripped of his medals for using it), which is why synthetic EPO is prohibited by the World Anti-Doping Agency (World Anti-Doping Agency, 2024).

Beyond hematologic and muscular benefits, metabolic improvements have also been documented: Tetiana Serebrovska and colleagues (2017) found that controlled hypoxic exposures could improve glucose homeostasis in prediabetes patients, potentially preventing type 2 diabetes development. Perhaps most surprisingly, cognitive domains may benefit as well. Urike Bayer and colleagues (2017) found significant improvements in cognitive performance in geriatric participants exposed to short bouts of hypoxia, hypothesizing that mild oxygen primes neuroplastic pathways.

These protocols activate ancient, deeply embedded systems that evolved to help us survive when air was scarce, and those same systems can now be tapped to improve metabolic efficiency, resilience, and even brain function (Burtscher et al., 2024). When done right, it's not deprivation, it's a form of precision stress. Just as with all other forms of adaptive stress, the timing and dosing of hypoxic exposure prove critical. Short bursts (3-5 minutes) of moderate hypoxia (15-16% oxygen) interspersed with normal breathing appear to maximize benefits while minimizing stress. This pattern allows cellular stress response pathways to activate without overwhelming adaptive capacity.

Breath-Holding and the Mammalian Dive Reflex

Perhaps the most accessible form of intermittent hypoxia comes through breath-holding exercises, which activate our innate mammalian dive reflex, which is an evolutionary adaptation that optimizes oxygen utilization during underwater submersion. When we hold our breath, particularly with our face immersed in cold water, a cascade of remarkable physiological changes occurs. One of the most immediate responses is bradycardia, a marked slowing of the heart rate up to 30%, which reduces oxygen consumption by the cardiac muscle and helps preserve oxygen for critical organs (Caspers, Cleveland, & Schipke, 2011). Simultaneously, peripheral vasoconstriction occurs, rerouting blood away from the limbs and toward vital areas like the brain and heart—an intelligent prioritization system that maintains oxygen delivery where it matters most (Godek & Freeman, 2022). Another key component is splenic contraction, which releases a temporary surge of red blood cells into circulation, thereby boosting the oxygen-carrying capacity of the blood during the apneic episode (Persson et al., 2023). Alongside these circulatory shifts, skeletal muscle mitochondria exhibit reduced respiratory capacity and metabolic activity, effectively downregulating cellular processes to conserve oxygen and energy during prolonged hypoxia (Kjeld et al., 2018). Over time and with training, individuals can also develop an enhanced tolerance to carbon dioxide, allowing them to function more effectively under conditions of elevated CO_2—a shift that increases respiratory efficiency and delays the urge to breathe (Elia & Lemaître, 2025). These orchestrated responses reveal a deep evolutionary intelligence embedded in the body's stress systems, allowing for survival under conditions of limited oxygen availability.

These responses optimize oxygen utilization under stress and appear to strengthen with consistent practice. Freedivers routinely demonstrate exceptional physiological control, with elite practitioners able to hold their breath for over 10 minutes and dive to depths exceeding 100-200 meters on a single breath (Tetzlaff & Muth, 2024; Murphy, 2020). For the average person, implementing simple breath-holding protocols can provide meaningful hormetic benefits. One approach gaining scientific support is the "CO_2 table" technique, involving progressively longer breath-holds with short recovery periods, gradually building tolerance to hypercapnia (elevated CO_2) and intermittent hypoxia.

A 2024 study published in *Sports* found that static apnea training improved maximum breath-hold time, enhanced breath control (evidenced by improved lung capacity and reduced heart rate), and previous research suggests it may also increase hematocrit levels, which suggests a greater capacity for oxygen transport and overall diving performance (Bezruk et al., 2024; Schagatay, Haughey, & Reimers, 2005). What this means is better breathing, better energy, and faster bounce-back, and it costs you nothing but a little focus.

Oxygen Fluctuation: The Ultimate Stress Signal

The evidence increasingly suggests that neither chronic hyperoxia nor chronic hypoxia represents optimal physiology. Rather, fluctuation between these states, which mirrors our evolutionary experience of varying activity levels and environmental conditions, provides the most powerful hormetic stimulus.

This concept aligns with findings from the emerging field of "environmental conditioning," which suggests that our modern environment's stability may actually compromise physiological resilience. Our ancestors regularly experienced fluctuations in oxygen availability through varying activity levels, altitude changes, and environmental challenges. These fluctuations, rather than constant homeostasis, may have driven the development of our robust adaptive systems.

A 2023 review published in *Aging and Disease* found that exposure to mild hypoxia can induce metabolic quiescence and activate cellular stress resistance pathways, ultimately promoting longevity—suggesting that controlled oxygen restriction may enhance lifespan without provoking harmful chronic stress responses (Nisar et al., 2023). Oxygen sits at the center of both our vital functions and our aging processes. Learning to manipulate this fundamental element, through both strategic abundance and limitation, represents perhaps the most fundamental form of hormesis available to us.

This research clearly demonstrates that neither chronic hyperoxia nor chronic hypoxia optimizes human function. Rather, it's the controlled, intermittent exposure to both states that drives the most profound adaptations. This mirrors what we've observed with every hormetic stressor covered in this book; the adaptive capacity emerges not from constant challenge nor complete avoidance, but from rhythmic cycles of stress and recovery. As with all forms of hormesis, the key lies in finding your personal sweet spot—the level of oxygen challenge that stimulates adaptation without overwhelming your recovery capacity. Start conservatively, progress gradually, and always prioritize recovery. The resulting resilience may not only enhance performance but potentially extend the functional lifespan of every oxygen-dependent cell in your body.

Conclusion: The Rhythm of Breath

Oxygen isn't just essential, it's foundational. It fuels our metabolism, drives cellular energy, and—when mismanaged—accelerates our aging. When used with intention, oxygen becomes more than a necessity. It becomes a signal, a stressor, and a tool for growth.

In ancient traditions, breath wasn't viewed as just biology—it was life-force. In yogic philosophy, prana, though I had mocked it playfully in the previous chapter, is considered to be the vital energy carried through breath, woven into every function of the body and mind (Kanojia, 2024). To breathe consciously is to touch that force. Today, modern science is only now catching up, showing that when we train our breath, we train resilience. We don't just survive; we regulate stress, sharpen focus, and stretch our capacity for life.

What the research shows—and what ancient wisdom already knew—is that chronic exposure to either end of the oxygen spectrum doesn't serve us. Constant excess leads to oxidative damage. Constant deprivation leads to degeneration, but strategic fluctuation, meaning cycling between abundance and limitation, creates space for adaptation. That's the throughline of every hormetic practice in this book. Stress isn't the enemy, but rather *rhythmic*, well-dosed stress is what makes the system stronger.

As always, the real key is calibration. Your system is unique, and so is your recovery capacity. Start light, pay attention, and adjust based on how you sleep, think, recover, and feel. The goal isn't to prove how much you can tolerate, it's to trigger just enough signal to make every cell that depends on oxygen more capable, more efficient, and more resilient.

You already breathe. Now learn how to wield it to breathe in power.

CHAPTER 7:

Alcohol: A Useful Poison

Introduction: Clarity in Ethanol

There's a certain kind of clarity that arrives to me after a few glasses of wine; not always, but under the right circumstances: circumstances which I had learned to create. I needed to be alone, or, immerse myself in my own world for this. My favorite rituals involved a bottle of red wine, home alone, with introspective music playing and my laptop handy to write; a similar stream of red wine was poured during travel, with my noise-cancelling headphones playing introspective music, and my phone or laptop to write musings. Then, a bottle of red was drunk while cooking alone, with—you guessed it—introspective music playing. The latter sometimes led to ruined dinners, as when thoughts would arrive I'd forget I was cooking to rush to my computer or phone to write down my revelations.

What each of these specific routines had in common was that they created stillness, quieting my mind from the anger, contempt, and anxiety of life and business. This stillness allowed insights to emerge from the void, coalescing from the fragmented thoughts and experiences weighing heavily on me. This stillness gave me moments of clarity. For years, alcohol was the only tool I had to enter this space, so I abused it. During the ritual, a point always came where the stillness dissolved from revelatory to blurred and incomprehensible. My thoughts would deteriorate, my intellect would abandon me, and I'd be left in an entirely different state, typically one of unbridled emotion, although interestingly, never the emotions I was trying to strip, but often self-aggrandizing confidence, or maybe hedonistically driven enthusiasm, and in rare, but notable cases, deep sorrow. Never anger, contempt, or anxiety.

As I leaned into the drink more and more, the benefits I received from it began to vanish. There is a certain culture to drinking, and the emotional and intellectual fondness I maintained for my ritual led to me indulging out of ritual; with friends, family, and my fiancée. Indulging in company has an almost opposite effect on me, my mind races with conversation and excitement. As time went by, I forgot the reason why I had started drinking altogether. So, I quit, and after the fog cleared my mind started working again, and in time I realized that the stillness alcohol led me to in the past was accessible sober. I'm not sure what changed, but my mind is quieter, calmer, and less anxious than it was before. The crutch of alcohol is no longer needed, and has become nothing but a hindrance.

My experience is not unusual, countless writers, thinkers, philosophers, and scientists have been here before. Henry Miller once said, "Write drunk, edit hungover."[6]

[6] This quote is sometimes attributed to Ernest Hemingway, another drunk.

Picture 13. Alcoholism, pt. 1

Photosynthesis? More like photodrunkthesis.

Amusing advice, although personally, I'd prefer to edit once the hangover's gone. There's no shortage of examples—Ernest Hemingway, Charles Bukowski, Christopher Hitchens, all drank to excess. The times I have met Aubrey De Grey, a prominent biogerontologist, he's been verging on the inability to stand or sit up straight. One of these times, he managed to stagger to the stage, and handily trounce a professor from Stanford in a debate. Similarly, one of my editors mentioned Guy Debord, a man who drank not just to numb himself, but to *reach* something, something sharp, something essential.

Debord wrote:

> *Of the few things I have liked and known how to do well, what I have assuredly known how to do best is drink… I have written much less than most people who write, but I have drunk much more than most people who drink.* (Biblioklept, 2009)

I've read more than most, but less than I would like. I've written enough content for a lifetime, but finished fewer of my writing projects than I care to admit, stranding my thoughts, books, and written aspirations in the land of mostly finished, never perfected. During this, I've definitely drunk more than I should have—likely a large contributor to both shortcomings. For a long time, I thought alcohol helped: it made

conversation easier, and writing easier. In particular, it calmed my anger, useful both for writing and for people, perhaps dumbing me down to some level where I could suddenly relate.

This is not to say I regret the years I spent drinking. I don't believe in regret, only learning and moving forward. Without my learning experiences, many of which were undoubtedly foolish, I would not be the man I am today. I *like* who I am, and am proud of who I am, so I cannot logically regret my past. That said, liking who you are does not obligate you to perpetuate the less-than-optimal decision-making processes you employed in the past. I have radically reduced my alcohol intake, because I have learned from my past, and can no longer justify the cost.

The cost is not just to the body, it's to the mind. It's to cognition, to performance, and purpose. Eventually, the alcohol that once opened doors started closing them. This didn't happen all at once, just slowly enough that I could pretend it wasn't happening, until it did, and I could no longer ignore it. This chapter isn't a confession, it's not a warning label, it's a confrontation—with the way we use alcohol to simulate things we haven't built yet: ease, community, clarity, and peace. It worked for a while, or so I thought, until it became obvious that it didn't.

Picture 14. Alcoholism, pt. 2

When the wine collection outgrows the book collection, it's time for edits.

In short, alcohol's not your friend, but it doesn't have to be your enemy either. It's your occasionally useful adversary. Like all adversaries worth engaging, the only way to benefit is through respect, control, and ruthless honesty about the limits—all of which require a modicum of understanding.

Picture 15. Walking in the forest, trying to unblock writer's block

Learning how to walk again. How to breathe, how to be. One step, one leaf, one moment at a time.

Every year the media seems to shout out to the world a new "definitive truth" regarding alcohol. Either it is "good for us" or "unequivocally bad for us." One of the issues with the media in general, regardless of whether it is health and science reporting, or politics, is that there is a need to create attention-grabbing headlines. The problem isn't just the headlines, either: it's that most journalists writing these pieces don't actually understand the studies they're summarizing. They're trained in storytelling, not biochemistry, and so what you get isn't interpretation, it's distortion:

Picture 16. Same forest stroll

Turns out the view's better when you're not seeing double.

a game of scientific telephone played for clicks, not clarity. Since most journalists lack a background or understanding of the science they are covering, they have a tendency to either accept conclusions without scrutiny, or reject them entirely as a form of designed opposition to their ideological adversaries, with the end result being that the messaging ends up extreme and polarized.

Alcohol is the perfect subject for distortion, and this is because almost everyone drinks, and almost everyone wants to know whether that drink is helping or hurting them. So when a new "definitive" claim surfaces, it doesn't just make the rounds, it goes both viral and *tribal,* as apparently one's preferred alcoholic beverage is an ideological hill worth dying on. As a result, people share the polarized articles to justify their habits, to moralize others' choices, or to plant their flag in a culture war about what it means to be healthy. That's because alcohol isn't just molecular, it's *emotional*. It's also social and psychological. Your experience with it depends not only on your genetics or metabolism, but also on who you're with, how you feel, what you've eaten, how well you slept, and how you're managing the rest of your stress load.

As I've specifically and repeatedly written throughout the first six chapters of this book, no form of hormesis is universally safe or beneficial. Individual variability in tolerance, and even intra-variability for each individual based on environmental factors on any given day, can cause massive variance. Regarding the hormetic stressors discussed in this book, at least, these statements are most true for alcohol.

There is a very fine line between beneficial and harmful when it comes to alcohol, and we do not yet know where that line definitively lies, nor do we have the ability to properly predict or estimate said line for any individual on any given day. We may be able to determine average consumptions of specific mg or g/kg levels of ethanol consumption, however, individual benefits and harms will be guaranteed to vary, and

Picture 17. Drunk after a Metallica concert (I'm not the one who's passed out in this photo, by the way. You'd think people would notice that a bald, Mexican man isn't me, but nothing surprises me!)

For a little while, it really does feel like winning.

not by a small factor. Additionally, the timing of consumption relative to sleep and circadian rhythm is likely to play a substantial role. As I have stated in previous chapters, intra-individual tolerance and effects could drastically shift day to day, situation, and timing dependent. For a stressor like ethanol with a narrow potential therapeutic window, this shifting target is of greater concern.

Further negating the benefit, the physiological stress of the hangover far outweighs the momentary delight of intoxication, at least from my current perspective. As I am sure many reading can relate to, this calculus was much different in my teens and twenties, and only began to become a very real consideration as I approached my thirties. At forty, there are few occasions where my desire to be intoxicated outweighs the knowledge of the impending consequences.

Since starting my neuropeptides, particularly selank, semax, and dihexa, while also incorporating a small dose of tesofensine, which is a triple agonist for dopamine, serotonin, and norepinephrine, I have had zero issues with focus, or creative thinking. As such, I have had no desire to consume alcohol. Due to these pleasant alterations in my regimen, I have happily quit drinking to intoxication, and am able to count the total alcoholic beverages I have consumed in the last 8 months on 2 hands, with ample fingers to spare: 4. That said, I can be coerced into a glass of wine, but rarely two, through peer pressure during special occasions/celebrations.

Blue zone populations, which have been studied extensively for their unusual longevity, are known to consume moderate amounts of daily alcohol, typically wine (Benedetto & Carboni, 2023). This observational data in part led to journalists confidently explaining that the "why" in red wine's longevity link had been found, with the attribution going to resveratrol. I will dive into that topic in this chapter, but as a preface, there is much more to the story than just one molecule found only in one type of alcohol.

Despite this chapter being likely to create controversy, I'm certainly not the first to suggest that alcohol is a form of hormesis from which we can determine therapeutic dosages with optimal levels. There is no shortage of crude epidemiological studies suggesting a recommended consumption of between one and three drinks a day, claiming alcohol may lead to lowered risks of dementia (Xu et al., 2017), cardiovascular disease (Chaudhry et al., 2024), osteoporosis (Godos et al., 2022), and mortality (Mostofsky et al., 2016; de Labry et al., 1992). Other studies report that with even a slightly higher daily intake, the benefits are not only negated, but risk factors sharply increase over abstinence for all of the aforementioned disease states (Hayes, 2007; Pricket et al., 2004; Calabrese & Baldwin, 2003; Pohorecky, 1977).

The media understands that consumers respond best to oversimplified messaging, which often means the message isn't just simplified, it's incorrect. Considering different studies, even different reviews, can report wildly different conclusions, media simplification not only removes the nuance but creates complete contradictions in the instructions that the general population is given. Further compounding the confusion is that the general population is largely unaware of the concept of hormesis.

Messaging for other hormetic stressors, like exercise, rarely discusses the inherent dangers of overtraining. Conversely, with alcohol, the messaging arrives erratic in tone and structure, confusing readers who are not well-versed in the science. One headline warns of drastic shortening of lifespan and the risks of alcohol, while the next talks of the potent longevity benefits. Alcohol causes cancer in the headlines one day, and 2 drinks a day reduces cardiovascular disease risks the next. The amusing part of all of this is that there are review papers in which all of these conclusions could be drawn, but the media rarely offers the entire picture.

As time has gone on, the rate of stories suggesting alcohol has beneficial health effects has declined. The prevailing message as of late has been that "no amount of alcohol is safe," even being echoed by the World Health Organization (WHO), as reported in the *Lancet*. Interestingly, the WHO alleges that the burden of alcohol consumption falls on vulnerable populations, including racial and ethnic minorities (The Lancet Rheumatology, 2023). There are certainly individuals who shouldn't drink at all (but this is not, nor is anything else in this book, medical advice). To generalize the risk of alcohol consumption to average individuals based on the allegedly vulnerable, whatever race they may be, is patronizing and belittling, and it is indicative of a state (or international agency) acting in blatant disregard for what is true, infantilizing the population through curated intellectual tyranny. "Trusted" institutions should only be trusted if their mission is to the truth, embarking on a quest to illuminate the populace with knowledge. When orchestrating an ideological plan to obfuscate the truth, subverting counter-messaging, these institutions should be condemned, not revered.

There are indeed those who believe, contrary to the *Lancet* reporting, that the quality of life gained from a drink outweighs its potential harms (Harvard T.H. Chan School of Public Health, 2022; MacMillan, 2017). Despite my recent decision to largely abstain, I would never venture to condemn another for this choice, provided they are not harming others, and I would certainly not conspire to deceive and manipulate perception.

Institutions have repeatedly opted to behave this way, despite the clear intellectual and ethical violations. As such, every year the suggestion that alcohol can be *good for you* is becoming an intellectual hurdle too large for many to grasp. This is often exacerbated by negative experiences in times of overconsumption. As I have repeated in each chapter, all forms of hormesis, including ethanol, can be either a poison or a therapy, or more simply, the dose makes the poison.

Hormesis, Hazard, or Both? Ethanol: The Dose, and Timing, Make the Poison

Ethanol has been noted to affect a plethora of animal and human behaviors in a two-phase, or biphasic, dose-dependent manner, with low doses being stimulatory and higher doses being inhibitory (Kurta & Palestis, 2010; Pohorecky, 1977). This

has been observed in studies on anxiety and depression, with low levels improving conditions and high intake interfering with proper mood function (Gémes et al., 2019; Williams, 1966). This can be quite problematic, as those suffering from depressive disorders are more likely to drink to excess, exacerbating their condition (McHugh & Weiss, 2019; Cooney et al., 1997). Since alcohol also tends to alter "the reward center" of our brain, increasing our proclivity to gamble and take chances (Brevers et al., 2014; Hurst et al., 1969), the cumulative results can be devastating for those who suffer from alcoholism.

Alcohol tends to either promote mildly beneficial physiological outcomes, as compared to abstinence, or aggressively drive towards a "disease" state when overdone, similar to other forms of hormesis—but with substantially less forgiveness in overdoing the stress, and higher risk when administered in excess. Similarly to exercise, where moderate training in men who had previously been sedentary dramatically improves testosterone (Hayes et al., 2017; Hawkins et al., 2008) and chronic overtraining significantly decreases testosterone (Hackney & Aggon, 2018), mild to moderate alcohol intake has shown to increase testosterone in both men (Sarkola & Eriksson, 2003) and male rats (Calabrese & Baldwin, 2003; Cicero & Badger, 1977), while chronic consumption in excess of the low levels inducing beneficial adaptive stress, or at inopportune timing, decreases testosterone (Smith et al., 2023), in addition to many other negative impacts. Yet, in controlled settings, moderate drinking has shown some surprising physiological effects. Studies show boosts in testosterone (Sierksma et al., 2004), improvements in cardiovascular function (Chaudhry et al., 2024), and even cellular repair (Ellison, 2011).

That's the lesson here: alcohol can be used, but it's a precision tool, not a blunt instrument. It doesn't build you the way exercise or fasting does. It builds slowly, if at all, and it breaks you quickly. When it goes wrong, it goes very wrong, and that's the tradeoff. So the old cliché that "alcohol kills brain cells"? Not quite. What alcohol kills, when overused, is the machinery of renewal. The scaffolding of plasticity—the thing that keeps us adaptable and sharp. It's not just the brain, either.

Alcohol has been researched heavily in both cardiovascular disease and its prevention. Some of the benefits of moderate alcohol intake could very well be from the release of heat shock proteins (Sato, Fraga, & Das, 2004), as well as endothelial nitric oxide synthase (eNOS) production (Abou-Agag et al., 2005). While clinical data studying alcohol for cardiovascular function is non-existent (due to ethical concerns), observational studies, as well as research looking into modes of action, and rodent studies, are all important in ascertaining potential benefits and harms (Lucas et al., 2005).

In covering other forms of hormesis, we know that autophagy is a critically important physiological process that needs to be regulated properly, neither chronically activated nor inhibited. Autophagy can be activated by other forms of

Picture 18. Enjoying a full-bodied red with my fiancée

Not every glass is a mistake. But every glass carries a question.

hormesis such as caloric restriction, fasting, molecular hydrogen, and exercise. Alcohol, also, has data to support autophagic activation, at least, when it comes to mice. The role of FOXO3a plays a critical role in expressing this outcome, with overexpression of a dominant form inhibiting autophagy. Additionally,

pharmacologically-activated intermittent, or acute, stress promotes autophagy in the liver and helps to attenuate alcohol-caused liver damage, while inhibition exacerbates it (Ni et al., 2013). Finally, mTOR signalling may also play a role in alcohol and autophagy, as well as in overall mortality and life extension, and it has been noted that mild to moderate alcohol consumption inhibits mTOR (Chang et al., 2017; Elmadhun et al., 2014). A robust and informative review of alcohol and aging, largely in line with my perspective on the subject, was authored by Stuart Adamson, Leah Brace, and Brian Kennedy (2017), and published in *Translational Medicine of Aging*.

The line between "mildly helpful" and "clearly harmful" is thinner with alcohol than with almost any other stressor. We still don't know exactly where that line lies *because it moves*. It depends on who you are, how you metabolize ethanol, what else is in your system, how well you've been sleeping, and, critically, what kind of stress load you're carrying, both mental *and* physical. In short, the line isn't just moving from person to person, it's moving for each person, each day.

A hot day, an exhaustive workout, or even just emotional strain can all degrade your ability to process alcohol. Your tolerance drops—not just in terms of how drunk you get, but in how disruptive that drink becomes to your recovery, mood, and sleep (Hensel et al., 2021; Lakićević, 2019; Söderpalm & de Wit, 2002). Context matters, and your state matters. Drinking in good company, in a safe environment, after a restful week can land very differently than drinking alone after a stressful day under the sun with no food in your system. Alcohol is a social tool, a cultural signal, and a neural modulator; in other words, it's a metabolic wildcard (Kirkpatrick & de Wit, 2013). A drink that relaxes you after a long day can also impair your recovery, degrade your sleep, suppress early night REM, spike your cortisol the next morning, and drop your HRV for 48 hours. All without you noticing, until the deficits compound (de Zambotti et al., 2021).

Picture 19. Tarnava beer!

Even when you find your name on the glass, it doesn't mean you have to keep drinking.

From a cautious health standpoint, the best choice is simple: abstain—or stay *well* under your tolerance threshold, not because alcohol is evil, but because the margin for benefit is razor-thin. I've made my peace with that. I used to love a full-bodied red, and I'll be honest, I used to write better after a glass or two. Thoughts moved faster and associations came easier, but that's the seduction: you get a taste of insight, and then you keep going, hoping the third glass brings more of the magic the first one offered. It doesn't. It brings sloppiness, and then it brings hangovers, and then it brings a morning of physiological regret—and possibly an e-mail or two that never should've been sent.

Ethanol: Poison or Therapy?

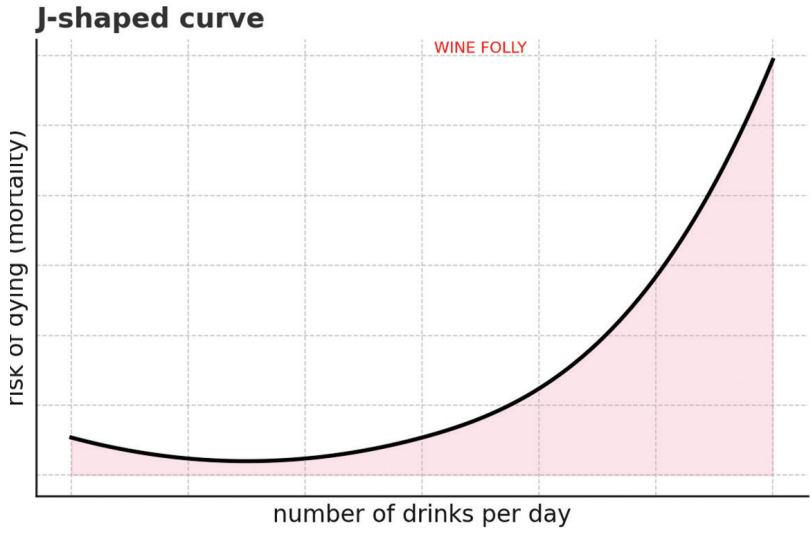
Alcohol: The Hormesis Paradox

Ethanol has been shown to promote neurogenesis in the hippocampus of rodents consuming moderate amounts of alcohol, without triggering apoptosis (Aberg et al., 2009), neurogenesis being the process in which new neurons are created in our brain. As most people "know," overconsumption of alcohol kills brain cells. In reality, overconsumption of ethanol kills the neuronal stem cells needed for neurogenesis (McGrath et al., 2017). Interestingly, the same study found that sex is a critical factor: female neural stem cells appear more vulnerable to alcohol's damaging effects than those of males, suggesting biological sex may modulate susceptibility to alcohol-induced cognitive decline. Ethanol also damages dendrites, the ends of our neurons needed for "proper communication" through our brain, specifically in regions that moderate "reward" (Zhou et al., 2007a). On the contrary, a large Australian cohort study of 7,485 participants found that light or moderate drinkers had superior cognitive function to those abstaining from alcohol altogether or those drinking in excess of hazardous levels (Rodgers et al., 2005).

Proper function of the glymphatic system, our internal system that deals with junk removal in our central nervous system (CNS), is critical in both the prevention and development of various neurological and CNS diseases (Zhou et al., 2007a). In a recent study, it was demonstrated that low-to-moderate amounts of alcohol activated the murine glymphatic system, which would hypothetically create a protective effect against many diseases, and significantly compress the glymphatic system with heavy consumption (Lundgaard et al., 2018). The differences were quite significant and the reduction in function at higher doses was much greater than the activation seen at low doses, indicating a dose-dependent bidirectional effect of alcohol on the glymphatic system. When those two glasses of wine turn to six, the therapy turns to poison. When a dose goes from therapeutic to deleterious in a very small change, it is known as having a steep "J" or "reverse J" curve, due to the graphical resemblance of a J. Alcohol's J curve looks something like this in terms of mortality:

In other words, the J is "reversed" for the glymphatic system, with small amounts "activating" and larger amounts "deactivating." While alcohol in moderate

Figure 2. Alcohol's j-shaped curve



Note. *From these authors.*

dosages may activate the murine glymphatic system, for humans, this may end up being more complicated. Lack of sleep, or specifically impaired deep non-REM sleep, deactivates the glymphatic system (Hablitz et al., 2019). Since even low use of ethanol before bed serves to impact REM sleep, therapeutic drinking should be avoided right before bed. It has already been established that alcohol is biphasic and perhaps the benefits to the glymphatic system can be achieved, while mitigating disruptions to sleep, if a glass or two is enjoyed in the early evening. Suggesting "day drinking" may be a tad risqué, but I suspect much of a gap is not necessary so long as no noticeable intoxicating effects are observed (and if you're intoxicated, you likely swept down the J curve into damage and harm).

Even small amounts of ethanol consumed before bed affect sleep architecture in a biphasic manner. During the early sleep cycles, ethanol suppresses REM sleep and increases slow-wave (deep) non-REM sleep. However, as ethanol is metabolized and cleared from the body, typically within 4 to 5 hours, this leads to sleep disruption characterized by frequent awakenings, which leads to an increase in REM sleep duration, along with a significant impairment in the continuity and quality of slow-wave sleep during the second half of the night (Roehrs & Roth, 2001). This leads to poor sleep quality and efficiency, impacting various repair and maintenance mechanisms, such as the glymphatic system. While alcohol in moderate amounts may improve sleep duration, it decreases REM sleep and total sleep quality (Roehrs & Roth, 2001). Even the use of alcohol for insomniacs has conflicting data, with some parameters improving and others, such as REM, decreasing (Roehrs et al., 1999).

Picture 20. Mariinsky Theater in Saint Petersburg, Russia (seeing *The Love for Three Oranges*; a ridiculous opera)

Turns out walking several miles a day earns you a front-row seat to not getting drunk.

When I hit my mid-30s, I began to find that if I drink my wine late at night after dinner and right before bed, even two to three glasses leave me hungover. If I drink my wine before that, such as during dinner in the early evening, or as a pre-dinner indulgence, and then go for a walk or am otherwise active in the evening, then I

wake up feeling fine. Each passing year is intensifying this trend, and, at 40, a single glass of wine after dinner, within 2-3 hours of sleeping, leads to an alarming spike in my resting heart rate, a startling drop in my heart rate variability, and leads to poor sleep quality—resulting in a hangover from a single drink which did not lead to even mild intoxication. The enjoyment of alcohol comes with a cost, and these days, if consumed in the evening, I pay the cost, without any enjoyment.

In 2024, I started to pre-plan my drinking escapes, ensuring that the event was "worth" the next day's trade-off: concerts, attending UFC pay-per-views live, date nights, and events with business colleagues and friends. Not as often as when I was younger, even a year or two before, but still more often than is ideal for health (maybe once or twice a month). This was all thrown to the wind when I travelled to Russia in September of 2024. Russia is a unique place for many reasons, but certainly that land's relationship to alcohol is one of them. It's an intense relationship, but it's one that reveals some hormetic principles about ethanol, that the dose makes the poison. While in Russia, my contacts ensured that I was intoxicated daily; although, the habit was substantially different than what I had adopted in adulthood, though, not unlike how I grew up witnessing drinking through my dad, who has Ukrainian heritage. As a quick digression, my dad once fermented his own wine, keeping the barrels at his shop, and opting to start every morning with a glass, or two, or three (and so on…) of red in his coffee cup, rather than coffee. Or, he would be drunk in the morning on the golf course, and sober up later working around the house and preparing dinner. It was familiar to me, but not my norm.

In Russia, we fully embraced this lifestyle: we were drinking during brunch and lunch. We consumed several bottles of wine, nastoyka (vodka infused with flavor, such as fruit), all while feasting to the point of gluttony. I could estimate consumption at ~10+ drinks per day, over a ~2 hour period late morning and early afternoon. I'd be intoxicated, with a warm, slightly drunk feeling, mitigated only by the heavy eating during the drinking. Then, we would go and walk all day. Through museums, sightseeing, and other foot traffic-heavy activities. We'd go to the sauna and cold plunge, too, and each day would feel like several. I was tracking 25-30,000 steps per day, every day.

We didn't drink after this initial burst, and our dinners were much lighter than our breakfasts and lunches. After dinner, we would walk the city more, lasting late into the night. By the time I went to sleep, I had been sober for 8-9 hours. I write all of this because what I experienced, regarding the "cost," was remarkable. During my time in Russia, I had the best sleep and recovery data I have experienced in years. My resting heart rate was consistently in the low to mid 40s, my heart rate variability almost doubled, and my sleep consolidation was much more efficient than is typical, and my sleep consolidation is already top tier. All of my measured markers were positively impacted, with a lowered body temperature and slower respiratory rate. Importantly, I woke up every day feeling great, with no hangover.

Picture 21. A beautiful building, somewhere in Russia

If you're gonna burn through a post-brunch buzz, make it baroque.

This was repeated every day for 7 days straight, and my numbers continued to improve as the week went on. Whatever lingering effects from jetlag that were still with me when I got to Moscow, were long gone by the time I left Saint Petersburg. Finally, I felt validated for my musings about the potential benefits of day drinking,

albeit, with my lifestyle and workload making this a regular occurrence is all but impossible. Of course, this is all anecdotal and I am not pretending this is definitive evidence, but, considering there is reasonable rationale for why this could be true, and that I argued this ***before*** I attempted it myself, I believe the future of ethanol research should explore timing of consumption in relation to sleep as a more important consideration than dosing.

Lack of Sleep Deactivates the Glymphatic System

The Timing Trap

While alcohol in moderate dosages may activate the murine glymphatic system, for humans, this may be more complicated. As already mentioned, lack of sleep, or specifically impaired deep non-REM sleep, deactivates the glymphatic system. In short, alcohol can initially boost deep sleep and glymphatic activity, but as it metabolizes it disrupts sleep cycles, reducing slow-wave sleep continuity, fragmenting rest, and ultimately impairing the glymphatic system and overall sleep quality. The data is conflicting, even for groups like insomniacs, as I shared in the last section. Of course, even the most complex problems are knowable with enough information and context, and I am not the type to abandon a curiosity just because it is complex.

This discussion leads me down a rabbit hole of thought that appears to be poorly studied. When I used to drink, if I was able to sleep in, I'd notice that my total time asleep would remain constant or even increase, and my total time in REM and deep sleep would remain constant, as well. In contrast, my total time in bed would dramatically increase, sometimes by 2+ hours, indicating the increased fragmentation and repeated awakenings due to alcohol use. I also noticed that despite this, I definitely felt the effects: slow thinking, headache, and a fatigued body. This led me to the hypothesis that fragmenting deep or REM sleep, even if total duration in the day remains consistent, could impair repair, recovery, and function. At least one team has hypothesized the same, finding that fragmenting slow wave sleep (deep) and REM, with total time in each of these stages remaining constant, impairs long-term memory consolidation in rats (Lee et al., 2016).

This perhaps explains why I seem to need much less sleep than is typical. In normal conditions, I am able to thrive on 5.5-6 hours of total sleep time. That said, within this short total sleep, over 50% is usually allocated to deep and REM stages, with a roughly equal breakdown in each. When I've compared my sleep data to others, their REM and deep tracking is similar, even lower, despite time asleep being substantially longer. As I write this, deep in sympathetic stress due to a colicky newborn, my total sleep has been substantially longer: 7-8+ hours each day, with much less total REM and deep sleep, often under 1 hour of each, and usually under 40 minutes of deep. Despite being in bed much longer, I am feeling fatigued, my thinking is impaired, and my body is feeling wrecked from workouts. Alcohol exerts this effect, by its nature, what is occurring to me now. It fragments your sleep, interrupting deep and

REM stages, leading to shorter durations in each sleep stage, even if total time within them remains constant. For this reason, anyone venturing to partake in what they believe to be therapeutic drinking should avoid consumption in the hours leading up to sleep.

It has already been established that alcohol is biphasic, and perhaps the waste-clearing benefits to the glymphatic system can be achieved, while mitigating disruptions to sleep, if a glass or two is enjoyed in the early evening, or earlier in the day. A fun way to think about it—although absolutely not advice, and suggesting this is a rule of thumb negates everything I have suggested previously, would be "3 drinks at breakfast," or "two at lunch," or "one at happy hour," and none after dinner. Not all in the same day, of course, as that would dramatically change the calculus. The point in that thought experiment is simply to suggest that the nearer you get to your sleep period, the less you can safely drink without affecting sleep. Also, the number of drinks should be kept below the amount that gets you intoxicated. After all, as I stated previously, if you're intoxicated, you likely swept down the J curve into damage and harm, anyways.

Resveratrol

The Resveratrol Excuse

Resveratrol is one of the most hyped anti-aging molecules of the last two decades. If you've spent any time in longevity circles, you already know the claims: resveratrol is supposed to improve metabolic function, reduce cancer risk, protect the brain, and repair the cardiovascular system. It's been pitched as a kind of molecular multitool, capable of slowing aging by hitting some of the same cellular pathways triggered by fasting or caloric restriction. On paper, it sounds like the perfect shortcut.

There's even data showing resveratrol can reduce advanced glycation end products (AGEs) which are one of the major biochemical contributors to aging and tissue stiffness (Maleki et al., 2020; Hajizadeh-Sharafabad et al., 2019). In one study, it inhibited AGE-induced cell proliferation and collagen synthesis, both markers of aging and fibrosis (Mizutani, Ikeda, & Yamori, 2000). I've also seen promising results in animal models of diabetes. In type II diabetic rats, resveratrol seems to help modulate blood glucose and insulin sensitivity (Rehman et al., 2018; Soufi et al., 2012). Mechanistically, it has merit.

It was a great story that many jumped on board with, and while it was in vogue to declare that the purported benefits of red wine were attributed to resveratrol, at least for a time, this claim cannot stand up to even the slightest bit of scrutiny. For instance, the actual dosage obtained through red wine is quite insignificant, at a fraction of a milligram per glass (Lachenmeier et al., 2014). In other words, before even approaching a dosage of resveratrol, which may offer a slight physiological effect, you will die of ethanol-induced toxicity (alcohol poisoning).

I'll offer one hail mary caveat; it is possibly relevant to note that the combination of resveratrol and ethanol may work synergistically in inducing autophagy as resveratrol decreases FOXO3a acetylation, while alcohol inhibits mTOR activity and increases NADH/NAD^+ ratios (Luo et al., 2017; Ni et al., 2013). This is a rather weak piece of conjecture which would need extensive substantiation, however, in the past I leaned on this conjecture, in part, as a form of confirmation bias, or self-serving justification, for my preferred poison.

Conclusion: The Line Is Tight

I submitted a commentary article on a review paper that recommended alcohol guidelines based on the population-level data and statistics, which is standard. My position was based on the irrefutable facts I have laid out throughout this chapter, backed by substantial citations. I argued that the assessments were correct, but the conclusions were illogical: recommending complete abstinence is likely to fail, and eventually, the truth will emerge, with the populace further losing faith in the scientific establishment, and governmental institutions.

I put forth an argument that the population should be cautioned that precise guidance is impossible due to inter, and intra-person variability, timing of intake, and current stressload. I went on to state that the future of guidelines lies in individualized monitoring and response, and that while not yet sufficient, wearable technologies offered the potential for accurate, real-time guidance in the future, collecting substantial data to craft recommendations for each individual at each specific point in time.

The first reviewer provided positive feedback, stating he largely agreed with what I had written. He suggested additions to further strengthen my paper, the challenge being I was already at the word limit. He recommended publication. The second reviewer wrote a visceral response, stating I was advocating for wearable technology without declaring a conflict of interest. The journal employed single blind peer review, meaning the reviewer could see my name and affiliation. The reviewer claimed that in his search he discovered I was financially tied to wearable technology. That was a lie, which I emphatically told the editor, asking for proof of this accusation.

The editor simply responded: "regardless we have made the editorial decision not to publish," stating that due to one reviewer being "extremely critical of your paper," with the other review being "positive, but wanting minor revisions," that the strength of the positive review didn't match the negative in force. I responded that the reviewer who was critical was basing his entire argument on a lie, to which the editor responded that he did not have the capacity to verify the accusation, and besides, I am not even part of academia.

Alcohol used to command a certain level of interest for me, hence my writing the commentary on the article, namely because I drank, and worked hard to justify my practices intellectually. I used alcohol deliberately for years: for stress modulation, to explore its interaction with supplements and sleep, and to calm my mind in certain social situations, such as in loud places full of cacophony. I tracked the effects, not with full blood panels per drink, but through wearables, subjective logs, and routine testing over time. I monitored my liver enzymes and logged my sleep architecture. Importantly, I tracked my performance, both in the gym and in terms of my intellectual output.

I didn't do this because I believed alcohol was harmless, but because I believed it could be used responsibly—with data, discipline, and clear boundaries. Over time, I stopped, not because the method was flawed, at least not for me, but because I kept breaking my own rules. I overindulged, ignoring the limits I set for myself, so I quit—not abruptly, but slowly, step by step.

My monitoring is a step below hard evidence, but several steps above random guessing. I was observing unblinded N1 trials which I wanted to succeed—don't get me wrong—but my results were repeated, reliable, and substantial. Even if it was all a placebo, because I wanted it to be true, it was working for me. The research, as I have stated, offers glimpses into the plausibility. Alcohol probably does offer some biological benefits if it's used with precision. That means low dose, right timing, a strong metabolism, and a clean recovery. It can function like any other stressor: a spark that triggers beneficial adaptation.

Here's the truth most people don't want to admit: for the majority of us, trying to walk that line ends in a stumble, not because we're weak, but because the margin for error is razor-thin, and the incentives to overdo it are everywhere. They're cultural, emotional, social, and psychological.

In short, I used to love a glass or two of wine. Reading, writing, listening to music, deep dinner conversation—wine made all of it feel more fluid, more textured, and somehow, more real. It enhanced the moment, and sadly, many of my best memories are of times my intoxication level was "just right," and the company I was sharing was in that moment with me. If I didn't have a better toolset, namely, if I hadn't found a protocol that made me sharper, clearer, more focused, healthier, and without the potential to fall flat on my face, I'd probably still be trying to thread that needle. I'm sure some nights in the future will lead to that attempt, and I'm also sure they'll end in disaster. The less I drink, the less equipped I am to even see where the eye of the needle is. So, perhaps the solution for me is to acknowledge that when I decide to have a drink, since I can't even see the needle, I won't bother trying to thread it.

Mentally, I have accepted this truth, and I am more than fine with it. I don't need alcohol for most of the reasons I previously believed I did, at least, I don't anymore. Since starting my neuropeptide and tesofensine stack, my mind works better without

it, better than it has in years, maybe ever. My writing flows, my focus holds, and I don't reach for the ritual anymore because the function it served has been replaced and upgraded.

For me, my new routine makes sense. For many others, injecting peptides and experimental drugs obtained off the black market may seem insane, and even riskier than indulging in a few adult beverages. For many, a drink or two in the evening is part of the rhythm of life. If it's working—truly working—without cost, that's your call to make. If the reward is stronger for you than the cost, then it isn't my place to say otherwise. The conversation about alcohol isn't about abstinence or indulgence, it's about honesty. Whether you drink or not, the real question is:

Do you know your threshold?

Do you understand the risk curve?

Can you tell when the benefit has flipped into harm?

The target is small, and if you're not paying attention, the lights go out, and you're aiming blind. This chapter wasn't about guilt, it was about clarity. If you drink, know what it's doing.

If you don't, don't pass moral judgment on those who do, focus on yourself, and your own progress.

That's how we train every stressor in this book: not with fear but with sovereignty.

CHAPTER 8:
Revealing Hydrogen

> *"To deny the feasibility of something that is alleged to have been done or the possibility of an event that is supposed to have been observed, merely because we cannot understand in terms of our hitherto framework how it could have been done or could have happened, may often result in explaining away quite genuine practices or experiences."*
>
> — MICHAEL POLANYI

Hydrogen saved my life, but not in the way you may expect. During, and after my health crisis, as my body broke down, my mind spiralled into a dark place. This mental collapse didn't take much, as exercise was one of my only self care routines at the time, and nothing I was doing in my life gave me any sense of purpose.

I've never had a hard time learning how to make money, or succeeding in projects I set out to accomplish in the short term, but what I have had a hard time with is sticking with them. No matter the field, the hobby, or the intellectual pursuit, it never mattered, the result was always the same: I'd dive in, excited, and quickly become obsessed. I'd devote my entire being, my time and mental energy, to learning about my new pursuit—all the while thinking "this is it, I've finally found it." This obsession would continue for a few weeks, maybe a few months, and then as suddenly as the obsession emerged, it would vanish, being replaced by boredom and disillusionment.

I believed nothing could capture my attention or inspire me to become great. I thought something was broken inside of me, and that I was destined to be a failure who managed to collect countless former hobbies and interests, all of which I maintained a surface level understanding of (or intermediate skill set for) and none of which I was truly great at. I was terrified that I was destined to amass a mountain of mediocrity. This fear drove me to try to stick with pursuits long after my mind had checked out, whether they were intellectual, physical, or designed to create wealth—which never interested me, until my perspective on its purpose changed. I learned, far too slowly, that I couldn't force my mind to stay somewhere it didn't want to be, no matter what the reward was.

Then, my health crisis occurred, and everything changed. Not only because I was desperate to fix my body, but because for the first time I had found something which could hold my attention. Molecular hydrogen wasn't just another trend or industry, it was something new, and it was something different. The science was

embryonic, with real questions and real unknowns. The industry wasn't locked down by old structures, it needed to be built from the ground up. With hydrogen, for the first time, after a few months I wasn't able to see the light at the end of the tunnel, meaning, I didn't have a clear picture of the exact commitment needed to reach the top—because there was no top, not yet. Somehow, the more I learned, the more there was to learn.

My own health, my selfish interest in fixing what had broken with my physiology, drove me to start down the road with molecular hydrogen. It propelled me well past the usual few months, maybe even for the first year. After that, it isn't what kept me going. As I learned more about H_2, and about what ailed me, I accepted that while hydrogen was likely to slow down my decline, it wouldn't rebuild what had already been lost. So I kept going, but not out of routine, not out of delusion, but because I had found a new reason to forge on.

I continued to push, diving deeper and deeper. What captivated me wasn't the hype, it wasn't the marketing, it wasn't my own health goals, and it wasn't the community: it was the work, the chance to build something foundational, to create new definitions where none existed, and to push through regulatory walls nobody had touched yet. The industry was nascent, and the science was green, but my instincts told me it was real, not a fad, but the future. I had promised myself years before I would never let an opportunity like this slip through my hands again, so I relentlessly pursued like an addict stopping at nothing to get their next hit. I use that analogy "like my addiction depended on it" because, in many cases, the brain's motivational circuits become so distorted by addiction that pursuing the substance feels more urgent than preserving one's own long-term health. It's not that people don't value their lives; rather, the addiction hijacks the sense of urgency and reward. In that moment, the next fix can seem more pressing than survival itself, not because survival isn't important, but because the brain has been rewired to treat the substance as essential to it.

The hydrogen tablets, the research, the whole field, became more than just survival: it was the first real purpose that stuck, it was my addiction, and as such, it became essential to my survival, to my very being. Even now, I know that nothing holds forever. Interests shift and passions evolve, but the foundation I helped lay is real. It will outlast my own attention span, and that's enough. That was the beginning.

This chapter isn't just mine. Input from Tyler LeBaron, Ph.D. a biochemist, physiologist, and exercise scientist, has contributed greatly both through manual additions he made to this chapter, and also the countless conversations we have shared over the years which have given me deeper understanding and perspective. Tyler is an integral part of this story, and without him, I doubt I would have continued on in this field. Without him, our industry may have devolved into the same fate seen in Japan and South Korea, which exist as industries rampant with

scams and pseudoscience, leading to reduced funding on research due to academia's embarrassment over the industry. Without him, molecular hydrogen would likely still be an esoteric industry destroyed by the weight of its own greed. Tyler has been a shining light guiding industry in North America, and anyone involved or interested in molecular hydrogen as a therapeutic owes him a debt of gratitude.

Early on in my journey with hydrogen, perhaps a few years in, I had a conversation with Tyler which I wish to relay. Though he had been a leading hydrogen researcher at the time, and he remains an even more prolific one today, he had raised an eyebrow at the direction the field was headed. *"I'm not so sure about this whole hydrogen nonsense,"* Tyler joked. He didn't doubt the biological plausibility, and he didn't doubt the evolutionary role of hydrogen. What he doubted was the noise; the bad studies, the overblown claims, and the shaky methodologies. Tyler is a scientist's scientist: rigorous, conservative, and cautious with his endorsements.

Over the next six years following this conversation, Tyler didn't become less skeptical of bad science. He became more confident in what the data suggested, not because the studies got cleaner, and not because the methods improved, but rather, despite the flaws, the signal stayed consistent: across models and across conditions, physiological effects kept showing up again and again. He still wouldn't say hydrogen "works." Not in the vague, overreaching way people tend to mean it, but he would say this: hydrogen has real, measurable biological effects on stress pathways, inflammation, redox regulation, and more. Those effects? They matter, not as a cure-all but as modulation.

What Michael Polanyi called personal knowledge seems appropriate here (Polanyi, 1958/2015). Polanyi was describing the kind of truth that isn't captured by formulas or fixed frameworks, but rather that emerges from lived experience, from commitment, and from staying with the thing long enough for it to show itself. For Polanyi, discovery wasn't about following a blueprint but about recognizing a pattern before it's fully formed: in other words, a leap of integration, not deduction. Tyler and I didn't "discover" hydrogen the way textbooks describe. It revealed itself, through collapse, through uncertainty, and through years of tracking, refining, and staying just this side of belief. As Polanyi warned, too rigid of a scientific framework can make us explain away genuine phenomena, simply because they don't yet fit what we're prepared to recognize.

This chapter is about that edge. It's about hydrogen, but not as a panacea, rather as a signal. A molecule small enough to pass through the mitochondrial membrane, and big enough to shift the trajectory of a broken life. Let's get into it.

Introduction to Molecular Hydrogen Research

Think of the birth of the universe: it began with a spark. The modern resurgence of research on molecular hydrogen, in regards to its physiological effects, began in

the same way: thrust forward into perpetual acceleration in countless directions. The ignition of molecular hydrogen into conversations across a seemingly endless amount of disease models started formally in 2007 with a pivotal study in Nature Medicine that demonstrated that molecular hydrogen (H_2) could selectively reduce harmful free radicals within the body (Ohsawa et al., 2007). Since then, scientific interest, research, and populational awareness has only grown, in fact expanding with near cosmic force, accelerating year on year. As of 2025, there are over 3,000 peer-reviewed papers and more than 200 human clinical trials exploring hydrogen's effects across 200+ disease models. These aren't isolated findings but discoveries that span every major organ system.

Unlike the expansion of the universe, however, molecular hydrogen's expansion has had to overcome substantial friction and resistance. At first, no one could quite pin down how it worked. The mechanisms were elusive, the pharmacodynamics slippery, and skepticism from academics and consumers alike was astronomically high. Typically, therapies which researchers and proponents purport to be a panacea are almost always snake oil. Of course, hydrogen isn't a panacea, and we now understand much of the mechanisms behind how, and why, it exerts a physiological effect. Over time, the picture sharpened, and we now know hydrogen doesn't behave like a conventional antioxidant, or in a manner which fits the definition of a drug. Skepticism remains, but it is diminishing with each passing day.

Hydrogen is pleiotropic, meaning it affects seemingly unrelated pathways, systems and genes in the body. For instance, it modulates stress pathways, balances redox states, and influences cell signaling. Because these are all universal processes, hydrogen's reach is broad, at times seemingly limitless. I want to be clear that limitations do exist, no matter what your favorite influencer has told you. That said, whether you're an elite athlete, a chronically inflamed desk worker, or someone navigating aging, the data suggests H_2 has something to offer.

Evolutionary Adaptation to Hydrogen

> *If God did create the world by a word, the word would have been hydrogen*
>
> —HARLOW SHAPLEY, ASTRONOMER

Hydrogen gas (H_2) is not a molecule introduced by modern science, it is a molecular signal interwoven into the evolutionary fabric of life itself. It is ancient, far more ancient than us, than the beginning of life, than this planet, solar system or galaxy. Hydrogen is the beginning, and was the substrate that gave rise to every element on the periodic table. Hydrogen was not discovered in modern times, but rather, we became tacitly aware of its relevance only recently. It was always there, it is only our understanding of its role that has changed.

Just as hydrogen gave birth to all other elements, and as such, the universe as we know it, it may have also given rise to life as we know it. According to the hydrogen hypothesis, the mitochondria inside our cells, which are the engines of our energy, were born from a pact between two ancient microorganisms. A hydrogen-consuming archaeon made a deal with a hydrogen-producing bacterium: *energy in exchange for shelter*. It was a hydrogen economy at the molecular level, and it worked well enough to power the explosion of life that followed. That history never left us, it's still there, stitched into our biology. Hydrogen was never just a gas we drifted past in the atmosphere, it has always been part of us.

For most of human history, hydrogen's rhythm was built into the environment. It came and went with what we ate, how we lived, and what lived inside us. Heavily influenced by microbiota composition, dietary fiber intake and digestive activity, the spikes of hydrogen from microbial fermentation were erratic but expected, with production and exposure differing between individuals and across time, even within individuals of a specific time on a day to day basis. In ancestral environments, access to fermentable carbohydrates was inconsistent, yet generally much higher than modern intake, which would have led to said erratic, but repeated, hydrogen spikes from microbial-driven fermentation in the gut.

This variability may have played a formative role in shaping human biology, with life evolving to expect this erratic and fleeting rhythm of intermittent hydrogen exposure. Our cells learned to listen for this rhythm, learned to expect it. Now, in a world of sterile diets and broken microbiomes, that signal has gone quiet. Fiber intake has plummeted, and with it microbial diversity has crashed. Bacteria, like any living thing, die when access to fuel is cut off; our modern diet perpetrating a genocide of these pivotal hydrogen-producing species. Today, hydrogen exposure has become rare, and limited, where once it was a steady drumbeat underneath life.

Hydrogen's role as a cellular signal may reflect a form of anticipatory hormesis: a non-toxic stressor that primes adaptive responses not through damage, but through informational shifts in redox status, transcriptional signaling, and mitochondrial behavior. The rapid and widespread occurrence of modern deficiency has created an anomaly which we have lacked the knowledge and understanding to address. Before we knew molecular hydrogen played a critical role, the various signs of deficiency, namely dysregulated stress responses, were little more than random noise.

Defining molecular hydrogen's role in our physiology is challenging. It shares considerable overlap with various forms of physiological hormesis, however, many traditional hormetic stressors such as heat, cold, or radiation have tight and constantly variable therapeutic windows. This is exacerbated by the fact that missing these small, moving, targets can transform the desired therapy into a damaging attack on your resiliency. Hydrogen, however, displays a non-toxic dose-response. High concentrations of exogenous H_2 do not induce pathological damage; instead,

some benefits tend to scale non-linearly with dose, constrained only by delivery method,solubility and feasible rates of consumption.

This suggests that hydrogen's evolutionary role is not as a "mild poison" triggering compensatory repair, but as a signal of environmental and metabolic context, tuning cellular systems toward adaptive homeostasis. Molecular hydrogen is a molecular echo of our evolutionary past, and through exogenous supplementation, we may be restoring a lost rhythm of endogenous signaling once shaped by ancestral diet, microbial ecology, and even our prehistoric atmosphere, once abundant in hydrogen—so abundant that the oldest water supplies we have discovered on the planet still contain measurable levels of dissolved hydrogen gas (Gohd, 2018; Tian et al., 2005).

Reintroducing hydrogen's signal, especially in populations burdened by metabolic dysfunction, chronic inflammation, and sedentary behavior, may help realign fundamental processes such as mitochondrial regulation, redox balance, and inflammatory resolution toward an optimized physiological state. In this light, hydrogen is not a supplement, not a drug, not even a therapy in the traditional sense. It's a lost supervisor encoded in our genetic memory, and reintroducing it, through water, inhalation, or other novel delivery systems, may help restore a part of ourselves that modern life allowed to atrophy. In a population suffocating under inflammation, oxidative stress, and metabolic collapse, hydrogen is not a miracle, it's a reminder.

Modern Deficiency in Hydrogen

The tragedy concerning modern deficiency doesn't end with our physiology, it even affects our agency. Awareness of the problem, and motivation to change our lifestyle and diet, is potentially futile for combatting hydrogen deficiency. As research has begun to demonstrate, for some, particularly middle weight and older metabolically impaired individuals, the consumption of dietary fiber doesn't lead to hydrogen production, but rather, to the production of methane (Kumpitsch et al., 2021; Fernandes, Wolever, & Rao, 2000). Starved of their fuel source, hydrogen producing bacteria slowly die off, ultimately being replaced by methanogens, which are bacteria that consume hydrogen and produce methane.

This shift in microbial content is not innocuous. Breath methane, now detectable in increasingly large segments of the population, has been associated with constipation, IBS, obesity, insulin resistance, cardiovascular disease, and even all-cause mortality (Mathur et al., 2016; Mathur et al., 2013; Kunkel et al., 2011). In other words, as hydrogen fades from the internal ecosystem, methane rises, and its presence correlates with systemic dysfunction.

Unlike classical nutrients, where deficiency is measured by overt dysfunction or disease, hydrogen deficiency is more insidious, and much harder to detect: a signaling silence leading to a slow and steady decay, rather than an easily diagnosed acute

pathology. The subtlety of the harm its absence creates, paradoxically, could in part explain our modern health crisis. So many of our physiological systems are increasingly dysregulated in modern populations. Metabolic, neurological, and immune issues are increasing at alarming rates. While it is unlikely hydrogen deficiency can explain all of these epidemics, it is becoming more and more evident that its reintroduction through hydrogen water, or inhaling hydrogen gas, has the potential to be not just therapeutic, but restorative, and perhaps, preventative. Reintroducing hydrogen, for those deficient, could very well reactivate ancient biological rhythms which our environment has muted, but our bodies have not forgotten.

Hydrogen & Metabolic Regulation

As I write this, in the spring of 2025, for the first time I am confidently able to relay that molecular hydrogen (H_2)[7] isn't just riding a wave of theoretical promise, and that the clinical research results, both their trajectory and progression, are indicating tangible physiological benefits across nearly every major axis of metabolic health. H_2 has repeatedly demonstrated the ability to improve lipids, glucose, insulin sensitivity, inflammatory markers, appetite hormones, and even central signaling in the brain involved in regulating appetite. Hydrogen has demonstrated this in numerous clinical trials, many of these targets repeatedly replicated. On its own this is remarkable, but in context this becomes even more profound. What makes hydrogen different isn't that it fixes one thing, rather, it's that it regulates many things at once. Unlike drugs that target a single receptor or pathway, hydrogen doesn't hammer systems into submission, but instead restores regulatory intelligence. It helps your body remember how to function properly, guiding complex systems back towards optimal performance.

Two recent meta-analyses confirmed consistent effects across numerous clinical trials. One, by Nikola Todorovic and colleagues (2023), found that hydrogen-rich water (HRW) significantly lowered triglycerides, LDL and total cholesterol, without impacting HDL. Another, by Hamid Jamialahmadi and colleagues (2024), confirmed similar trends in patients with metabolic syndrome: improvements in blood lipids, insulin function, and inflammatory markers. The team, however, reported that the lack of alteration of HDL cholesterol indicates an inconsistency, fundamentally misunderstanding the mechanisms in which molecular hydrogen employs its physiological effects.

[7] There are many methods of producing hydrogen-rich water, which lead to potentially exponential differences in concentrations and dosages. The high-concentration open cup hydrogen tablets I invented deliver the highest concentration and dosage of any technology, consistently. They are able to do this by utilizing elemental magnesium and organic acids, processed and compressed in a manner that facilitates the production of very small nanobubbles in the 10-30nm range. At the time of this writing, the open-cup hydrogen tablets have more published clinical research than all other commercial hydrogen-water technologies combined.

Interestingly, concerning Jamialahmadi and colleagues (2024), the team opted not to include two specific clinical trials with positive findings that were reported in the Todorovic and colleagues (2023), Zanini and colleagues (2021) and Korovljev and colleagues (2019), for reasons not disclosed. Jamialahmadi and colleagues (2024) reported that they had removed trials which were not double blind RCTs; however, both of these removed studies were double-blinded, placebo-controlled, and randomized. Jamialahmadi instead opted to include two additional papers with substantially weaker findings. Of note, one paper cited by Jamialahmadi and colleagues (2024), which consisted of the largest study group in their analysis, delivered roughly 1/10th the dosage of molecular hydrogen reported in the removed studies (Liang et al. 2023), and the other, also of substantial sample size, did not, in fact, report any concentration or dosage of hydrogen, with the trial finding null results (Ogawa et al. 2022).

While the omissions may seem suspicious at first glance, a more likely explanation is methodological incompetence rather than malice: Jamialahmadi's team had no prior experience in hydrogen research, and like many groups entering a new field, likely believed conducting a review was the best way to get up to speed. This is a common and understandable approach, however, for complex topics with an oft misunderstood mechanism, such as hydrogen, this strategy can lead to incorrect conclusions through inaccurate assumptions. Without deep familiarity, such reviews often misjudge study quality or relevance, especially in a field where nuance matters.

Concerning the currently published clinical trials, the most robust evidence comes from Dr. Tyler LeBaron's 6-month RCT, published in two waves (2020, 2022), where participants consumed over 11 mg/day of H_2, provided from the open-cup hydrogen tablets. Without diet or exercise interventions, participants experienced the following outcomes:

- Triglycerides dropped by approximately 47 mg/dL
- Total cholesterol decreased by up to 27.5 mg/dL
- Fasting glucose fell from 121.5 to 103.1 mg/dL
- HbA1c levels declined by 12%
- Systolic and diastolic blood pressure normalized to healthy ranges
- BMI and waist–hip ratio were both reduced
- Inflammatory markers, including TNF-α and IL-6, showed significant decreases
- Plasma nitrite levels increased, indicating enhanced nitric oxide production

Changes like this would usually indicate participants underwent substantial lifestyle alterations, such as a strict dietary program, introducing exercise to a sedentary

population, or both. Of course, this was neither, it was a placebo-controlled study and indicates that simply adding high-dose hydrogen water to their daily routines has meaningful therapeutic benefits. This was not a pharmaceutical intervention, in which we may discover downstream damages years in the future; it was a slow recalibration of the physiological functions which had drifted off course due to lifestyle choices.

For the metabolically impaired, hydrogen water's effects extend to liver health, also. In patients with non-alcoholic fatty liver disease (NAFLD), which is a lifestyle induced and reversible syndrome closely related to metabolic syndrome, a 28-day trial (Korovljev et al., 2019) showed reductions in liver fat and inflammation. Hydrogen-rich water intake also showed a tendency to increase serum insulin levels in NAFLD patients, although this change was not statistically significant in the published results ($P = 0.15$). However, a secondary analysis using the HOMA2 model of insulin sensitivity was later presented as an abstract at the 24th Annual Congress of the European College of Sport Science, suggesting preliminary improvements in insulin dynamics (Korovljev et al., 2019b). Moreover, follow-up studies (Sumbalova et al., 2023; Kura et al., 2022) demonstrated improved BMI, triglyceride/HDL ratios, lowered NF-κB, mitochondrial reprogramming, and reduced lipid peroxidation. Across all trials, the story was the same: regulation, not suppression. Of note, all 3 of these research groups utilized the open-cup hydrogen tablets.

Even hormonal regulators are in play. In a 2024 double-blind placebo controlled RCT following obese individuals consuming 15 mg/day of HRW, generated by the open-cup hydrogen tablets, or placebo control, for 8 weeks, the authors reported that the HRW group saw a significant rise in GLP-1, a reduction in cravings, improvements in reported sleep parameters, and lowered cholesterol (Todorovic, 2025a). In another trial utilizing the open-cup hydrogen tablets, ghrelin, often popularly referred to as "the hunger hormone," despite its numerous other functions, saw a sharp increase in the hydrogen group, along with improvements in other metabolic measurements (Korovljev et al., 2023a). This is relevant, because in metabolically broken systems, such as overweight or obese individuals, ghrelin is often dysregulated. In healthy individuals we expect to see ghrelin spike when hungry, and plummet after eating. In metabolically compromised people ghrelin is often stagnant, never rising and never plummeting, leading to a perpetual state of feeling slightly hungry. Additionally, ghrelin plays a critical role in insulin secretion, glucose homeostasis, energy homeostasis, and provides neuroprotective functions (Akalu et al., 2020; Lin et al., 2019; Kunath et al., 2015). Dysregulating this critical hormone can have substantial ramifications, and hydrogen demonstrated the ability to restore proper function.

H_2's restorative effects on the drivers of hunger don't stop with hormones, either. A 2023 pilot trial, again using the open-cup hydrogen tablets, used MRS imaging to

show shifts in central neurometabolites linked to satiety after 12 weeks of daily high dose HRW consumption: glutamate and glutamine dropped in key regions, GABA fell in the prefrontal cortex, and taurine increased (Korovljev et al., 2023b). These are the same kinds of shifts you'd expect from fasting, ketosis, or deep caloric restriction. This suggests that hydrogen influences central satiety circuits, not just peripheral metabolism.

Additionally, the aforementioned 2023 pilot trial by Korovljev and colleagues (2023a) showed that HRW boosted gut-derived short-chain fatty acids (SCFAs), which are key players in mitochondrial function, neuroendocrine signaling, and inflammation resolution. Propionic acid levels spiked and acetic and butyric acids trended up, all within two weeks. Central to H_2's potential effects on SCFAs may be its promotion of a healthier and more diverse microbiome. H_2 has shown to positively impact the gut microbiota across numerous studies in various species and models (Ostojic, 2021), including the demonstrated ability to raise akkermansia in a model of NAFLD (Jin et al., 2021). Akkermansia is substantively linked to metabolic regulation, with one systematic review of its role posing the question if it is the "holy grail for ameliorating metabolic diseases" (Yan, Sheng, & Li, 2021).

What ties all of this together is that hydrogen isn't acting like a drug, it's acting like a high level corporate auditor, inspecting underperforming offices or facilities to identify and fix problems that have led to poor optimization. Like the auditor, hydrogen isn't building anything new, but simply correcting mismanaged protocols in order to improve performance and avert disaster.

When considering metabolic function, H_2 doesn't suppress appetite, spike hormones, or lower glucose by force, it is not acting as a direct agonist or antagonist. Instead, it recalibrates the system so those outcomes happen on their own. This is the difference between a drug, and a lifestyle intervention. H_2 is undoubtedly the latter: true regardless of our awareness. Lifestyle is more than what we intuitively know, or have passed down through culture, written into our consciousness. H_2 represents an integral component of our evolutionary life, one that silently accompanied our journey through time and space, until very recently. It is a lifestyle component we didn't lose, per se, because we weren't aware we had it; it is a silent guardian forced to abandon us due to our choices.

Necessity is the mother of all invention, but it is also the mother of all of our drive to understand. We never had to understand hydrogen's role, not until we lost it. Now that it has all but vanished, we must learn to put the pieces back together and seek to understand the role it has played guiding our physiological processes and protecting us from the stressors of the world. In a world where our fundamental health pillars have been toppled by modern life and corporate greed, H_2 may be the easiest to restore, and it can be the backbone giving those struggling to start the strength to resurrect the rest.

Hydrogen and Extending Healthspan

When I first started down the rabbit hole with molecular hydrogen (H_2) in 2015, studying its early research, my younger self was convinced that hydrogen could substantially extend human lifespan. By the time my tablets got to market in late 2016, my position had softened. However, I still referred to H_2 as "the most promising anti-aging supplement on the market." As I write this in 2025, my positions have notably, and quite substantially, changed. I say this while arranging to initiate a massive rodent healthspan and longevity study utilizing the hydrogen tablets. I do not expect any substantial extension in maximal lifespan, but I do expect considerable improvements in health span and reduction in age-related degenerative changes.

Understanding this difference is important. A promising healthspan extension intervention addresses the root causes of age-related deterioration, working to delay disease onset by preserving healthy function of the body through protecting cellular integrity, signalling, preservation of hormonal levels, and slowing the deterioration of mechanical degradation, i.e., wear and tear of our musculoskeletal system. Interventional studies across multiple species, including human clinical research, have demonstrated H_2's ability to shift key biomarkers of aging and improve late-life physiological performance.

Hydrogen has demonstrated a multifaceted influence on key aging regulators. Importantly, H_2 has shown to simultaneously affect multiple markers critical for health, and implicated in longevity research. For instance, H_2 demonstrated the ability to protect SIRT1 activation, NAD+/NADH ratios, and prevent cellular senescence when exposed to substantial external stress. Fumihiko Hara and colleagues (2016) reported that when human umbilical vein endothelial cells were exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), which is the toxic compound found in Agent Orange, substantial impairment of NAD+/NADH ratios, SIRT1, and an increase in cellular senescence were all observed. In contrast, the cells exposed to the same toxin in conjunction with a hydrogen-rich medium did not experience any of these deleterious effects.

This is significant as hydrogen doesn't merely elevate NAD+ levels, or activate a single enzyme like SIRT1 in isolation, but instead harmonizes the entire system. The combination of improved redox signaling, SIRT1 activation, and senescence inhibition suggests hydrogen works across multiple aging axes rather than through a single pathway. This is characteristic of a truly effective geroscience intervention, and this stands in contrast to NAD+ precursors which, despite considerable hype, have shown minimal benefits beyond raising NAD+ itself, even at doses 10-15 times higher than typically marketed to consumers.

Hydrogen's relationship with autophagy exemplifies its regulatory, rather than stimulatory, nature, as well. Studies have revealed hydrogen's remarkable ability to

bidirectionally modulate autophagy based on cellular needs; activating this cellular recycling mechanism when beneficial while inhibiting it when excessive activation would prove harmful. Several studies demonstrate hydrogen's capacity to upregulate autophagy in neuroprotective contexts (Zhang et al., 2021; Wang et al., 2018; Bai et al., 2016), while others show its inhibition of excessive autophagy in models where overactivation would cause harm (Gong et al., 2022; Zhang et al., 2017). This precise calibration of autophagy is crucial for aging, as both insufficient and excessive autophagy contribute to age-related dysfunction.

Hydrogen's simultaneous ability to inhibit cellular senescence and regulate apoptosis, demonstrated across multiple tissues and models, suggests it maintains cellular quality control through complementary mechanisms. By preserving functional cells while facilitating the removal of damaged ones, hydrogen maintains tissue homeostasis without triggering the inflammatory cascade typically associated with senescent cell accumulation. This positions hydrogen as a uniquely balanced intervention in addressing the 'cellular garbage' theories of aging.

Biomarkers of Aging and Human Trials

In a 6-month randomized controlled trial of elderly participants, aged 70 and older, Dragana Zanini and colleagues (2021) administered 7.5 mg/day of HRW, generated by the open-cup hydrogen tablets, and assessed a wide range of phenotypic and molecular markers of aging. Findings included: telomere length increased by 4.1% in the HRW group (from 0.99 ± 0.15 to 1.02 ± 0.26), while they decreased 11.1% in the control group (from 0.92 ± 0.27 to 0.79 ± 0.15), with a significant time × treatment interaction ($p < 0.05$). The dramatic contrast between telomere changes in the two groups is explained by the study's timing during the early COVID-19 pandemic. Elderly participants were effectively under lockdown conditions, experiencing minimal mobility and increased psychosocial stress from isolation, both of which are factors well-documented to accelerate biological aging (Faraji & Metz, 2021). The placebo group's 11.1% loss of telomere length in just six months reflects these harsh conditions, while the hydrogen group's telomere preservation and growth suggests HRW may buffer against both environmental and psychosocial stressors, not just biological ones (Zanini et al., 2021).

TET2 expression, a gene associated with age-related regenerative decline and mitochondrial stability, significantly increased in the HRW group (from 0.81 ± 0.52 to 1.13 ± 0.82). This elevation of TET2 expression is particularly noteworthy as this epigenetic regulator has been implicated in the rejuvenation effects observed in "vampire mouse" studies, in which young blood infusions restore aged mice's muscle regeneration and stem cell activity (Zanini et al., 2021). In addition to its role in skeletal tissue repair, TET2 has also been linked to enhanced neurogenesis and cognitive function, further underscoring its significance in age-related regenerative processes (Gontier et al., 2018; Conese et al., 2017). TET2 serves as a molecular switch that influences stem cell function, inflammatory resolution, and tissue

regeneration. Hydrogen's ability to upregulate this key epigenetic regulator may mechanistically explain the improved chair stand performance observed in the HRW group, connecting molecular changes to tangible physical benefits, as TET2 plays a critical role in muscle stem cell maintenance and inflammation resolution. You read that correctly; after 6 months of isolation and stress, the hydrogen water group experienced an increase in physical fitness, not a decline, which is truly remarkable (Zanini et al., 2021).

Improvements in brain metabolites were also recorded: choline and N-acetylaspartate increased in multiple cortical regions, while creatine levels rose in white matter. Tangible real world improvements also occurred. As noted, HRW participants showed significant improvements in chair stand performance, and also in sleep quality, and the pain domain of quality-of-life scores (VAS) (Zanini et al., 2021).

Meanwhile, oxidative stress markers such as malondialdehyde and ferric reducing antioxidant potential strongly trended toward reduction, while serum magnesium levels also increased marginally, consistent with reduced inflammation and improved energetic efficiency. Of note, the placebo control in the study delivered an identical amount of magnesium as the magnesium-based hydrogen water tablets utilized for the experimental group (Zanini et al., 2021). These outcomes further indicate that HRW may attenuate biological aging across multiple domains widely debated as imperative for any intervention, such as telomere attrition, redox imbalance, neurometabolic decline, mitochondrial insufficiency, and loss of physical function.

Elevated Endogenous Hydrogen in Centenarians

Supporting this is an observational study by Yuji Aoki (2013), which found that Japanese centenarians had significantly higher levels of exhaled hydrogen gas (59.4 ± 62.6 ppm) than both healthy younger adults (17.7 ± 19.6 ppm) and elderly patients with type 2 diabetes (23.2 ± 19.0 ppm). The authors proposed that:

> *Increased intestinal production of hydrogen gas might contribute to longevity in Japanese centenarians, and it is presumably related to the diet and gut microbiota.* (Aoki, 2013)

Importantly, breath H_2 levels in centenarians' offspring were intermediate (37.8 ± 27.2 ppm), suggesting a possible heritable or environmental component, potentially mediated by shared microbiome traits or early-life exposures rather than direct genetic inheritance. This natural experiment, occurring without intervention, suggests hydrogen may be a persistent marker, and possibly driver, of extreme healthspan.

Preclinical Evidence Across Model Organisms

In addition to human data, multiple animal studies support the role of hydrogen in extending lifespan or delaying age-related decline. In a 2020 study, hydrogen gas

exposure significantly extended lifespan and reduced oxidative stress in C. elegans (nematode worms). Gene expression shifts included downregulation of age-1 and let-363 and upregulation of ins-18, indicating modulation of known aging-related pathways such as insulin/IGF-1 signaling and mTOR (Zhang et al., 2020). Likewise, exposure to hydrogen-rich water improved survival rates in fasting fruit flies, suggesting a potential increase in stress resistance and resilience, a core attribute of extended healthspan (Chao, 2019).

Finally, in a 2018 study, transgenic mice with elevated oxidative stress showed reduced neurodegeneration, improved memory, and increased median lifespan when given hydrogen-rich water. In the accompanying human arm (MCI patients), cognitive improvements were significant in APOE4 carriers. Importantly, the mice received a relative dose roughly 42.7x higher than the human participants, with a substantially longer duration of treatment as compared to biological aging of the respective organisms. Humans received an estimated 0.36mg of molecular hydrogen via hydrogen water, however, to match the dosage the mice received they would have needed to receive 15.36mg of H_2 through hydrogen water (Nishimaki et al., 2018).

These findings converge on a consistent theme: hydrogen may not just protect against disease, but delay the processes that lead to its emergence, preserving mitochondrial signaling, stem cell viability, redox balance, and neurocognitive function.

From Disease Mitigation to Functional Preservation

Healthspan is not measured in years but in capacity. Across species, hydrogen intake is associated with extended survival under stress, delayed functional decline, and improved markers of systemic resilience. In humans, even in the absence of dramatic structural change, markers such as telomere integrity, mitochondrial biogenesis, redox status, and neurometabolite balance all point in the same direction.

As I have repeatedly stated, hydrogen's role appears to be regulatory, not stimulatory. Hydrogen's role is not one of forcing growth or preventing death, but rather buffering the system against collapse, preserving flexibility, and extending the duration of physiological competence.

Redox Homeostasis and Inflammatory Regulation

One of the most profound misunderstandings in both modern medicine and supplement science is the idea that oxidative stress and inflammation are unambiguously damaging, and that their complete suppression is the key to improving health and extending lifespan. This rationale has fueled decades of overuse of high-dose antioxidant supplements, and pharmaceutical agents designed to be anti-inflammatory. Yet paradoxically, interventions designed to eliminate these processes often increase mortality, accelerate disease progression, or blunt physiological adaptation.

For decades, we've been sold the idea that more antioxidants equal longer life, and this messaging did not stop when research on the subject set off alarms. As early as 2007, large-scale meta-analyses began to tell a different story. One landmark review (Bjelakovic et al., 2007) covering 68 randomized trials found that high-dose vitamin E, beta-carotene, and vitamin A increased all-cause mortality. Later studies reinforced the finding: antioxidant megadoses interfere with the very processes they're supposed to protect (Merry & Ristow, 2016; Ristow et al., 2009). They derail adaptive signaling, distort immunity, and blunt the physiological responses we rely on to grow stronger. Research on the subject has slowed down drastically, scientists in the field clearly getting the message and understanding the risk. Unfortunately, the same does not hold true for industry, or society at large.

The same story plays out for anti-inflammatories. Chronic use of NSAIDs, particularly COX-2 inhibitors, is linked to gastrointestinal bleeding, renal impairment, and an increased risk of cardiovascular events (Grosser, Yu, & Fitzgerald, 2010). While short-term inflammatory suppression can provide symptom relief, long-term inhibition of inflammatory cascades often impairs healing, delays adaptation to stress, and increases vulnerability to infection and chronic disease.

Neither oxidative stress nor inflammation are inherently pathological; they are signaling systems which are integral to mitochondrial function, immune activation, tissue remodeling, and neuroplasticity. The problem is not their existence, but their chronic dysregulation. What the body needs is not suppression, but intelligent recalibration.

For all physiological processes, it's paramount that they are never permanently stuck in the "on" or "off" position, each being equally as damaging. Imagine yourself in a nuclear shelter, deep underground, with no access to natural light. If the artificial lights are stuck "on" all of the time, you will slowly go mad; your circadian rhythm will be disrupted, your sleep will deteriorate, and with it your health and sanity. Conversely, if the artificial light breaks, permanently stuck in the "off" position, your very ability to survive will be impaired. Navigating challenges, even day to day tasks to maintain survival, will become difficult, even insurmountable. This analogy holds true for both inflammatory response, and oxidative stress, with a key and added piece of nuance: the lights in our body shouldn't just maintain the ability to switch on or off, but they should do so alongside an appropriate "dimmer" switch, appropriately setting the intensity for each task.

Hydrogen as a Redox and Inflammatory Regulator

This is where molecular hydrogen (H_2) stands apart. It does not indiscriminately scavenge free radicals, nor does it block cytokine signaling. Instead, it functions as a regulatory molecule that restores redox balance and inflammatory tone without impairing physiological responsiveness.

Unlike high-dose antioxidants, hydrogen selectively neutralizes only the most cytotoxic ROS, such as hydroxyl radicals (•OH) and peroxynitrite ($ONOO^-$), while preserving essential ROS like superoxide and hydrogen peroxide that are vital for cell signaling, immune activation, and adaptation (Artamonov et al., 2023; Tian et al., 2021). This selectivity is key: hydrogen acts where needed, but does not interfere with beneficial oxidative eustress.

At the transcriptional level, hydrogen has been shown to activate the Nrf2 pathway, increasing the expression of antioxidant response elements such as glutathione peroxidase, superoxide dismutase, catalase, and heme oxygenase-1. This upregulates endogenous defenses, enhancing the cell's capacity to neutralize stress without shutting down the system (Barancik et al., 2020).

Simultaneously, hydrogen attenuates NF-κB activity, the master transcription factor driving pro-inflammatory cytokine production. Studies have shown reductions in TNF-α, IL-6, and CRP in both animal models and human clinical trials following hydrogen therapy (Yildiz, LeBaron, & Alwazeer, 2025). Yet critically, hydrogen does not push the immune system into immunosuppression. It does not induce tolerance or block antigen presentation. Instead, it seems to restore baseline setpoints, toning down hyperreactivity without compromising responsiveness.

This balancing act has been demonstrated across multiple trials using hydrogen-rich water. In the LeBaron et al. 6-month RCT, participants saw significant reductions in: TBARS, malondialdehyde, and diene conjugates (oxidative stress markers), CRP, IL-6, and TNF-α (inflammatory markers), alongside increases in vitamin C and E, suggesting the preservation or enhancement of endogenous antioxidant pools.

In a trial by Zuzana Sumbalova and colleagues (2023), patients showed:

- Decreased TBARS (–11.8%, $p = 0.025$)
- Increased CoQ10 and tocopherols in platelets
- Improved mitochondrial function and redox control at the cellular level

Sumbalova and colleagues (2023) observed milder changes as compared to LeBaron and colleagues (2019), possibly due to the shorter trial duration, primarily suggesting mitochondrial reprogramming, with noted increases in platelet levels of CoQ10. While not dramatic, these findings hint at a pattern: hydrogen may support redox homeostasis and mitochondrial efficiency through subtle, system-wide adjustments rather than blunt-force biochemical shifts.

Why Regulation Matters

Aging is not caused by oxidative stress alone, nor is it driven solely by inflammation. Instead, I argue that it is driven by the loss of resilience, defined by the body's inability to turn these processes on and off as needed. The failure to resolve

dysregulated inflammation, the chronic production of low-level ROS, or the senescent cells stuck in a pro-inflammatory state, all of which represent stuck feedback loops, leads to systemic breakdown and ultimate collapse.

Hydrogen is demonstrating the capability to break these loops. It restores the signal-to-noise ratio in biological systems. It allows the immune system to respond, then resolve. It allows mitochondria to generate ROS during energy production, then recover. It clears damaged molecules without damaging the functional ones. In short, it creates the conditions for homeostatic equilibrium to re-emerge.

This regulatory function mirrors how other beneficial hormetic stressors operate. Exercise, for instance, acutely increases both oxidative stress and inflammatory markers, triggering a cascade of ROS production and inflammatory cytokine release. Yet this transient stress activates adaptive responses that ultimately improve redox balance, enhance antioxidant enzyme production, and optimize inflammatory resolution capacity. The body emerges stronger precisely because these systems were challenged, not because they were protected from activation. This explains why blunt antioxidant supplementation can sometimes block exercise benefits, namely by potentially interfering with the necessary redox signaling that drives adaptation. Hydrogen's selective action preserves these vital signaling pathways while preventing their chronic dysregulation, making it uniquely compatible with other hormetic interventions rather than working against them.

In this way, hydrogen occupies a unique role in modern intervention science. It is not a stimulant. It is not a suppressant. It is a resilience enhancer, working at the level of the cellular control system itself.

Hydrogen as an Exercise-like Mimetic

Exercise is one of the most powerful interventions for extending healthspan, improving metabolic function, and modulating the hallmarks of aging. Its benefits arise not from avoiding stress, but from engaging with it: exercise acutely increases oxidative stress, inflammatory cytokines, and mitochondrial ROS production, triggering a cascade of adaptive responses that ultimately enhance resilience. This dynamic, biphasic effect—initial disruption followed by systemic adaptation—is the signature of hormetic stress.

Yet, while the benefits of exercise are well-established, they are not always accessible. Injury, frailty, disability, or chronic disease often prevent individuals from engaging in the intensity or duration of physical activity required to achieve meaningful physiological adaptation. This has driven interest in exercise mimetics: compounds or interventions that trigger overlapping molecular responses to those induced by physical activity, without requiring the same mechanical input.

Molecular hydrogen (H_2) appears to be one such agent. Unlike stimulants that force short-term energy output or pharmacologic agents that suppress metabolic feedback,

hydrogen mimics the adaptive stress signature of exercise itself. H_2 has demonstrated the ability to enhance redox tone, improve mitochondrial function, increase fat oxidation, and activate stress-response pathways such as PGC-1α, AMPK, and Nrf2, all of which are also central to exercise physiology.

Overlapping Molecular Signatures Exercise and Hydrogen Both Modulate:

ROS generation → not by eliminating ROS, but by using them to signal adaptation

Inflammatory tone → increasing acute signaling (e.g., IL-6 from myokines) while improving resolution (e.g., IL-10)

Nrf2 activation → increasing endogenous antioxidants like SOD, catalase, glutathione peroxidase

Mitochondrial biogenesis and efficiency → via redox modulation and SIRT1/PGC-1α signaling

AMPK activation → enhancing glucose uptake, lipid oxidation, and autophagy

Skeletal muscle secretome → including potential upregulation of irisin, a myokine critical for mitochondrial uncoupling, fat browning, and neuroprotection

Studies have demonstrated that hydrogen influences PGC-1α, a central driver of irisin expression (Todorović et al., 2025b; Kamimura et al., 2016). By upregulating PGC-1α, hydrogen could enhance irisin release, mimicking the metabolic adaptations seen with exercise. These adaptations include increased mitochondrial density, improved glucose handling, and increased white-to-brown fat conversion. While direct evidence in humans is still emerging, this mechanistic pathway positions hydrogen as a possible modulator of the exercise–irisin axis, with relevance for both obesity and aging.

Preclinical evidence supports these links. In a study by Jonatas Nogueira and colleagues (2018), rats undergoing a forced swim test, which is a model of exercise-induced oxidative stress, showed that hydrogen supplementation potentiated the ROS response, rather than suppressing it. This amplified oxidative signal contributed to enhanced mitochondrial adaptation, reinforcing hydrogen's role as a hormetic modulator rather than an antioxidant suppressor.

Functional Parallels in Human Trials

Clinical trials support these mechanistic overlaps. Tyler LeBaron and colleagues' 6-month RCT, highlighted in previous sections, provides insights for hydrogen's role as an exercise mimetic, also. Hydrogen-rich water led to a reduction in resting heart

rate, lower systolic and diastolic blood pressure, improved lipid profiles and fasting glucose, and increased endogenous antioxidant levels, with reduced markers of oxidative damage. These are the same endpoints that improve with long-term aerobic training, even though participants in these studies made no changes to physical activity or diet.

In Zanini and colleagues (2021), elderly subjects consuming HRW for 6 months demonstrated improved sit-to-stand performance and brain metabolite restoration, indicating improvements in neuromuscular coordination and metabolic energy supply—both of which typically decline with age and improve with resistance training or mobility exercises. This preservation of strength and muscle has also been shown in rodent models. Seyedeh Nazari and colleagues (2023), in which I was the 2nd author and Dr. LeBaron was the co-corresponding author, explored the effects HRW had on a casted-limb model in mice. HRW mitigated the decline in muscle mass and strength observed from immobilization induced atrophy, subsequently potentiating improvements in both regards during the recovery phase. Additionally, HRW reduced muscle fibrosis, serum troponin I, IL-6, TNF-α and MDA, demonstrating its potential as a new therapeutic during physical rehabilitation.

In a separate exercise context, Toshio Mikami and colleagues (2019), co-authored by Dr. LeBaron, reported that drinking hydrogen water improved endurance performance, reduced psychometric fatigue, and preserved exercise-induced oxidative signalling. These findings demonstrate that hydrogen supports, rather than blocks, the adaptive stress response initiated by exercise training.

These effects have also been observed in structured training interventions. In a 4 week randomized, placebo-controlled trial involving elite female athletes, Mkrtych Ogannisyan and colleagues (2025), which both myself and Dr. LeBaron co-authored, found that HRW significantly reduced creatine kinase (CK) and myoglobin levels post exercise, while facilitating positive alterations in body composition through an increase in muscle mass, and decrease in fat mass. Additionally, the athletes experienced an improvement in peak torque in isometric training—more prominently post-exercise demonstrating the anti-fatigue effect—all while increasing levels of IL-10, which is an anti-inflammatory cytokine, without blunting IL-6, thereby preserving training induced signalling.

A second trial by Jovan Kuzmanovic and colleagues (2024), in which I am an author and helped present at ESPEN in Milan, September 2024, investigated HRW in exercise-naïve adults over 50 undergoing a 6-week resistance training program. In this study, the placebo group experienced substantial stress, with sharp and alarming rises in creatine kinase and myoglobin, demonstrating the initial harm of introducing exercise to older, uninitiated individuals. In contrast, the HRW group saw no significant rise in either CK or myoglobin. Additionally, important hormonal changes occurred in the HRW group, with the HRW group observing

beneficial alterations of cortisol, showing better stress adaptation, and also increased DHEA and free testosterone. Specifically in the female participants, HRW led to improvements in subjective recovery, sleep quality and training tolerance.

These studies demonstrate that hydrogen not only mimics certain molecular signatures of exercise but also improves functional training outcomes, recovery, and redox-inflammation balance across the extremes, namely, in both younger elite athletes, and unfit and aging populations.

These findings were recently synthesized in two systematic reviews. A 2024 meta-analysis concluded that H_2 supplementation can reduce exercise-induced fatigue and perceived exertion while simultaneously improving aerobic capacity and antioxidant status, though additional large-scale trials are still needed for confirmation (Li et al., 2024). Additionally, a 2024 meta-analysis published in Frontiers of Nutrition, for which Dr. LeBaron was a guest editor, analyzed hydrogen in the context of physical training and found statistically significant reductions in lactate and perceived fatigue, with pooled data suggesting a meaningful ergogenic effect on exercise performance and redox recovery (Li et al., 2024).

Long before these studies and reviews entered the literature, a 2019 article by LeBaron and colleagues, published in the *Journal of Physiology and Pharmacology* predicted these effects, proving quite prescient. LeBaron and colleagues wrote:

> *Beneficial exercise and H_2 administration promote cytoprotective hormesis, mitochondrial biogenesis, ATP production, increased NAD^+/NADH ratio, cytoprotective phase II enzymes, heat-shock proteins, sirtuins, etc.... we propose that hydrogen may act as an exercise mimetic and redox adaptogen, potentiate the benefits from beneficial exercise, and reduce the harm from noxious exercise. However, more research is warranted to elucidate the potential ergogenic and therapeutic effects of H_2 in exercise medicine.* (LeBaron et al., 2019b)

This proposed hormetic model of H_2 as an exercise-like mimetic has continued to garner scientific support. Dr. LeBaron's review acted as the guiding light for the research direction observed in the following years. As much as Tyler has helped guide this emerging industry from the commercial side, protecting it from greed and dishonesty, he has also played an integral role in guiding academia and research direction. His contributions to this field cannot be overstated.

In short: hydrogen doesn't replace exercise, but it won't impair it like some supplements, and it may even amplify its benefits, preserve its molecular signaling, and possibly provide partial mimetic effects when exercise is not possible.

An Adaptive Stress, Not a Substitute

It is critical to distinguish between mimetics that trigger output (e.g., stimulants

increasing heart rate or lipolysis) and those that mimic adaptation. Hydrogen falls in the latter category. It does not increase performance by overriding fatigue, nor does it suppress stress systems in a way that blocks adaptation. It modulates stress, which allows mitochondrial signaling, redox fluctuation, and inflammatory cascades to occur within a controlled range.

This places hydrogen in a unique category: a redox-mediated exercise-like mimetic that can support adaptation in the elderly, the injured, and the metabolically compromised, while also serving as a potentiator in healthy individuals already training. As with physical activity, the benefits of hydrogen are not the result of suppression or pharmacologic overcompensation. They arise from engaging with stress: intelligently, proportionally, and through the body's own regulatory machinery.

Hydrogen as a Calorie Restriction Mimetic

Calorie restriction (CR) is the most robust non-genetic intervention known to extend lifespan across a wide range of organisms. Its benefits arise from a systemic shift in how the body perceives and responds to energy scarcity: increased autophagy, enhanced mitochondrial efficiency, improved insulin sensitivity, and the activation of stress-responsive transcription factors like AMPK, FOXO, PGC-1α, and SIRT1 (Jun et al., 2024; Wu et al., 2022; López-Lluch & Navas, 2016).

Since the beginning of the 21st century interest in calorie restriction mimetics (CRMs) has exploded, CRMs being compounds that attempt to activate the same pathways which calorie restriction triggers without requiring a sustained caloric deficit. Among the most researched are acarbose, resveratrol, and curcumin, but what unites them may not be the pathways they target directly, but rather a shared, underappreciated mechanism: increased endogenous hydrogen production.

Acarbose: Fermentation-Driven Hydrogen as the Primary Driver

Acarbose delays carbohydrate digestion in the small intestine, allowing more undigested starch to reach the colon. There, it becomes substrate for bacterial fermentation, which leads to increased hydrogen gas production in the gut (Suzuki et al., 2009; Weaver et al., 1997). This may be central to its effects. Mice treated with acarbose exhibit elevated breath hydrogen, improved glucose control, and extended lifespan. Interestingly, these effects are especially prominent in males (Harrison et al., 2019). Traditionally, these effects were attributed solely to glycemic blunting, but emerging evidence suggests that endogenous H_2 produced via colonic fermentation may be the real modulator. By increasing endogenous H_2, antioxidant and anti-inflammatory pathways are engaged, protective gene networks are upregulated, and key effects of CR are mimicked through hydrogen-mediated signaling. In this context, hydrogen is not just a downstream byproduct, it may be the bioactive messenger carrying the signal of caloric reduction throughout the system.

Curcumin: Microbial and Redox Interactions that May Elevate H_2

Curcumin is a polyphenol with widely documented anti-inflammatory and antioxidant effects, but its low systemic bioavailability has long posed a paradox: how does it exert such broad systemic influence if it barely reaches circulation? One answer may be its interactions with gut microbes, which metabolize curcumin into smaller, bioactive molecules. This biotransformation may lead to increased hydrogen gas production. Some studies have suggested that curcumin may stimulate microbial populations capable of generating hydrogen during polyphenol fermentation (Servida et al., 2024; Enayati et al., 2023).

In parallel, curcumin modulates mitochondrial ROS and redox-sensitive transcription factors like Nrf2 and SIRT1, which are mechanisms identical to those activated by hydrogen (Ashrafizadeh et al., 2020). Whether curcumin's most meaningful effects are mediated through hydrogen has not yet been fully established, but the overlap is striking.

Resveratrol: A Mitochondrial Source of H_2?

Resveratrol activates SIRT1 and improves mitochondrial function in several models, particularly under metabolic stress (Denu, 2012; Lagouge et al., 2006). Intriguingly, recent work has proposed that resveratrol metabolism itself may produce molecular hydrogen as a byproduct. The hypothesis notes that this may be possible, particularly within mitochondrial compartments where redox reactions are most intense (Pshenichnyuk & Komolov, 2015).

This raises the possibility that resveratrol's actions, long attributed to direct SIRT1 activation, may also involve redox modulation via hydrogen generated in situ. In this scenario, the benefits of resveratrol are not just due to gene activation, but to localized mitochondrial signaling, and hydrogen molecules produced during its metabolism. If validated, this would place hydrogen at the core of intracellular calorie restriction mimicry, not just at the periphery.

Hydrogen: The Common Denominator

Viewed through this lens, hydrogen is no longer just a calorie restriction mimetic. It may be a key messenger shared across multiple interventions that extend lifespan, improve stress resistance, and enhance metabolic flexibility.

Hydrogen's biological reach is surprisingly broad for a molecule so simple. As previously discussed, at the metabolic level it activates AMPK, SIRT1, and PGC-1α, which are all critical regulators of energy sensing, mitochondrial health, and cellular adaptation. Mitochondrial function and biogenesis improve not just in damaged cells but in healthy tissue as well, suggesting a general upshift in resilience. Hydrogen also appears to regulate autophagy in both directions: promoting it when needed for cleanup, and dialing it back when excessive breakdown becomes counterproductive.

As I write this 6 or 7 years after I first made these observations, a thought occurs to me (I will caution that I have yet to properly think about the implications, and it could very well be meaningless): during our hunter-gatherer days, both as homo sapiens and the hominins which preceded us, times of caloric scarcity were likely characterized by failed hunts and limited, or completely restricted, access to meat. During these times, early humans likely relied on increased consumption of plant-based foods. These foods, prior to modern horticulture techniques, were substantially richer in fiber and lower in caloric density than the plant foods we consume today. As such, early periods of forced caloric restriction likely were accompanied by considerable spikes in endogenous hydrogen production. What role this played in our evolution, and molecular hydrogen's link to both improved survival during fasting (in drosophila), and simultaneous activation of CR induced pathways, is unclear. What is clear to me is that this observation is poetic, either in coincidence, or in our evolved adaptation to our past.

Hydrogen and Stress Resilience

Modern life subjects us to an unprecedented array of stressors, including environmental, chemical, physical, and psychological. While the adaptive stress response is essential for growth and resilience, chronic or excessive activation leads to systemic dysfunction. Molecular hydrogen (H_2) is, again, a uniquely positioned intervention due to it acting both as a mild initiator of adaptive stress, and as a regulator of excessive stress.

A Regulatory Hormetic: Beyond Traditional Stress Adaptation

Unlike traditional hormetic stressors that require substantial damage to trigger adaptation, hydrogen initiates a minimal mitochondrial ROS signal, just enough to activate stress-response pathways without collateral damage. This positions hydrogen at the inflection point of the hormetic curve: the smallest possible signal that still achieves systemic benefits.

What truly distinguishes hydrogen is its dual capacity to both induce mild hormesis and regulate the stress response to other agents (Murakami, Ito, & Ohsawa, 2017). This makes it uniquely suited for combination with virtually any other intervention, amplifying benefits while mitigating excessive damage (i.e., exercise, fasting, heat, cold, polyphenols, etc.).

Radiation Protection: The Ultimate Test Case

Radiation presents perhaps the most extreme oxidative challenge to biological systems. Here, hydrogen's protective effects are profound and well-documented across multiple models.

In rodent studies of whole-body irradiation, hydrogen administration significantly reduced mortality, protected against DNA damage, and preserved organ function

(Zhang et al., 2017). When hydrogen was administered to chickens following the Fukushima incident, researchers observed preservation of immune function and reduced radiation biomarkers (Sawajiri et al., 2016). The precise mechanisms have been elucidated in comprehensive reviews, confirming elevated levels of SOD and phosphorylated-AKT, a critical cell survival signaling molecule. While doing his Ph.D. work, Tyler along with his colleagues also published a number of studies showing that drinking HRW could protect the heart from radiation by regulating microRNAs, and modulating inflammatory and oxidative stress response (Kura et al., 2019).

Most significantly for clinical applications, hydrogen-rich water mitigated radiation side effects in cancer patients undergoing therapy without compromising treatment efficacy, which is a critical advantage over conventional radioprotectants that often interfere with therapeutic outcomes (Kang et al., 2011). This selective protection has led NASA scientists to propose hydrogen as a countermeasure for astronauts during long-duration missions, where cosmic radiation exposure presents a major health risk (Schoenfeld et al., 2011).

Chemical and Pharmaceutical Stress

Hydrogen's ability to regulate stress responses extends to xenobiotic challenges; both environmental toxins and pharmaceutical agents. In controlled models, hydrogen water significantly ameliorated lung injury induced by paraquat, a toxic herbicide linked to Parkinson's Disease (Liu et al., 2011). Similarly, as previously detailed, in cell cultures exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin (a component of Agent Orange), hydrogen preserved NAD+/NADH balance while attenuating cellular senescence (Hara et al., 2016)—addressing two critical features of toxic stress and aging.

For pharmaceutical agents, hydrogen demonstrates remarkable protective capacity while preserving therapeutic benefit. Studies confirm hydrogen water protects against aspirin-induced gastric damage and acetaminophen-induced hepatotoxicity (Zhang et al., 2015; Zhang et al., 2014). Most impressively, in a landmark 152-patient randomized controlled trial, hydrogen water significantly reduced the side effects of mFOLFOX6 chemotherapy while enhancing effectiveness against colorectal cancer (Yang et al., 2017). Additional rodent studies with 5-fluorouracil (5-FU) and cisplatin show similar protective effects, suggesting hydrogen can address one of the most significant barriers to effective cancer treatment, namely, toxicity to non-target tissues (Asgharzadeh et al., 2022; Kamimura et al., 2009).

This was perfectly embodied in Fereshteh Azgharzadeh and colleagues (2022), which both myself and Dr. LeBaron co authored, in which our team demonstrated HRW's ability to potentiate the stress response of the chemotherapeutic 5-FU within the cancer cells. This trial perfectly demonstrated that molecular hydrogen (H_2) does not, in fact, act as a direct antioxidant in vivo. We designed this study with four groups:

control, 5-FU, HRW, and 5-FU and HRW. The HRW and 5-FU groups saw similar reductions in tumor weight and size as compared to control, and also in reduced collagen content in the tumor. As expected, 5-FU increased ROS and decreased antioxidant activity, where HRW increased antioxidant activity and decreased ROS. The combination group, however, saw something entirely different. The tumor weight and size were significantly lower than either the HRW or 5-FU groups on their own, and the integrity of the tumor was substantially impaired, collagen content dropping to just 3%.

For contrast, the HRW and 5-FU groups had experienced collagen reductions to 13% each, respectively, with the control group having 24.6% collagen content. Importantly, the stress within the cancer cells was substantially higher in the HRW + 5-FU group as compared to 5-FU alone, with ROS significantly higher, and antioxidant capacity significantly blunted. HRW had potentiated the stress from the chemotherapeutic within the cancer cells, without harming the healthy cells. This ability to selectively modulate stress responses, enhancing beneficial signaling while minimizing maladaptive damage, positions hydrogen as an ideal adjunctive therapy for treatments with narrow therapeutic windows.

Self Administered Stress

Sometimes, the stress that batters our physiology is of our own design, the intent not just drifting from adaptive resilience, but entrenched in self destruction. Of course, I discuss one source of self-administered stress which rarely leads to adaptive improvements, namely alcohol, in its own chapter (7). In this domain molecular hydrogen poses a potential for real, and substantial, protection.

Anecdotally, hydrogen water is the leading go-to for those in the know, to recover from a late night where one too many drinks were indulged in. It's one of the first, and possibly the most consistent, testimonials we have received over the years from the hydrogen tablets. The feedback is so consistent, across all markets where hydrogen water is present, and in such voluminous amounts, that many years ago Dr. LeBaron posed the question to Prof. Ohta in Japan as to why there is no research on hydrogen water for hangover recovery. Prof. Ohta chuckled, and told Tyler, "*Tyler, there is no need to study this as everyone already knows it works*," with the additional caveat, after the joke, that getting grant funds to purposefully intoxicate people poses challenges.

Since then, a small amount of research relevant to this discussion has been published. In Satoshi Yano and colleagues (2021), researchers investigated the protective effects of electrolyzed hydrogen water (EHW) against ethanol-induced cytotoxicity in human liver-derived HepG2 cells. EHW significantly decreased intracellular levels of acetaldehyde, the toxic intermediate produced during ethanol metabolism. Mechanistically, EHW suppressed the activity of alcohol dehydrogenase (ADH), reducing ethanol's conversion to acetaldehyde, while simultaneously enhancing the

activity of aldehyde dehydrogenase (ALDH), accelerating the detoxification of acetaldehyde into acetic acid. These dual effects lowered both aldehyde toxicity and intracellular reactive oxygen species (ROS). The protective actions were directly linked to the concentration of dissolved molecular hydrogen (H_2) in the water, and were abolished when the hydrogen was removed via degassing, confirming that H_2 is the active agent. Additionally, non-electrolyzed hydrogen-rich water with high dissolved H_2 provided comparable protection, suggesting that hydrogen, not platinum nanoparticles or alkaline pH, is primarily responsible for reducing ethanol-induced hepatocellular damage. For those interested, the finding that H_2 is the exclusive agent in electrolyzed water was clearly demonstrated in a comprehensive review by Dr. LeBaron, along with important caveats and safety concerns (LeBaron, Sharpe, & Ohno, 2022).

In Xiang Lv and colleagues (2022), 20 healthy adult participants underwent a randomized, double-blind, placebo-controlled, matched, crossover trial comparing the combination of hydrogen gas and hydrogen water administration against placebo following alcohol intake (100 mL of 40% liquor). In the hydrogen condition, participants inhaled a hydrogen/oxygen gas mixture for 1 hour and consumed hydrogen-rich water post-alcohol. Breath alcohol concentrations (BrACs) were significantly lower in the hydrogen group at 30, 60, and 90 minutes post-drinking, as were hangover symptoms, with a total absence of severe symptoms in the hydrogen group. Additionally, hydrogen intake improved cognitive test scores, particularly in attention and executive function.

Haixia Liu and colleagues (2023) explored chronic administration, wherein mice were exposed to an acute alcoholic liver disease (ALD) model and treated with hydrogen gas (H_2) inhalation to assess its therapeutic potential. H_2 inhalation significantly reduced liver injury, oxidative stress, inflammation, and lipid accumulation in the liver. Mechanistically, H_2 improved intestinal barrier function and reshaped the gut microbiota, specifically increasing beneficial taxa like Lachnospiraceae and Clostridia while reducing potentially harmful taxa such as Prevotellaceae and Muribaculaceae. This microbial shift was associated with accelerated alcohol metabolism, improved lipid homeostasis, and enhanced immune regulation. H_2 also blocked activation of the LPS/TLR4/NF-κB inflammatory pathway in the liver, a key driver of alcohol-induced hepatic damage. Notably, fecal microbiota transplants (FMT) from H_2-treated mice replicated the protective effects, confirming a gut-mediated mechanism. The study concludes that H_2 exerts protective effects in ALD through antioxidant and anti-inflammatory pathways as well as modulation of the gut–liver axis, making it a promising candidate for preventing or treating alcohol-related liver injury. Of note, this study uses hydrogen inhalation, although hydrogen water is better equipped to deliver higher concentrations to the gut, and the liver.

The potential protective effects don't stop at alcohol, with emerging evidence suggesting H_2 may be able to protect against toxicity from illicit recreational

drugs, such as methamphetamine. Di Wen and colleagues published a pair of studies in 2019 and 2020 exploring these protective effects in male C57BL/6 mice. In the first study (Wen et al., 2019), the mice were exposed to high-dose methamphetamine, then treated with hydrogen-rich water (HRW) ad libitum for 7 days. Compared to regular water, HRW significantly inhibited methamphetamine-induced spatial learning and memory impairments (assessed by the Barnes maze and Morris water maze tests). Additionally, HRW "significantly restrained the neuronal damage" and reduced ER stress, which is implicated in neurodegeneration, while simultaneously suppressing the rise in pro-inflammatory cytokines.

In the second study (Wen et al., 2020), the mice were administered escalating doses of methamphetamines over 7 days to induce behavior sensitization, designed to mimic addiction and relapse in humans. Mice treated via hydrogen-rich saline injections every 3 hours during both the acquisition and transfer phases of the sensitization protocol experienced significantly lower meth-induced behavioral sensitization, without affecting baseline locomotor activity. Molecular analysis revealed that expressions within the nucleus accumbens implicated in reward and addiction were significantly lower in the HRS group as compared to control. Additionally, HRS reduced markers of oxidative stress.

Psychological Stress and Cognitive Resilience

Emerging evidence indicates hydrogen's stress-regulatory effects extend to neurological and psychological domains. In rodent models, hydrogen water attenuated depressive-like behaviors, while hydrogen gas inhalation mitigated social deficits associated with chronic stress (Zhang et al., 2016). In a valproic acid model of neurological stress, hydrogen water significantly reduced autistic-like behaviors, demonstrating its neuroprotective potential (Guo et al., 2018).

Human studies further support hydrogen's role in cognitive stress resilience. In two controlled trials comparing open-cup hydrogen tablets to caffeine under conditions of sleep deprivation in habitual coffee drinkers, hydrogen demonstrated both comparable and superior effects. In both studies, hydrogen matched caffeine in improving attention network performance, which supports its role in preserving alertness without stimulant dependence (Todorovic et al., 2021; Zanini, Štajer, & Ostojic, 2020). In the second study in particular, hydrogen led to greater improvements in brain metabolism, specifically increases in the creatine-to-choline ratio, which is a key marker of neuronal energy regulation, as compared to both caffeine and placebo (Todorovic et al., 2021). Moreover, hydrogen led to greater increases in the choline-to-creatine ratio, a marker associated with brain viability and metabolism, in several brain regions, including frontal and paracentral areas, compared to caffeine and placebo. This suggests that hydrogen may exert distinct and potentially broader effects on brain metabolic activity and neuronal energy processes, differing from the mechanisms of caffeine.

These neurometabolic protective effects extend beyond acute stress. In previously discussed studies, elderly and overweight participants who consumed hydrogen-rich water demonstrated significant improvements in brain metabolite profiles. Increases in N-acetylaspartate, choline, and creatine were observed across cortical regions, suggesting hydrogen supports neuronal integrity, mitochondrial function, and cognitive resilience under chronic physiological stressors such as aging and poor diet (Vuković et al., 2024; Zanini et al, 2021).

Taken together, this emerging evidence supports hydrogen's role in protecting brain function across a spectrum of stress conditions ranging from short-term cognitive load to long-term metabolic and age-related decline. Unlike stimulants, which offer temporary alertness at a metabolic cost, hydrogen appears to stabilize and restore underlying neurometabolic balance.

A small 26-participant trial further suggests hydrogen water may positively impact mood and anxiety, though larger studies are needed to confirm these effects (Mizuno et al., 2018). The mechanisms likely involve hydrogen's ability to modulate neuroinflammation, preserve mitochondrial function in neural tissues, and regulate redox-sensitive neurotransmitter systems.

The Universal Stress Regulator

What makes hydrogen unprecedented is not its action within any single stress domain, but its capacity to act as a universal stress response regulator. By initiating a minimal hormetic signal that activates key adaptive pathways (Nrf2, SIRT1, AMPK, PGC-1α), hydrogen primes cellular systems for resilience without adding significant toxic burden (Da, Chen, & Shen, 2024; Kasai et al., 2020; Lee et al., 2017).

Unlike traditional hormetic stressors such as exercise, fasting, heat, cold, and radiation, all of which have clear upper limits where benefits reverse into harm, hydrogen maintains a favorable profile even at high doses. It does not exhaust adaptive capacity or trigger compensatory pro-inflammatory responses. Instead, it enhances the body's intrinsic ability to respond appropriately to diverse stressors, potentiating beneficial adaptations while accelerating recovery from damage (LeBaron et al., 2019a).

This regulatory precision has been demonstrated across a range of stress models. During exercise, hydrogen manages to preserve ROS signaling, which is essential for adaptation, while simultaneously reducing markers of muscle damage like creatine kinase and myoglobin (Todorovic et al., 2020). In a radiation study, it attenuates DNA damage but does so without compromising the fidelity of repair mechanisms, suggesting a kind of molecular triage that knows what to shield and what to let signal (Abou-Hamdan et al., 2016).

Recent clinical evidence confirms these benefits extend even to healthy individuals. A controlled trial demonstrated that hydrogen water reduced inflammation, enhanced

antioxidant responses, and decreased apoptosis in healthy participants, with particularly pronounced effects in those over 30, which suggests that hydrogen addresses even subclinical stress burden that accumulates with age (Sim et al., 2020).

By functioning simultaneously as a mild hormetic signal and a regulator of other stress responses, hydrogen addresses the fundamental challenge of modern stress management: how to build resilience without adding to the already substantial stress burden most individuals carry. Hydrogen is not merely another therapeutic agent, but potentially a foundational component of comprehensive stress resilience strategies for the 21st century.

Hydrogen in Neurodegeneration and Traumatic Brain Injury

Without our mind, what are we? It's a question I have pondered many times. The possibility of substantial neurodegeneration is perhaps the only future which frightens me more than my previous loss of physical capabilities. My mind having been intact, I found ways to overcome the circumstances that shaped my new reality. My mind is still intact, and I am prepared for any random tragedy life decides to surprise me with. However, with an impaired mind, defeat is inevitable, purpose is rendered obsolete, and hope becomes futile. Loss of the mind, with enough cognition remaining to understand what has been lost, strikes a fear into the core of my being which the prospect of death never has.

Saying this, it should come as no surprise that I take the health of my brain seriously, even if some of my past actions suggest otherwise, such as my previous proclivity for drinking, and getting hit in the head repeatedly (kickboxing). Rather, perhaps, I should say that as I age, and realize the weight and capability of my mind, I have taken the preservation of its health more seriously. I have quickly, and substantially, altered my routines, protocols, and hobbies to preserve my brain, rather than to squander it. In fact, one of the key drivers that has kept me working towards a specific purpose over the years, namely the development and launch of mine and Dr. LeBaron's patent-pending hydrogen inhalation unit, is the knowledge that hydrogen inhalation, when administered appropriately, leads to a higher spike of molecular hydrogen (H_2) in brain tissue as compared to hydrogen water.

This is important, as hydrogen therapy has demonstrated meaningful neuroprotective effects in both acute and chronic neurological models. In rodent studies, hydrogen water and gas have consistently attenuated oxidative stress, suppressed neuroinflammation, and preserved neuronal viability across models of Parkinson's disease, Alzheimer's disease, vascular dementia, traumatic brain injury (TBI), and hypoxic-ischemic encephalopathy (HIE) (Lee, Jo, & Choi, 2025; Lin et al., 2022; Wu et al., 2019; Yuan et al., 2018; Fujita et al., 2009). These effects appear to converge around core stress-adaptation pathways, including Nrf2, SIRT1, and mitochondrial stabilization, rather than targeting any disease-specific lesion.

In Alzheimer's models, hydrogen has shown the ability to reduce amyloid-beta cytotoxicity, suppress amyloid-beta-induced inflammation, and mitigate oxidative stress responses linked to plaque formation (Wang et al., 2011; Li et al., 2010). In models of tauopathy, it has reduced tau aggregation and phosphorylation following traumatic injury (Dohi et al., 2014). Additional rodent studies have demonstrated neuroprotective effects in non-amyloid models of neurodegeneration, including ALS and multiple sclerosis (Zhao et al., 2016). Collectively, these findings point toward hydrogen's capacity to influence multiple upstream drivers of neurodegenerative pathology.

This broad-spectrum activity is further exemplified in Parkinson's disease (PD). Early rodent work showed that hydrogen water preserved dopaminergic neurons and improved motor performance. One study attributed these benefits to increased ghrelin secretion (Matsumoto, 2013), while another showed hydrogen remained effective even in ghrelin knockout mice, indicating multiple, redundant mechanisms (Yoshii et al., 2017). Additional hypotheses link impaired microbial hydrogen production to PD development, with published opinion papers proposing that exogenous hydrogen may compensate for this loss (Suzuki, 2018; Ostojic, 2018). Hydrogen's ability to favorably shift microbial populations has been documented in both animal and human studies, adding weight to this theory.

Translating these findings to human trials has been challenging, and also revealing. A 48-week randomized, double-blind, placebo-controlled trial showed improvements in the Unified Parkinson's Disease Rating Scale (UPDRS) (Yoritaka et al., 2013). However, a subsequent 72-week multicenter RCT failed to replicate the effect (Yoritaka et al., 2018). As stated in *The Final Thought War*, "*Other researchers immediately raised concerns about how the placebo was prepared. The study claimed to have used H_2-producing canisters but said they had been reused, resulting in 'negligible' hydrogen exposure for the placebo group. But independent, unpublished testing showed that this method didn't actually work—some canisters still produced therapeutic levels of hydrogen.*" This intermittent placebo contamination likely confounded the results and plausibly explains the absence of a between-group difference, as both arms may have received biologically active doses. A smaller study using hydrogen gas inhalation found no benefit (Hirayama, 2019), which was consistent with rodent findings that hydrogen water is more effective than inhalation in PD models.

Hydrogen's neuroprotective effects are not limited to chronic disease models. In traumatic and ischemic brain injury, its ability to mitigate secondary injury cascades has been repeatedly demonstrated. Rodent studies show that hydrogen administration after stroke or TBI reduces infarct size, preserves blood-brain barrier integrity, suppresses inflammatory cytokines, and improves neurological scores (Hu et al., 2022; Takeuchi et al., 2015). In a 2020 rat study, high-dose open-cup hydrogen tablets combined with minocycline produced a synergistic effect, yielding

significant improvements in post-stroke recovery metrics while attenuating markers of neuroinflammation and oxidative damage (Jiang et al., 2020).

These preclinical results have begun to extend into early-stage human data. A case report described rapid recovery from concussion in a professional soccer player following high-dose hydrogen tablet use (Javorac, Štajer, & Ostojic, 2019). Additional pilot studies in acute ischemic stroke have shown encouraging signals (Ono et al., 2017; Ono et al., 2012; Ono et al., 2011), and a large U.S.-based post-stroke recovery trial using open-cup tablets is now underway. Hydrogen has also shown benefit in neonatal hypoxic-ischemic encephalopathy (HIE), improving survival and neurodevelopmental outcomes, though these results remain to be replicated (Yang, Li, & Chen, 2016).

Even in cognitive decline, where human trials have so far delivered mixed results, dosing appears to be the limiting factor. In a previously discussed study by Nishimaki and colleagues (2018), participants with mild cognitive impairment (MCI) consumed an average of only 300 mL/day of 1.2 ppm hydrogen water which is an estimated 0.36 mg/day of H_2, far below the 0.5 mg/day minimum recommended dose proposed by the International Hydrogen Standards Association. Mice in the same study consumed the same hydrogen concentration ad libitum, receiving over 42 times more hydrogen per body weight. Unsurprisingly, the mice showed strong cognitive and lifespan improvements. In contrast, humans showed significant benefit only among APOE4 carriers, a subgroup more sensitive to oxidative and metabolic stress. This discrepancy may underscore the importance of dose alignment as well as genotype stratification in human trials.

Across both acute trauma and progressive neurodegeneration, hydrogen has repeatedly demonstrated its ability to modulate key stress pathways: amyloid-beta, tau, neuroinflammation, oxidative stress, and mitochondrial dysfunction. Whether administered following injury or used preventatively in high-risk populations, hydrogen does not act as a disease-specific therapy, but rather as a regulator of the brain's intrinsic stress response systems.

Toxic Threshold? When Is It Too Much?

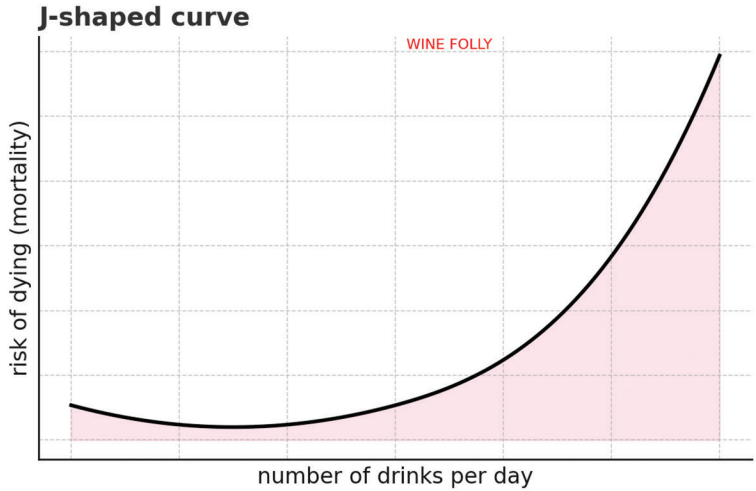
Data we have accumulated from mixed sea diving has led to the understanding that molecular hydrogen can start creating narcotic-like effects at very high doses, when administered under high pressure (Taylor, n.d.). However, it has been shown only to happen at doses higher than even nitrogen, which we know has a high safety threshold, making up 78% of our atmosphere (UCAR Center for Science Education, n.d.). Furthermore, molecular hydrogen has been shown to suppress some of the neurological symptoms of high pressure nervous system symptoms, even when hydrogen narcosis is present, providing another benefit in deep sea diving (Abraini, 1994).

The dosage of hydrogen needed to exhibit signs of narcosis is significantly higher than any commercial method could feasibly deliver to consumers. Thus, hydrogen narcosis (which exhibits no known long-term damages, toxicity, or side effects) is of small concern.

Further demonstrating the safety of molecular hydrogen, clinical research has yet to determine where scaling dosages in various models plateau in beneficial effects, let alone when it leads to adverse events and toxicity. In certain cell lines, molecular hydrogen produces a clear dose-dependent effect—approaching the saturation level (Ren et al., 2016)—but these levels exceed what can be safely or practically achieved in human participants. Research has routinely observed that higher concentrations and dosages, particularly for hydrogen water and research utilizing the open-cup hydrogen water tablets, lead to more profound effects (Timón et al., 2021; Sim et al., 2020; LeBaron et al., 2019b). This seems to be accompanied by a duration effect, as well, with longer durations of treatment trending to stronger results.

As I have repeated throughout this book, other hormetic stressors need to be monitored to prevent them from becoming harmful. For instance, we know that alcohol has a very sharp J-shaped curve, with benefits in a tight range, and harm and damage following very closely after. Conversely, when considering the extremely high safety profile of molecular hydrogen, this warning becomes obsolete.

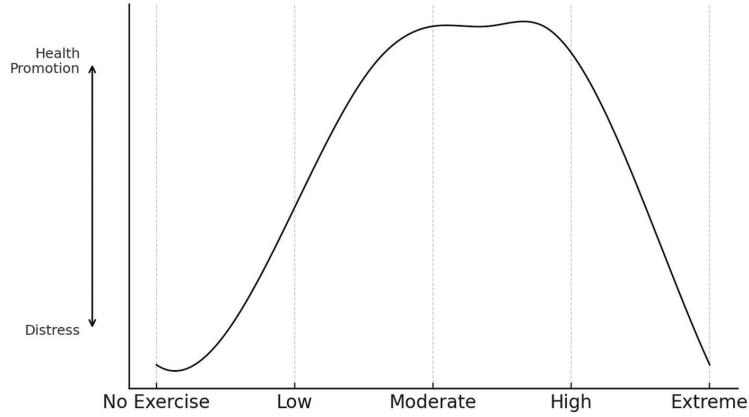
Figure 3. J-shaped curve (again)



Note. From these authors.

Even exercise has a reverse J curve in terms of healthy gene expression that many people overdo. A rough curve may look something like this:

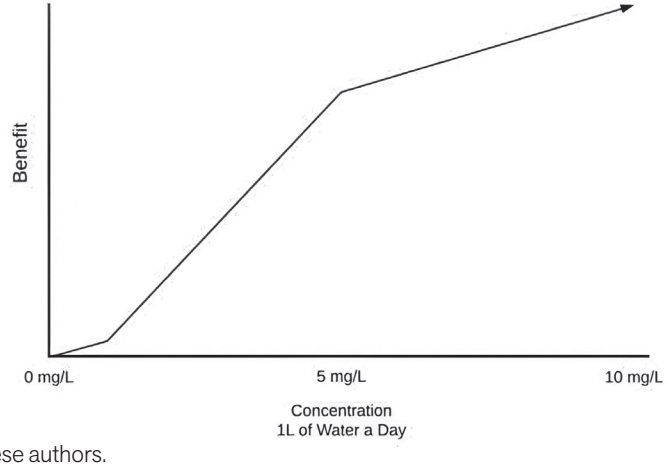
Figure 4. Reverse j-shaped curve



Note. From these authors. "Low," "moderate," "high," and "extreme" levels would be based on individuals, intensity, and many other variables.

Concerning the research on H_2, we have yet to establish plateau's for many indications, nor have we established a downward trend in dose scaling for any indication. In the published research, as dosages increase, findings have either shown stronger physiological effects, or consistency with the lower dosage in beneficial adaptations, with no emergence in side effects. This is incredibly impactful as it shows that molecular hydrogen can be utilized safely alongside other hormetic agents and (likely) medications to improve outcomes in a wide range of models. These benefits will likely be conferred with limited or no inherent risks. A graph of our study on hydrogen water and benefits may look something like this:

Figure 5. Hypothetical H_2 benefits graph



Note. From these authors.

Conclusion: Returning to What Was Missing

Molecular hydrogen (H_2) doesn't ask the body to do anything unnatural. It doesn't override systems, or force rigid responses that may lead to deleterious downstream effects. What it does is simpler, more profound, and fundamental to our vitality. It reminds our biology how to regulate itself, how to heal, adapt, and strengthen. That's what makes it different.

Hydrogen isn't just another hormetic tool in the kit, it's a layer beneath the others, a regulator of regulators. It initiates mild stress, but it also mitigates the fallout from other, more aggressive forms of stress, including but not limited to exercise, fasting, heat, cold, medication, and radiation. It not only tolerates those interventions, it supports them, sometimes potentiating their benefits, sometimes buffering their costs, and often both in succession. H_2 does this without the narrow therapeutic windows that most interventions demand, without known toxicity, and without needing pristine health to be effective.

Whether you're testing your physical limits, recovering from an injury or illness, dealing with chronic dysfunction, or simply aiming to age with more strength and clarity, hydrogen seems to adapt to the state you're in. It doesn't push your system in one fixed direction. Instead, it scales its effects, meeting you where you are and supporting the specific kind of balance your body needs at that moment.

It's not a cure, but it is a consistent, evolutionary input—a molecular rhythm modern life has largely erased. Reintroducing that rhythm, in water or gas form, may be one of the most sane, safe, and strategic moves you can make for long-term resilience. This isn't hype; it's just the kind of quiet signal your body has always known what to do with.

CHAPTER 9:

Stacking Hormetic Stressors

When it comes to hormetic agents, and especially stacking hormetic agents, the religion of double-blind RCTs, which holds that clinical evidence can only come from double-blinded, controlled studies, falls apart. Don't get me wrong, double-blind RCTs are the gold standard for *most* therapies. Hormesis is just different, and it's something we cannot trust average responses for any individual case. For hormesis, the ideal stress exposure varies between individuals and within those individuals, changing from day to day. The differences in tolerance, the threshold at which a therapy becomes effective, and the point at which it becomes harmful, cannot be predicted using population-wide data or large groups.

Picture 22. I don't look half bad when not getting hit in the face.

One's ideal stress load for positive adaptation will change every day, for every person, as a result of one's cumulative stress, baseline health, and the stressors you have stacked. Before I enrage too many researchers in the field, I need to state that double-blind RCTs aren't completely useless in helping guide you through hormetic protocols, and until we are actually able to utilize real-time wearable and biometric monitoring for personalized guidance reliably they are the best tool at our disposal; they just shouldn't be viewed as gospel. Consider them to be more like a guiding light to help you reach the starting line. Once you've arrived, it's up to you to scale up or down to match what works best for your own physiology. Additionally, there are several ways in which RCTs could be improved specifically for hormesis, but that is a topic for another discussion.

The future of hormesis is in monitoring, wearable tech, and personalized protocols that adapt based on real-time data. We aren't there yet, so the best advice sounds like "bro science": namely, *you need to listen to your body*. Where I differ from bro science advice is that I will not tell you to push past the pain and toughen up. For every individual aspiring to be strong, their duty is to learn how far they can push and to understand when pushing farther sets them back, rather than springboarding them forward.

Currently, some wearable technology can be beneficial. The Oura ring, for example, monitors your apparent stress state (I speak about the Oura ring in detail in Chapter 12). The caveat being, we do not know how reliable it is, and the monitoring has a

Pictures 23-24. Crossblocking out of necessity due to one functional arm. Sometimes it works, sometimes I bite on a kick feint and eat a shot behind the ear. Them's the breaks.

delay, which is not particularly useful for real-time adaptations. That said, some meaningful data can be tracked. For instance, I've noticed that certain days when I pushed hormesis too far, my stress state spiked and didn't go back down, at least not fully. I remained in a sympathetic state for a long time. Other times, a smaller spike led to a stress decline into the restoration phase, indicating I entered a parasympathetic state. I will say that in these last few weeks with a colicky baby, times when I am assisting with childcare and my daughter is inconsolable, especially during the "witching hours" in the evening, the Oura is tracking substantial stress spikes. I have auditory hypersensitivity, and loud, chaotic, or cacophonic noises cause me physical distress and disorientation.

This is one of the reasons I used to drink in social settings, previously, as the alcohol abated this stress and accompanying physical pain (I spoke about this in detail in Chapter 7). These spikes, namely their duration and intensity, are perfectly correlated to my sleep quality; during this time, my resting HR has been elevated, my HRV has plummeted, and I am experiencing a substantial reduction in deep sleep, as low as ⅓ of the total deep sleep time as my previous norm. This gives me faith that wearable techs are not that far off from giving us real-time data.

When testing hormetic protocols, and especially stacking hormetic stressors, it's best practice to keep a journal. Mark down how you feel—not on a scale made for generalizations, but how you truly feel as compared to your baseline, or your best. By recording heart rate, HRV, body temperature, and sleep quality, specifically duration, total REM, and total deep sleep, you can ascertain your level of recovery. Wearable technologies can currently accomplish this.

Then, experiment and track trends in your own data. After a handful of sessions of lengthening one hormetic stress, how did your body respond? Did it improve your subjective performance and measurable data on days you felt great, and/or harm you on days you didn't? Over time, you can develop a finely tuned intuition about what your body can handle and what will lead to heightened stress and diminished recovery. In time, technologies will improve, and our guesswork will be taken out of the equation.

Now, since we've gotten the uncomfortable part out of the way—namely, my verbose explanation of everything we don't know—it's time to give some general advice on what is known, or I can hypothesize on. Namely, answering the question, with my conjecture, on which hormetic agents stack the best with each other.

Improving Exercise Performance

Stacking hormetic stressors around training can amplify both performance and recovery, but timing and sequencing matter. Molecular hydrogen taken pre-exercise transiently amplifies the mitochondrial stress response—potentiating ROS signaling that drives adaptation—while accelerating the rebound to homeostasis, leading to

stronger gains without prolonged damage (Chaoqun et al., 2021). It has also been shown to aid recovery post-exercise, likely through its role in modulating inflammation and restoring redox balance (Nogueira et al., 2018). In both untrained individuals and elite athletes, hydrogen improves fatigue resistance and supports better chronic redox modulation, with multiple clinical trials confirming improved endurance, lower lactate buildup, and enhanced metabolic efficiency over time (Mikami et al., 2019; Ara et al., 2018).

Heat exposure before training increases blood plasma volume and vasodilation, which improves oxygen delivery and amplifies the physiological stress signal induced by exercise, strengthening the adaptive response (Mee et al., 2018). On top of this, heat exposure before training may help reduce long-duration fatigue and support recovery. Studies found that heat exposure prior to exercise increased heat shock proteins that enhance stress resistance in muscle cells, helped muscles recover better after tiring, and lead to significantly reduced post-exercise fatigue (Touchberry, 2012; Iguchi & Shields, 2011). However, it's important to note that pre-exercise heat exposure is not without risk, particularly in hot or humid environments, or for individuals with cardiovascular conditions, where it may raise the likelihood of dehydration, heat exhaustion, or impaired performance. Adequate hydration and environmental awareness are critical when using heat as a pre-conditioning tool (Périard, Eijsvogels, & Daanen, 2021).

Over time, repeated post-exercise heat exposure, such as sauna bathing, can expand plasma volume, enhancing cardiovascular efficiency and endurance by improving oxygen delivery to working muscles (Lee et al., 2022; Scoon et al., 2007). When applied after training, heat therapy can enhance recovery and support muscle adaptation by elevating muscle temperature and blood flow, activating key molecular pathways, and upregulating heat shock proteins to drive protein synthesis and repair (Kim, 2020 et al., McGorm et al., 2018; Méline et al., 2021). It also improves mitochondrial function, leading to faster recovery of muscle strength, reduced soreness and supports glycogen resynthesis to replenish energy stores after eccentric exercise (Kim, 2020). Passive heating can have an influence on muscles by increasing heat shock proteins and activating special muscle cells called satellite cells, which multiply and assist in the repair and growth of muscle fibers (Rodrigues et al.,, 2020). Overall, these effects create a healthy environment that promotes muscle growth. However, most of these benefits have been observed after short to medium periods of heating, and more research is needed to determine the best ways to use this method for long-term muscle development. Repeated use alongside training has demonstrated additive effects on cardiovascular health and metabolic regulation, enhancing performance and recovery beyond what either can achieve alone (Lee et al., 2022; Scoon et al., 2007).

Oxygen manipulation through intermittent hypoxia training adds another layer—upregulating HIF-1α and promoting mitochondrial efficiency, angiogenesis, and

erythropoiesis (Li et al., 2020). Hypoxia has also been shown to increase fat oxidation during submaximal workloads, reinforcing its value for long-duration endurance and metabolic efficiency (Kelly & Basset, 2017). Cold exposure, used strategically before endurance efforts, can reduce core body temperature and delay thermal fatigue, but cold during or immediately after resistance training should be avoided unless recovery is the sole objective, as it may blunt the inflammatory signaling needed for adaptation.

Red light therapy introduces a timing-sensitive variable: when applied pre-exercise, it has been shown to improve muscular performance, increase strength gains, and enhance endurance in both trained and untrained individuals. When applied after training, it supports muscle recovery by reducing oxidative damage, downregulating inflammatory signaling, and accelerating tissue repair. Red light therapy has demonstrated reductions in exercise-induced creatine kinase elevation, pointing to tangible protection against muscle damage.

Fasting / Calorie Restriction for Metabolic Health or Weight Loss

Molecular hydrogen complements fasting and caloric restriction by modulating oxidative stress and inflammation without suppressing the adaptive stress signals these interventions rely on. Rather than acting as a direct antioxidant, hydrogen selectively regulates redox balance and inflammatory tone, supporting mitochondrial efficiency and metabolic flexibility during caloric deficit. Clinical trials have shown

Pictures 25-26. I can really only get away with shifting stance mid punch in a blitz once per session. This time I waited until the last 15 seconds of the last round. The motivator was definitely that I saw the photo of my face being squished the round before.

Picture 27. My fiancée won't like seeing this one. It hasn't been that long since I got my nose fixed from all the previous breaks (no break here, thankfully).

improvements in insulin sensitivity, appetite hormone regulation, lipid metabolism, and central neurometabolites following high-dose hydrogen water intake, suggesting broad support for both fasting physiology and weight loss efforts. It has also demonstrated potential to preserve ghrelin dynamics and upregulate GLP-1, both of which play central roles in hunger signaling and glucose homeostasis. From an evolutionary standpoint, intermittent caloric restriction and microbial fermentation likely co-occurred, with increased endogenous hydrogen acting as an internal signal of environmental scarcity. Reintroducing exogenous hydrogen during modern fasting may restore part of this lost physiological rhythm—mimicking the ancestral conditions under which our metabolism evolved. This idea is reinforced by preclinical findings showing that hydrogen-rich water extended the survival of fasting Drosophila, suggesting hydrogen may act as an adaptive buffer that improves resilience under caloric stress.

Heat exposure further amplifies the metabolic benefits of fasting and caloric restriction. Preclinical and clinical studies have shown that sauna use improves insulin sensitivity, increases expression of heat shock proteins, and supports cardiovascular function—all of which reinforce fasting-induced benefits. When used during caloric deficit, heat stress may potentiate fat oxidation and enhance nutrient partitioning through mitochondrial adaptations and hormonal shifts, including increased adiponectin and improved endothelial function. Repeated exposure can strengthen the adaptive response by compounding the effects of metabolic stress in a controlled and recoverable way.

Short bouts of fasted exercise further intensify the hormetic signal, increasing fat oxidation, AMPK activation, and mitochondrial biogenesis. This combination—caloric restriction, fasted training, and supportive stressors like hydrogen and heat—engages multiple metabolic pathways without triggering excessive cortisol or systemic fatigue, provided it is appropriately dosed.

Red light therapy may offer additional support during caloric restriction by enhancing mitochondrial ATP production and maintaining energy availability in a low-calorie state. Some studies have demonstrated localized fat reduction and improved metabolic parameters, and rodent data suggest improved glucose regulation and reduced adiposity under metabolic stress. While not a primary driver of systemic weight loss, RLT may serve as a supportive modality for preserving metabolic function and promoting tissue resilience during extended calorie restriction.

Cold Exposure

Molecular hydrogen may assist in navigating cold stress by modulating inflammatory tone, redox status, and thermoregulatory control. In rodent models of systemic inflammation, hydrogen inhalation prevented fever during mild stress and potentiated hypothermia under severe conditions, alongside suppression of TNF-α, IL-6, IL-1ß, and hypothalamic prostaglandin E_2. These findings suggest

Pictures 28. One of the closest I have ever been to getting dropped. Knees buckled, the shot wasn't even that hard. Dialing up the intensity in sparring at 40 years old may not have been advisable.

Picture 29. Pretty sure this was a miss.

that hydrogen alters thermal regulation under physiological strain and may enhance tolerance to cold in compromised states by preserving autonomic control and mitochondrial function. In regenerative models, hydrogen has also demonstrated synergistic effects when paired with cold atmospheric plasma—a redox-intensive therapy—by promoting stem cell survival through Nrf2 activation and mitochondrial stabilization. These data support hydrogen's role in buffering cold-mimetic oxidative stress without suppressing adaptive signaling.

Alternating cold with heat amplifies both stress signals and recovery dynamics. The vascular rebound from constriction to dilation increases endothelial function, improves perfusion, and accelerates clearance of metabolic byproducts. Repeated exposure to cold-heat cycles—whether through sauna and plunge, or cryo and infrared—enhances mitochondrial remodeling and broadens the adaptive window while minimizing residual strain.

Light physical activity before and after cold exposure helps prime thermogenic systems and reintroduce blood flow post-immersion without blunting the hormetic signal. Breathwork, particularly intermittent hypoxia or CO_2 tolerance training, reinforces autonomic activation and supports metabolic flexibility. Red light therapy applied after cold may further aid tissue perfusion and ATP regeneration while preserving the adaptive stress.

Cold tolerance is highly state-dependent. Hunger, low glycogen, and sleep deprivation reduce mitochondrial output and thermogenic capacity, sharply narrowing the adaptive window. Under these conditions, cold exposure becomes destabilizing rather than conditioning. These variables must be accounted for when stacking cold with fasting, exercise, or caloric restriction, as overreaching in a depleted state shifts the outcome from hormesis to harm.

Cold exposure works best when integrated into a broader system—hydrogen to refine the stress, heat to expand the envelope, breath, and movement to stabilize the return to baseline. Like all stressors in this framework, its effect depends not on how hard you push, but how precisely you calibrate.

Heat Exposure

Alternating heat with cold amplifies both the stress signal and the recovery window. The rapid shift between vasoconstriction and vasodilation improves vascular flexibility, enhances mitochondrial signaling, and accelerates the clearance of metabolic byproducts. This stacking not only deepens the hormetic effect, but also reduces residual fatigue when timed correctly.

Infrared therapy and red light therapy (RLT) extend the benefits of heat exposure by stimulating mitochondrial function without requiring high external temperatures. Near-infrared wavelengths activate cytochrome c oxidase, increasing ATP production, improving local perfusion, and upregulating antioxidant defenses. These effects can enhance recovery when used post-exercise and may support metabolic adaptation when used alongside conventional heat exposure.

Molecular hydrogen has shown protective effects against heat stress in multiple biological systems. In rodent models of heat stroke, inhalation of 2% hydrogen improved survival and reduced shedding of the vascular endothelial glycocalyx by attenuating oxidative stress and inflammatory cytokines. In mice, hydrogen-rich water mitigated behavioral and intestinal damage from heat stress, improving redox balance and modulating gut microbiota—effects enhanced further when combined with tea polyphenols. Even in marine ecosystems, hydrogen preserved photosynthetic efficiency in heat-stressed *Acropora* corals, suggesting a conserved cellular protective effect under thermal load. These findings support hydrogen's role as a regulatory buffer during heat stress, preserving redox control and inflammatory balance without blocking the adaptive signal.

Light exercise, movement, or stretching during heat exposure can increase benefits beyond passive sauna use. Dynamic movement under heat improves tissue pliability, circulation, and mobility, and may further stimulate the release of heat shock proteins. When applied consistently, this approach can support joint health, flexibility, and neuromuscular coordination, especially when integrated as part of a recovery or mobility-focused session.

Ethanol

If you're going to drink, and you care about minimizing damage, molecular hydrogen should be considered one of the most promising interventions available in this context. In human and animal studies, hydrogen has been shown to reduce blood alcohol concentration, improve subjective hangover symptoms, protect the liver from ethanol-induced oxidative damage, and modulate both alcohol dehydrogenase and aldehyde dehydrogenase activity (Lv et al., 2022; Yano et al., 2021; Lin et al., 2017). It also helps preserve gut barrier integrity and mitigates inflammatory cytokine surges that typically follow alcohol intake. The breadth of hydrogen's protective effect spans hepatic, neurological, and gastrointestinal systems, making it one of

Picture 30. These days my Philly shell degenerates into my right hand hanging fully below the waist by the end of the first round. Not doing myself any favors.

the few interventions that supports multiple axes disrupted by ethanol (Lin et al., 2022; Peng et al., 2022; Yano et al., 2021; Lin et al., 2017).

Exercise prior to drinking may offer additional benefit by improving hepatic blood flow, raising glycogen stores, and accelerating alcohol clearance. Aerobic activity before alcohol consumption has been shown to lower peak blood alcohol levels and increase metabolic processing, likely through upregulation of detox enzymes. However, intense training immediately before heavy drinking is ill-advised, as systemic fatigue may amplify alcohol's negative effects.

Fasting before drinking is strongly discouraged. Ethanol is absorbed more rapidly in a fasted state, leading to sharper intoxication curves, higher acetaldehyde buildup, and increased oxidative stress. Post-drinking fasting is also risky: most individuals experience glucose dysregulation and transient insulin resistance the next day. Adding fasting on top of this often results in dizziness, brain fog, irritability, or in some cases, fainting. Unless alcohol intake was minimal and recovery has clearly begun, fasting is more likely to delay recovery than enhance it.

Cold exposure prior to drinking may offer some benefit if used moderately and not immediately before consumption. Short-duration cold can enhance metabolic tone and norepinephrine release, particularly when followed by a nutrient-rich meal. However, prolonged cold exposure depletes energy reserves and may reduce alcohol tolerance. Heat exposure prior to drinking is not advised, as it increases dehydration and vasodilation, both of which compound alcohol's cardiovascular strain and amplify intoxication effects.

The next day, light aerobic movement can aid recovery by supporting hepatic clearance and improving insulin sensitivity. Red light therapy may stabilize mitochondrial output and reduce neuroinflammation. Heat exposure, when used after hydration has been restored, may assist vascular rebound and detoxification. Cold exposure during this phase should be avoided, as ethanol impairs thermoregulation and may worsen vasodilation and rebound hypothermia.

Recovery after alcohol requires system-wide stabilization: restoring hydration, resolving glucose instability, reengaging circulation, and minimizing oxidative

stress. No single intervention resolves all of it. But when hydrogen is paired with movement, light, and properly timed recovery tools—while avoiding compounding stressors like fasting or abrupt cold—it's possible to blunt a large portion of the physiological fallout. If you're going to drink, this is how you limit the damage. Not by pretending it's harmless, but by responding to it with precision.

Oxygen Manipulation

Molecular hydrogen can be stacked with both hypoxia and hyperoxia to buffer oxidative stress and preserve mitochondrial function. In hypoxic conditions, hydrogen has been shown to reduce reperfusion injury, stabilize redox tone, and protect cellular structures without interfering with the adaptive signaling pathways triggered by low oxygen. In hyperoxic environments, including therapeutic oxygen exposure and HBOT, hydrogen has been shown to reduce tissue damage, suppress apoptosis, and improve organ function in both pulmonary and systemic models.

Exercise is the only other intervention supported for stacking in both oxygen extremes. Under hypoxia, aerobic and endurance-based training increases mitochondrial biogenesis, stimulates angiogenesis, and upregulates HIF-1α, producing well-characterized improvements in endurance capacity and oxygen efficiency. Hydrogen may help extend the adaptive window by reducing oxidative damage during repeated or prolonged exposure. Under hyperoxia, exercise has been shown to increase peak power output, endurance time, and training load, particularly when paired with high-concentration oxygen breathing during interval and sprint protocols.

No other stressors discussed in this framework—such as heat, cold, fasting, or light therapy—have demonstrated benefit when stacked with hypoxia or hyperoxia. Their use in proximity to oxygen manipulation may compound systemic stress and should be avoided unless specifically supported. In both extremes, the effective pairings remain narrow and well-defined. Stack accordingly.

Picture 31. At least he looks tired, too.

Universal Stackers

Hydrogen can be paired with every stressor in this chapter without interfering with the intended adaptation. It doesn't suppress the signal—it modulates it. When the stress is appropriate, hydrogen supports and potentiates the response. When the stress exceeds the threshold for adaptation, it blunts the excess before it causes damage. This dual role makes hydrogen unique. It preserves mitochondrial structure, maintains redox balance, and regulates inflammatory signaling without flattening the necessary stress-response cascade (Cheng et al., 2023). Whether used with heat, cold, fasting, hypoxia, hyperoxia, exercise, or radiation, hydrogen widens the adaptive window and reduces the cost of getting it wrong. No other intervention in this framework can be used universally, across all conditions, without the need for cycling, timing, or restriction.

Red light therapy may eventually belong in the same category. Mechanistically, it supports mitochondrial function, enhances tissue repair, and modulates inflammation across various contexts. But unlike hydrogen, the data are still narrow. Its potential is clear—the evidence isn't. Until that changes, it remains a targeted tool, not a universal one.

Picture 32. All smiles until I tried to stand up and walk to my door from the car an hour later. A dozen good photos, a dozen days of my back being out.

CHAPTER 10:

Out Of Sync: The Sleep Rhythm You Can't Cheat

Three days of broken sleep cost me three weeks of performance. This isn't an exaggeration.

After months of running on 5.5 to 6.5 hours of sleep, I felt sharp, stable, even strong, *but then came the "day" trip to China*: three consecutive nights of sub-3-hour sleep on either side of long-haul flights, departing on a Tuesday and home on a Friday, and everything collapsed. A single recovery night at 10 hours wasn't enough. The damage had already been done: I dragged through the following week, and the next. My brain slowed and my metrics tanked. It would take three weeks to return to my baseline.

This acute sleep deprivation stress was telling—what was less telling, but substantially more impactful, was the years of sleep deficiency I subjected myself to while launching my hydrogen tablet business. For the first 2-3 years, as I obsessively worked towards my goals, I routinely slept just 4-5 hours per night. The sleep I did receive was scattered; my brain would awake, racing with ideas, and I'd need to write them down quickly before returning to slumber. Additionally, the alcohol I was drowning myself in pre-sleep, even though it usually failed to bring me to significant intoxication, was undoubtedly interfering with the quality of sleep, during the limited sleep I was getting. It is impossible to prove how much of my deteriorating metabolic health was caused by sleep deprivation, as there were numerous other confounding variables, but it was likely a substantial driver of my prolonged health decline.

The inverse was no better: during a period of illness and professional pause, talked about at the start of this book, I slept 16+ hours a day. My strength held somewhat in the gym, but my reactive ability vanished. Mentally, I was a shadow of myself, barely functional, moving through fog, with brief windows of clarity that would crash again within hours. I wasn't working, I wasn't reading, I was just trying to survive.

Oversleeping didn't help me. On the contrary, it wrecked the few usable hours I had. I learned the hard way, at my lowest point, that you can't fix exhaustion by flooding the system with sleep: it simply doesn't work that way. Recovery, like stress, needs to be in the right dose. More isn't better, we need harmony between the two. When this harmony is disrupted, either from insufficient recovery, or an abundance of it, our health deteriorates—both physically and mentally. Sleep debt, and the subsequent fatigue and exhaustion, can't be paid back. Your body has taken a hit, and the only road back is slow, mindful and intentional recalibration. There are no quick fixes, but there are simple solutions.

Picture 33. Milan, Italy

Champagne in my hand, not hers. Post pregnancy test—we didn't consider the challenge of it being in Italian, with a code to read the Italian language print. Really took all the surprise out of the moment.

Why Sleep is Critical

It's cliché by now to state that we spend roughly one third of our lives sleeping; however, for many this fact has not meaningfully affected decision making where proper sleep prioritization is concerned. The science is clear, and lacking controversy or debate for one statement: we know getting sufficient sleep, of sufficient quality, is critical to good health. Poor sleep quality, or insufficient quantity, leads to increased risk for almost all diseases and deleterious health consequences, not the least of which is an increase in all-cause mortality.

Sleep disturbances, particularly poor sleep continuity and insomnia, play an important role in many mental disorders like depression and PTSD (Baglioni et al., 2016). Non-depressed people with insomnia have twice the risk of developing depression compared to those without sleep difficulties (Baglioni et al., 2011). If these sleep issues are identified and treated early, it could interrupt the progression of mental health problems and improve overall outcomes (Baglioni et al., 2016). Poor sleep also robs us of quality of life prior to a potential untimely death, with the research being clear that it leads to decreased productivity and reduced emotional well-being. Integrating sleep interventions into clinical care is crucial for enhancing overall mental and physical health (Badri et al., 2023; Blackwelder, Hoskins, & Huber, 2021; Baglioni et al., 2016; Institute of Medicine, 2006). Despite the clear, and substantial, consequences of sleep deprivation, prioritization is often limited to small sections of society, such as biohackers, longevity enthusiasts, and high performers looking to optimize. Even then, many in these groups give inadequate attention to this foundational task, opting for high-cost flashy devices, supplements, therapies, and experimental drugs, instead. So why has sleep been so widely ignored? Why do we have biometrics on every step we take and calorie we burn, but only now are we starting to measure what happens when our eyes are closed? Answering these questions is the first step to change, so by necessity, it is where the chapter begins.

Awareness

Consequences of poor sleep result in a myriad of physical and mental health issues affecting a shockingly large percentage of the population. According to the National Institutes of Health (NIH), nearly 40% of adults in the United States report issues related to sleep deprivation, such as unintended daytime sleep, and up to 70 million Americans have a chronic sleep disorder (National Heart, Lung, & Blood Institute, 2022). These statistics are staggering, rivaling obesity and metabolic syndrome, making it almost unbelievable that we are not more aware of the problem.

Both Donn Posner, Ph.D., clinical associate professor of psychiatry and behavioral sciences at the Stanford University Medical Center, and Matthew Walker, Ph.D., professor of neuroscience and psychology at UC Berkeley, have stated something that sounds plainly absurd (and somewhat concerning): the average Medical Doctor receives *just 30 minutes of schooling on sleep*. This is such a low number that it

seems improbable two highly respected professors would make it up, although I have not been able to verify it. The papers I have read suggest it is closer to the ~3 hour mark, however, some schools lack any time dedicated to sleep education (Romiszewski et al., 2019; Mindell et al., 2011).

Both Posner and Walker have also commented that if the deleterious effects of lack of sleep were instead reframed as the consequences of something like diabetes, with the number of afflicted being the same, we would be in full panic at the epidemic. In my estimation, it is only since roughly 2019 that sleep hygiene and quality have emerged as a trend in health circles, and as I mentioned previously, it is still woefully undervalued.

Bravado

We've all heard the anecdotes of the revolutionary geniuses or high-powered CEOs who seem to require little sleep. While it is undoubtedly true that different people require different amounts of sleep to function effectively, and each person will have different daily sleep requirements based on the totality of stress of each given day, the cultural framing that minimizing the sleeping hours is a talent or special ability has had considerable, and negative, impacts on society. We are taught that depriving ourselves of this fundamental need leads to more hours for productivity, completely ignoring deviations in effectiveness and competence observed through changes in sleep quality and duration.

As it is likely clear from the Introduction, I have struggled with sleep issues throughout my life. No matter how hard I try, and how much effort I put in, I cannot sleep 8 hours a night for days on end. I will just lie in bed, awake and restless, and the over-rest makes me sluggish and lazy. I seem to operate best on between 5.5-7 hours of sleep per night, depending on how active I am and how much sleep I have logged in previous nights. Moreover, I have altered my routine so that I almost never require an alarm; I wake up when my body wants to wake up, ensuring that each day I feel rested and refreshed.

To ensure that my sleep metrics, particularly the amount of REM and Deep sleep I experience, remain adequate, and preferably ideal, I've been tracking my sleep with an Oura ring for many years. On days I naturally wake up after ~5.5 hours, my "readiness" remains acceptable, even desirable.

Even though I am "blessed" with needing less sleep than most others, able to operate for days on end with much less than 5.5 hours a night, that wasn't "good enough" for me in my younger years. Well into my 30s, I continued to treat sleep as an annoying afterthought. I fantasized about the day pharmaceuticals would allow me to be awake 24 hours a day, envisioning a reality where a drug gave me unfettered productivity with no serious consequences: the freedom to learn, accomplish, and live without the shackles of daily unconsciousness. During these days I would

typically force myself up to start working, writing emails, and writing notes the second I rose from a light sleep, sometimes in the middle of the night.

When friends, family, and colleagues would ask if I ever slept, or needed to, I would joke that "sleep is for the weak." This bravado is not unique to me and seems to be quite prevalent in many industries and professions. ER doctors and residents, for instance, may be expected to get 2 hours or less of sleep a night, by way of short naps. As I have aged, in wisdom and years, I have come to terms with the delusional nature of my fantasy. In life, there will always be trade-offs: sleep, and our need for it, is imprinted deep into our biology. Perhaps one day this riddle will be solved, but it is almost certainly not going to be in our lifetime. We have barely scratched the surface of understanding why we sleep, so hoping to replace it is a fool's errand.

Why We Sleep

It's a common misperception that sleep is a time when our mind and body shut down. In reality, sleep is an active period where many necessary physiological functions take place. During sleep, our body works towards restoration, strengthening, and completing important processing (Brinkman, Reddy, & Sharma, 2023). While we do not yet know exactly why we need so much sleep, and why it is so important, we know a couple of key points: it absolutely has a function, and virtually all species have evolved to require extraordinary amounts of time in slumber.

One important role of sleep is to store and sort through our memories. Throughout the day, our brains are constantly absorbing vast amounts of information. However, instead of being instantly stored, this input goes through a series of processing steps, many of which occur during sleep. During the night, fragments of short-term memories are reorganized and reinforced into more stable long-term storage in a process known as memory consolidation. Studies show that sleep improves memory retention and enhances performance on recall tasks. In addition to cognitive benefits, sleep is also critical for physical restoration, including muscle growth, tissue repair, and hormone production (Chennaoui, Léger, & Gomez-Merino, 2020; Elkhenany et al., 2018). For example, growth hormone, which stimulates the proliferation of stem cells, is primarily released during deep sleep. Melatonin, another hormone that increases when it gets dark, protects our stem cells and can inhibit adipogenesis, which is the process of fat cell formation. That's why good and enough sleep is essential for keeping our bodies healthy and able to recover from damage (Elkhenany et al., 2018).

One leading theory on why we sleep relates to energy conservation. During sleep, the body regulates and restores hormone levels and reduces metabolic activity, which may help conserve energy and maintain physiological balance (Jung et al., 2011; Sharma & Karuva, 2010). Another theory on why we sleep is that sleep facilitates the removal of toxic metabolic waste from the brain. During deep sleep, the

glymphatic system becomes more active: the fluid-filled channels between brain cells expand, which allows cerebrospinal fluid to flow more freely through the brain. This increased flow helps flush out toxic metabolic waste products, such as ß-amyloid proteins linked to neurological diseases such as Alzheimer's (Underwood, 2013). In this way, you can think of the glymphatic system as an overnight cleaning crew that comes into a facility and ensures working conditions are pristine when the staff arrive in the morning. Tasked with flushing out metabolic waste from the central nervous system, the glymphatic system's relevance to our long-term mental vitality is critical. Deep, or slow wave sleep, particularly REM, activates this system most effectively (the glymphatic system is also active during REM sleep, but does its heavy lifting during deep non-REM). Impaired in function, metabolic waste piles up, and cognitive decline becomes more and more likely (Mendelsohn & Larrick, 2013).

A UC Berkeley study found that people who report declining sleep quality in their 40s and 50s have *higher beta-amyloid and tau loads* later in life (Winer et al., 2019). These proteins don't always trigger dementia—but the correlation is real and strong.

In a study published in *Nature Communications* in 2019, researchers studied DNA repair mechanisms within the chromosomes of zebrafish embryos by genetically engineering them to have fluorescent chromosomes inside their neurons (Zada et al., 2019). Researchers noted that double-strand breaks were static during waking hours, but when asleep, DNA damage began dissipating. Further, by manipulating the zebrafish to keep them awake (such as tapping on the glass), the rate of DNA damage increased. The researchers suggested that, although sleep reduces environmental awareness and responsiveness, it is essential across species; from jellyfish and zebrafish to humans, because it enables neurons to carry out critical DNA repair processes. This function may explain why sleep is such a widespread and evolutionarily conserved behavior in animals (Zada et al., 2019). In every living cell, DNA can develop tiny breaks due to both internal processes and external factors, such as UV light (as discussed in Chapter 5). If these breaks aren't properly repaired, they can lead to mutations or even cause the cell to die. Importantly, DNA damage plays a key role in aging and age-related diseases, including cancer (Clarke & Mostoslavsky, 2022).

The Architecture of Sleep

To understand how sleep supports recovery and resilience, you need to understand what actually happens *during* sleep. It's not one continuous state—it's a progression through distinct phases, each with its own physiological purpose. If you shortchange one, the whole system suffers.

Stage 1 – Light Non-REM Sleep

This is the entry point. It lasts only 5–10 minutes and is easy to wake from. Personally, one of my biggest issues with Stage 1 is something called a hypnic

jerk—a sudden "falling" sensation that snaps me awake, heart racing, just as I'm drifting off. It's common, but frustrating. If the jerk doesn't wake you, your heart rate and breathing begin to slow as your body transitions inward (Summer, 2025; Ogilvie, 2001).

Stage 2 – Light Non-REM Sleep (Prep Phase)

This stage deepens the descent. Breathing slows further, heart rate continues to drop, and your muscles relax. Your brain begins producing sleep spindles, which are short bursts of activity that help block out external noise and aid in memory consolidation (Schönauer, 2018). Stage 2 sets the stage for the recovery that follows.

Stage 3 – Deep Non-REM Sleep

This is the phase of sleep that allows some people—like my fiancée—to remain oblivious to chaos; dogs barking, gunshots ringing, you name it. For some, deep sleep is almost absolute in its sedation. We likely evolved to allow this, as deep sleep is the kind of sleep your body doesn't just benefit from, it *needs* it. Heart rate and breathing reach their slowest rhythms here, and brainwaves drop into delta territory: slow, wide, and deep. This is when the body goes into full repair mode. Muscles are rebuilt, and tissues are patched up. The immune system retools itself for another day of microbial battle, while your body recalibrates hormonal levels.

As you get older, you don't just lose time in deep sleep, you lose efficiency getting into it. The process gets fragmented, but that doesn't mean it's less important. If anything, it becomes more vital. Especially if you're breaking yourself down on purpose—through training, fasting, stress exposure—this is where the dividends get paid. This is where hormesis cashes out (Uygun & Basheer, 2022).

Stage 4 – REM Sleep

REM begins about 90 minutes into your cycle. The first round might last 10 minutes, but each successive REM phase gets longer, with the final one lasting up to an hour. REM is when your brain lights up, your dreams intensify, and your heart rate and breathing speed back up. This is the domain of procedural memory: the kind that encodes how to do things, not just memorize them. This is essential for skill development, learning, creativity, and emotional regulation for this reason (Fogel et al., 2015).

A study showed that uninterrupted REM sleep is critical for amygdala reactivity, meaning your brain's ability to emotionally adapt and regulate stress is tied directly to REM quality (Wassing et al., 2019). This has massive implications for trauma recovery, mood regulation, and psychological resilience. As with deep sleep, REM also tends to decline with age—but again, that doesn't mean it's unimportant. If anything, it becomes even more critical.

Chronotypes: The Sleep Clock You're Born With

> *I'm not an early bird or a night owl. I'm some form of permanently exhausted pigeon.*

The first time I saw that meme, I laughed out loud. It was funny, but also a little too real. I've had several friends share this meme with me over the years, and when I've brought it up to others, many have commented they've seen it. The fact that it resonated so widely says something important: *we've normalized exhaustion*. We joke about it, meme about it, but at some point we have to stop laughing and start listening. Because if you're constantly in a fog, you're not just tired, you're misaligned.

Here's the truth: being a "night owl" or an "early bird" isn't just a personality trait, it's biological. Each of us has a built-in chronotype, which is a genetically influenced preference for when we sleep and wake. It's governed by your circadian rhythm and, for most people, sits within a 2–3 hour window. Some people are naturally wired to go to bed and rise early, whereas others function best when their sleep schedule is later (May, Hasher, & Healey, 2023). Neither is wrong, but when your internal clock clashes with your external obligations—school, work, social commitments—you start to break down. If the mismatch is severe enough, it can manifest as a circadian rhythm sleep disorder, where your ability to participate in "normal" life erodes (Kim, Lee, & Duffy, 2013).

If your body is forced to perform when it's biologically meant to rest, your recovery suffers. Your mood tanks, and your hormone regulation, immune function, and cognitive sharpness all take a hit. Ideally, we would all structure our lives to work with our rhythm, not against it. If you have flexibility in your schedule, use it. Align your deep work, workouts, or demanding tasks with your natural peaks. That's not laziness, it's optimization.

For most of my life, I was a textbook night owl. My clearest thinking, my best work, it all clicked between 9 PM and midnight, long after most people had shut down for the day. That rhythm shaped everything from my training schedules to my caffeine intake, but something shifted recently: I've become an early riser. Not just tolerating mornings, but thriving in them. I suspect this change is tied to the peptide protocols I've been experimenting with. The shift wasn't forced, it emerged gradually. While I don't yet fully understand the mechanisms, the result has been consistent: I fall asleep earlier, wake earlier, and feel sharper doing it. It's a fundamental change in my sleep architecture I never thought possible.

My fiancée, by contrast, sleeps significantly more than I do, especially now as I write this, her being eight months into pregnancy. Even before that, she typically slept one to two hours more than I did. These days it's closer to three or four. She's naturally biphasic: she'll wake in the middle of the night, stay up for an hour or two, then fall

back asleep. It's a rhythm that works for her. Years of graveyard shift work did a number on her cycle, but we've gradually rebuilt a routine that supports her energy and recovery. She goes to bed when I do now, but stays in bed longer, and it seems to suit her.

Interestingly, what might seem like an unusual sleep pattern today, such as my fiancée waking for an hour or two in the night, was once the norm. Prior to the industrial revolution and widespread artificial lighting, most humans followed a biphasic or "segmented" sleep pattern: a first sleep shortly after nightfall, followed by a waking interval in the early hours, and then a second sleep until dawn (Ekirch, 2001). During that in-between time, people would pray, write, complete housework, have sex, even visit neighbors. It was a culturally sanctioned, neurologically natural rhythm: one that started to disappear only when factory schedules and electric lights compressed sleep into a single uninterrupted block. That historical context helped me reframe how I think about sleep architecture. What looks like insomnia through the lens of modern sleep hygiene might, in fact, be a residual biological rhythm trying to reassert itself.

That shift in perspective didn't just validate my fiancée's sleep patterns, it forced me to reconsider my own. The real takeaway is that sleep isn't a competition. There's no medal for the earliest riser or the deepest sleeper. Your system has to work *for you*. Whether you're monophasic or biphasic, someone who thrives on six hours or needs nine—what matters is alignment. Chronotype matters, recovery quality matters, and knowing when you function best, and living accordingly, matters. Looking back, I used to wonder how much of my early sleep dysfunction, and the inevitable caffeine spiral that followed, could've been avoided if I'd structured my obligations differently. If I'd honored my own rhythm instead of scheduling responsibilities into early mornings which I wasn't built for.

The truth is, that wasn't really the issue. I wasn't waking up early out of necessity—I was just being stupid, and creating an unnecessary routine I believed projected success, an image my younger self was desperate to portray. My caffeine addiction wasn't a product of overwork or obligations, it was recreational: *self-inflicted, and born of vanity*. Maybe once every week or two I had a real reason to be up early. The rest was just bad decisions, poor discipline, and a refusal to respect my own physiology. What I thought of at the time as discipline: forcing myself to wake up to an alarm, to be an "early riser," despite working evenings and into the night, was actually just dysfunction.

That's what made it worse: I *chose* the dysfunction, and it wrecked me, but hindsight, like sleep itself, only works if you *listen*. Want to know your chronotype? There's a simple questionnaire used in most sleep clinics and research settings. It won't give you a diagnosis, but it may give you clarity. As I argue throughout this book, clarity is the beginning of control.-

How Much Sleep Do You *Really* Need?

We know sleep needs change as we age. That part's obvious: newborns sleep almost all day, teenagers sleep like it's a survival mechanism, and older adults seem to need a little less. What's less obvious, though, and rarely discussed with nuance, is that sleep need is also highly individual**.** Yes, there are general guidelines. The National Sleep Foundation recently updated theirs, widening many of the recommended ranges:

- **Newborns (0–3 months):** 14–17 hours/day
- **Infants (4–11 months):** 12–15 hours
- **Toddlers (1–2 years):** 11–14 hours
- **Preschoolers (3–5 years):** 10–13 hours
- **School-age (6–13):** 9–11 hours
- **Teenagers (14–17):** 8–10 hours
- **Young Adults (18–25):** 7–9 hours *(new category)*
- **Adults (26–64):** 7–9 hours
- **Older Adults (65+):** 7–8 hours *(new category)*
  (National Sleep Foundation, 2020)

These ranges reflect biological averages, but you're not an average—you're a system—and no one can tell you exactly what you need without detailed feedback from your own body and mind. I've learned to trust my body and my mind upon waking; if I've only slept 5.5hrs but wake up full of energy and mind racing, I get up. If after 6.5hrs asleep I still feel groggy and exhausted, I close my eyes and go back to sleep. I purposefully schedule my first call availability of the day 2-3 hours after my standard time of waking to ensure I am never forcing myself to get up to an alarm. My body and mind are my alarm, and when they're ready to conquer the day I don't let governmental guidelines second guess them.

This is critical, as forcing yourself to sleep more when your body doesn't require it leads to issues not unlike sleep deprivation. Personally, when I have tried to force myself to sleep more, in order to fit a statistic that doesn't resonate with me, I feel tired and groggy. My performance, both mentally and physically, suffers. This can create a death spiral, as the less we do, the less capable we are of acting on anything. Forced rest can paradoxically create a situation in which we cannot force ourselves to do anything other than rest.

For those less in tune with their bodies, The National Sleep Foundation (2020) emphasizes a more pragmatic approach: instead of chasing a number, it's worth

paying attention to how your sleep shows up in real life. Do you feel alert and mentally sharp during the day? Can you get through your mornings without relying on caffeine? Is your body holding up under stress, or is it showing signs of fatigue and burnout? If you're frequently drowsy, even while driving or sitting still, that's not just a sign you need more sleep, it may be a signal that your current sleep pattern isn't doing what it's supposed to do. To reiterate, the goal isn't to hit some idealized benchmark, it's to make sure your sleep is serving you. If it's not supporting focus, recovery, and resilience, then it's not doing its job, and if it's not doing its job, your system can't adapt, and you can't do yours—by which I mean you will be rendered incapable of competently pursuing your goals, tasks, and purpose in this life.

The Damages of Sleep Deprivation

The science is no longer debatable: sleep deprivation kills. A systematic review and meta-analysis covering nearly 1.4 million participants found that short sleep duration increases all-cause mortality. Too much sleep also correlates with risk, but that's often a symptom of deeper dysfunction, such as chronic illness, depression, or metabolic imbalance, and not a cause (Cappuccio et al., 2010). Sleep deprivation undermines nearly every biological system we rely on for resilience. It sabotages performance, accelerates aging, and increases the likelihood of everything we're trying to prevent: disease, depression, burnout, and breakdown (Luyster et al., 2012).

Sleep and Mood: The Inflammation Link

Sleep plays a critical role not only in physical restoration but also in emotional regulation and social functioning. In 2018, a study with 18 human participants found that sleep loss increased social withdrawal and loneliness (Ben Simon & Walker, 2018). This is important because loneliness and a decreased sense of social purpose are very strongly tied to depression (Luo, 2023; Lee et al., 2021). Sleep loss has been linked to increased neuroinflammation, and the relationship between sleep disturbance and depression appears bidirectional: depression can lead to an inability to sleep, and poor sleep can exacerbate depression (Irwin & Opp, 2017). Inflammation likely mediates this link, as elevated neuroinflammation is both a consequence of depression and a risk factor for its development (Miller & Raison, 2016; Khandaker et al., 2014; Dantzer et al., 2008).

Beyond this, loss of sleep is well documented in raising inflammatory markers in otherwise healthy subjects (Mullington et al., 2010). An increase in systemic inflammatory markers, such as pro-inflammatory cytokines, can signal the brain and disrupt communication in reward circuits, leading to symptoms like loss of interest and pleasure. This disruption can decrease the availability of important brain chemicals such as serotonin and dopamine, which regulate mood, contributing to depressive symptoms (Miller & Raison, 2016; Khandaker et al., 2014). This shows how body-wide inflammation can influence brain function and lead to depressive

symptoms. The link between inflammation and depression has led to the use of anti-inflammatory drugs in the treatment of depression, with positive results being demonstrated in some patients and instances (Raison et al., 2013; Abbasi et al., 2012; Müller et al., 2006). Research teams experimenting with this treatment route have hypothesized that higher levels of inflammatory cytokines drive increased fatigue (Lee & Giuliani, 2019), which could further exacerbate sleep issues, and depression. If these links hold true, think of sleep deprivation and depression as a negative feedback loop powering a runaway train speeding down a hill with no brakes.

Sleep and Impairment: False Gains, Real Losses

One of the reasons why many high performers try to limit their sleep is this false belief that it will improve their personal, intellectual, or professional improvement and progress. I used to buy into this belief, maintaining the position that less sleep meant more time to learn, work, and grow. Then I dove into the research, and the evidence didn't validate my position, it undermined it, necessitating a rethink. One study found that being sleep deprived for 36 hours before learning reduces memory retention by 40% (Walker, 2009). Other studies show that moderate sleep deprivation impairs cognitive and motor skills to the same extent as being legally drunk (Williamson & Feyer, 2000; Centers for Disease Control and Prevention, n.d.).

Having previously been involved in breathalyzer fuel cell technology, I could write in length on the issues directly correlating to a particular blood alcohol concentration (BAC%), and how that relates to impairment. Blowing over the legal limit, usually 0.05%, doesn't necessarily mean you are drunk, however, for most people, most of the time, it does. As I wrote about in Chapter 7, I used to do some of my best writing and reading when registering a slight BAC%, blowing around 0.01-0.02%, however, once I exceed intoxication thresholds my thinking, writing, and information retention tend to rapidly decline. By subjecting yourself to deliberate sleep deprivation in the hopes of improving performance, it is potentially impairing the function of the most important aspects of what allows you to perform at a high level in the first place.

Sleep and the Body: Metabolic and Hormonal Breakdown

Sleep loss devastates more than just the mind, it also dismantles your metabolic health. One study found that just six days of sleeping 4-hours per night significantly disrupted glucose tolerance and spiked cortisol (Spiegel, Leproult, & Van Cauter, 1999). The shift is substantial enough to move an otherwise healthy individual into a temporary prediabetic state, increasing the risk of longer-term metabolic disease if sleep deprivation persists (Grandner et al., 2016). Poor sleep, both in duration and quality, not only affects metabolism directly but also promotes behavioral changes like increased calorie consumption and poor dietary choices, which together further elevate the risk of developing diabetes (Grandner et al., 2016). Short sleep is also

tightly linked to weight gain, impaired insulin sensitivity, and hormonal dysfunction (Knutson & Van Cauter, 2008). You don't just feel sluggish, your biology becomes uncalibrated.

Sleep also impacts testosterone, libido, and virility. Sleep also impacts testosterone, libido, and virility across both sexes, although the effects are more pronounced and better documented in males. While similar hormonal and sexual consequences of sleep disruption are also relevant to women, particularly during key reproductive phases, the existing evidence is less extensive and conclusive (Andersen et al., 2011). For both men and women, inadequate sleep is often the amplifier—turning manageable stress into systemic crash.

Lack of Sleep and Biological Risk Factors of Aging

Chronically elevated inflammation, which discussed above can be driven by sleep deprivation, is strongly linked to age-related conditions, and accelerated aging (Garbarino et al., 2021). Elevated oxidative stress is also strongly linked to sleep deprivation (Vaccaro et al., 2020), with elevated ROS also leading to disrupted sleep (Hill et al., 2018). This leads to yet another vicious pathophysiological negative feedback loop, amplifying the damage driven by both sleep deprivation and oxidative stress (Davinelli et al., 2024). In particular, sleep appears to play a dual role in managing oxidative stress: on one hand, it may protect cells from oxidative damage, while on the other, a minimal rise in ROS may be necessary to trigger the sleep process (Hill et al., 2018; Neculicioiu et al., 2023). The relationship between sleep deprivation, oxidative stress, and sleep regulation is likely bidirectional, where sleep loss increases oxidative stress and oxidative stress potentially impairing sleep, creating a vicious cycle (Hill et al. 2018; Neculicioiu et al., 2023; Vaccaro et al., 2020). However, the precise mechanism remains speculative, and variability in stress responses and experimental designs can obscure the true impact of ROS on sleep, underscoring the need for more rigorous, controlled studies.

Increased levels of oxidative stress, or dys-homeostasis of the redox status within our cells, is also considered one of the "pillars" of why we age, and develop conditions and diseases of aging (Leyane, Jere, & Houreld, 2022; Pomatto et al., 2018; Cui, Kong, & Zhang, 2012; Sohal & Orr, 2012). Telomeres,[8] likewise, are often discussed in relation to their role in aging, and age-related degenerative conditions, with sleep deprivation being linked to the shortening of telomeres (Jin et al., 2022; Jackowska et al., 2012). This is true even when researchers examine sleep deprivation in children (James et al., 2017). I remain skeptical of the long-

[8] Telomeres are protective caps of repetitive DNA sequences located at the ends of chromosomes. They prevent the loss of genetic information during cell division and protect chromosomes from deterioration or fusion with neighboring chromosomes. Over time, telomeres shorten with each cell division, and critically short telomeres can trigger cellular senescence or apoptosis (Blackburn & Epel, 2017).

term implications of these findings, as telomere length can rebound: acute stress in one year may accelerate shortening, only for subsequent behavioral changes to stabilize or even reverse the trend. This highlights the body's remarkable capacity for rebound and repair, particularly when stress leads to adaptive, hormetic responses rather than chronic breakdown (Burbano & Gilson, 2021; Schutte & Malouf, 2014). Further, a snapshot of telomere length at a given time has been shown to have no correlation with life expectancy, even in centenarians and those in advanced age, with the only markers statistically accurate in predicting mortality being those associated with elevated and chronic inflammation (Whittemore et al., 2019; Arai et al., 2015). However, one study found that among centenarians, those in relatively good health had significantly longer telomeres than their less healthy peers, suggesting that telomere length may reflect current functional status more than lifespan per se (Terry et al., 2008). In short, while static telomere length offers limited predictive value for individual mortality, especially in humans, future research may find greater prognostic utility in tracking telomere-shortening rate over time, though inflammatory markers currently remain the most reliable indicators.

As I have shown, sleep deprivation has also been shown to have negative consequences for testosterone in men (Andersen et al., 2011; Leproult & Van Cauter, 2011; Opstad & Aakvaag, 1982) and virility (e.g., male sexual function, libido, erectile capacity, and sperm production) (Liu et al., 2017; Jensen et al., 2013), although, so does stress (Mehta & Josephs, 2010; Daly et al., 2005), and it is not known if the negative consequences are long-term or can be abated with proper rest. What is clear, however, is that testosterone decline and loss of muscle mass are tightly linked to aging and mortality risk. If sleep deprivation reduces testosterone, even temporarily, it may accelerate this decline by contributing to earlier onset of sarcopenia, lower vitality, and reduced resilience. In this light, chronic poor sleep could act not just as a stressor, but as an amplifier of age-related degeneration.

The Gut Clock

One of the hottest topics in research is currently the effects of our microbiome on various conditions and overall health. Research suggests that our gut microbiome has its own circadian rhythm (Voigt et al., 2016). Disrupted sleep can impair microbial diversity and gut health, which potentially leads to a cascade of issues from metabolism to mood (Withrow et al., 2020; Voight et al., 2016). In fact, the U.S. Department of Defense awarded CU-Boulder $7.5 million to study the microbiome's interaction with sleep in mice, rats, and humans (Scott, 2016). Early results are hinting that better sleep = healthier gut flora, and possibly that targeted microbiome support could, in turn, improve sleep quality. The CU-Boulder team eventually tested a prebiotic fiber blend—galactooligosaccharides (GOS) and polydextrose (PDX)—on rats subjected to repeated sleep disruption. The results were striking: rats fed the prebiotic blend for four weeks didn't just weather the sleep disruption better, they logged more REM and NREM sleep

during the stress, and they rebounded with more total sleep during recovery (Thompson et al., 2020). These effects were linked to measurable changes in the gut microbiome, especially a rise in Parabacteroides distasonis, a bacterial species that strongly correlated with better sleep metrics (Thompson et al., 2021). In another study, the same prebiotic blend (GOS/PDX) was again shown to enhance both REM and NREM sleep during repeated sleep disruption and recovery, reinforcing its role in promoting resilience through microbiome shifts, especially the increased presence of Parabacteroides distasonis (Bowers et al., 2022).

While *P. distasonis* has been associated with beneficial effects such as immunomodulation, improved metabolic function, and circadian adaptation, its impact appears to be highly context-dependent; other studies link it to inflammatory responses and depressive-like behavior in disease models. Thus, its role in sleep resilience likely depends on host condition, strain specificity, and the surrounding microbial ecosystem, and warrants further mechanistic investigation. Since this is a prebiotic intervention, and not direct supplementation with this strain, it remains exceedingly unlikely these deleterious responses could occur from overgrowth, however, I'd be remiss not to mention. In short: support the gut, and the gut may help support your sleep—even under physiologic stress.

The Illusion of the Easy Fix

To the detriment of sales and the chagrin of some of the brands who are my customers, I'm often more supportive of the pharmaceutical industry than people expect. Over the years I have received my fair share of angry emails—usually from natural-health absolutists or anti-pharma crusaders—telling me they'll never buy products developed by me again, always in response to a nuanced position I put forth that fails to satisfy their extreme "black and white" thinking. Apparently, not parroting their ideology makes me untrustworthy. That's a consequence I am willing to pay, as I don't subscribe to any particular ideology, I relentlessly pursue the truth, utilizing the evidence we have and weighing it as carefully as possible.

The truth is that the pharmaceutical industry has brought us extraordinary advances: cures for once-fatal diseases, lifespans extended by decades, and reasonably safe interventions for those who refuse to modify their lifestyle. This doesn't mean I support drugs as a first-line solution for every problem, or even most problems, or that I think we should medicate away what lifestyle change could fix. It also doesn't mean that I am not highly critical, and extremely skeptical, of virtually every pharmaceutical driven narrative. I write about this in extreme detail in *The Final Thought War*. Despite my scathing criticism, bordering on rage, for certain decisions that undermine science and manipulate the truth, to pretend that medicine hasn't transformed human health would be a willful denial of reality, one I am not prepared to make in order to please an audience.

I mention that certain treatments can assist in situations where patients refuse to make lifestyle changes. Any exhausted medical doctor, burnt out from listening to an endless flow of excuses on why better nutrition and increased activity are impossible, will likely vouch for the statement that most people don't want to improve if it takes even a modicum of effort, they just want a magic fix. I also firmly believe that many people don't even want a magic solution—their victimhood, and weakness, have become too intertwined with their identity. To cure what ails them would be to kill who they believe themselves to be. It's a tragedy befalling our society, one which allowed for this disease of softness and fragility to creep in. A driver behind my desire to write this book.

In January 2020, before the pandemic, I found myself in the ER, hooked up to an IV antibiotic drip—the aftermath of an ill-timed intervention in a cat fight—my cat was involved, of course. The doctor treating me was sharp, attentive, and clearly passionate about science. We ended up chatting about a number of research topics while I waited, from epigenetics, to aging, to microbiome and more. Then I watched another conversation unfold nearby, involving the same ER doctor. A man in his late 20s or early 30s, accompanied by his mother, was describing his symptoms: poor sleep, chronic fatigue, headaches, back pain. He was, by any estimate, at least 100 pounds overweight, visibly sedentary, slouched in his chair with what can only be described as mechanical apathy.

The ER doctor, calm and professional, explained that after a full battery of tests—none of which showed anything clinically wrong—he believed the man's issues stemmed from lifestyle. His advice was to clean up the diet, start exercising, and move more. Basically, he was telling this individual to take responsibility for the systems breaking down under disuse. The patient stared in disbelief, mouth open, shaking his head like he'd just been simultaneously scammed and blatantly insulted. His mother did not share the same incredulous silence, she was absolutely livid. "You're not helping him," she firmly stated, almost a shout. "Can't you *prescribe* something?" What they wanted was a pill: a magic fix. Something to make the pain go away without doing any work, or taking any personal responsibility.

To his credit, the doctor didn't fold. He reiterated that no drug would solve what diet and movement could, but as the tension escalated, you could feel him waver. Eventually, he suggested chiropractic or massage therapy "to help with the back pain and maybe sleep." That seemed to pacify them, and they left still angry, but sufficiently placated. The son stopped to turn around and glare at the doctor on the way out. I'll be honest—my first reaction was contempt, anger, and frustration at the entitlement. I was angry at the refusal to take responsibility, but after a few hours of reflection, my anger shifted.

The deeper problem isn't that people want easy answers, it's that the *system* trains them to expect one. We hand out diagnoses like candy. We offer symptom relief

without root cause resolution. Most doctors are too overwhelmed to invest time into real behavior change, and most patients don't have the structure—or mental stability—to initiate that change on their own. When medicine refuses to entertain the fantasy of a quick fix, charlatans step in. They sell the illusion of personal care while pushing unproven protocols. They fill the emotional vacuum with false hope. These charlatans come by way of major pharmaceutical companies, and also of unscrupulous actors—influencers and companies alike—in the alternative and natural health space.

An added layer is the fact that failures in physical health may often be attributed to deteriorating mental health, even if these issues are not yet diagnosable. The ER doctor in question could not have reasonably spent enough time to get to know the patient to help them work through their issues, that is not his role in medicine. This is a key reason how charlatans often win over those in need; they give the illusion that they care about the individual while capitalizing on the situations described above. Most of us have experienced a physician unable to help us with a non-acute ailment. It will continue to happen more and more, as the reality is we are dealing with overworked doctors who often have too many patients, or ER or walk-in clinic doctors who do not have a personal connection with those they are treating.

We don't need more drugs, and we don't need more sham therapies lacking evidence. We need systems that work towards improved health and function, a system that prioritizes preventative care. We need an educational system and supporting infrastructure that promotes long-term behavior change and that treats people with dignity but also holds them accountable for their decisions and actions. Systems and solutions that distinguish between compassion and coddling. Because there *are* evidence-based interventions. We just need a system that is willing to do more than prescribe—and a population willing to do more than consume.

I digress. Let's get back to sleep—and the treatments that actually work.

Prescription Sleeping Pills: When the Cure Compromises the Repair

In many cases, sleeping pills can be necessary as a last line of resort. Even Peter Attia has admitted he has prescribed, and still will, sleeping pills in extreme cases as he tries to work out why patients have insomnia. This is a man who has quoted (to paraphrase): "*I tell patients I'd rather them put their genitals in a meat grinder than stay on sleeping pills permanently*" (Attia, 2022).

What Do the Experts Say About Sleeping Pills?

According to Matthew Walker during various talks, he has argued that powerful sedatives/hypnotics are not restorative sleep. He argues that simply being "unconscious" is not akin to the restorative qualities of sleep (Walker, 2017).

Dr. Daniel J. Buysse, a sleep medicine expert and professor of psychiatry at the University of Pittsburgh, argued towards a more "personalized" assessment regarding the use of sleeping pills, quoting: "*Do you feel more rested, more alert, more able to concentrate, less irritable on medication versus off*?" (Weintraub, 2015). Dr. Buysse said. "*If all those things are true then I would say it's more restorative. If a hypnotic drug leaves you feeling hung over or more anxious, if it causes you to order five hickory smoked turkeys on the Internet without remembering, then it's probably not good*" (Shoemaker, 2017).

This position seems to be supported by Dr. John Weyl Winkelman, a sleep disorders expert at Massachusetts General Hospital and Harvard Medical School. When asked what he'd tell a patient if asked whether medicated sleep was restorative, his response was "*I'd say: 'You tell me*'" (Weintraub, 2015).

While I've never taken sleeping pills, my experience with an opioid I was prescribed as a teenager following a nasty "near" compound fracture just below the shoulder, bone piercing through the muscle but stopping short of breaching the skin, and the accompanying rolling muscle spasms, left me hesitant to ever take an opioid again. Some of my experiences parallel what others have described with their memories of sleeping pill addiction and prolonged use. Said drug, trade name Darvon, is now recalled in many countries, including Canada and the USA. In my experience, it resulted in a minimal reduction in pain.

What it did do was put me in a haze for the three months I was taking it. In fact, it is the one period in my adolescence where I have no clear, strong memories. The closest I have is the searing pain I felt when slipping in the shower and bumping my broken shoulder. The opioid crisis, and ensuing black and white debate, is a topic for another time. Yet, I view that topic, and the sleeping pill debate, as being closely related. I believe these drugs have a use in some cases, but are overused and over-relied upon to a staggering degree I can only describe as a mix of enraging and nauseating. Some people legitimately need these drugs, perhaps they are desperate and have circumstances where other methods have not worked, and we shouldn't vilify their use in isolated cases. We absolutely should vilify their use, when viewed as the totality of their distribution.

So What Are the Risks of Long-Term Use of Sleeping Pills?

Sleeping pill-induced sedation not leading to restorative sleep, and the accompanying morning grogginess and forgetfulness some patients experience, is a major cause for concern, but the issues with sleeping pills don't stop there. There are several other concerns surrounding long-term reliance on sleeping pills. In a large epidemiological study, even occasional users (fewer than 18 pills per year) had a 3.6-fold increased risk of death compared to non-users, while those in the highest usage category (>132 pills/year) showed a 5.3-fold higher mortality risk (Kripke et al., 2012). Additionally, the heaviest users, defined as those taking more than 132 pills per year, demonstrated

a 35% increased risk of developing new major cancer compared to non-users. This landmark paper was written by a prolific team in the field, previously publishing 18 papers on links between sleeping pills and negative health effects over almost four decades (and have published more work on the subject since). Following the international attention received, the work has been replicated by other teams since (Hedström et al., 2020). A different meta-analysis examined the cancer risk component of the previous study and concluded: short-term or low-dose hypnotic use likely carries little or no increased cancer risk, while longer-term use and higher doses were important factors influencing cancer risk (Peng et al., 2020).

The FDA has recently ordered many manufacturers and marketers of sleeping pills to step up their warnings to consumers regarding serious side effects. They singled out Ambien, Sonata, and Lunesta, while bringing up worrisome behavioral reports, such as sleepwalking, sleep driving, and engaging in other activities while not fully awake. While this warning was announced in May 2019, the FDA has previously warned about sleeping pills and potential worrisome effects as early as 2007 (U.S. Food and Drug Administration, 2019). In 2024, the FDA reiterated these concerns, reminding the public that prescription Z-drugs like Ambien, Sonata, and Lunesta carry rare but serious risks (including injury and death from complex sleep behaviors such as sleepwalking, sleep-driving, and even self-harm) and urged patients to discontinue use and contact a healthcare provider immediately if such behaviors occur (U.S. Food and Drug Administration, 2024). A link was also found between sleeping pills and an increased risk of pneumonia (Jung et al., 2016; Obiora et al., 2013). As well, research has established a link between increased pneumonia, sleeping pills, and those with Alzheimer's disease (Huang et al., 2023b). It has previously been discouraged to prescribe sleeping pills to those suffering traumatic brain injury due to negative effects on neural plasticity (Larson & Zollman, 2010), while an animal model showed that animals dosed with sleeping pills slept longer than control animals, but had less, not more, brain plasticity. It led to a 50% unwiring of connections (Seibt et al., 2008).

CBT-I: When All Else Fails

Cognitive Behavioral Therapy (CBT) has become the flagship modality of modern psychotherapy, not necessarily because it cures (it doesn't), but because it scales. It is tidy, protocol-driven, and easily reimbursed. CBT offers structured sessions, reproducible worksheets, and symptom management strategies that help patients function within the very systems that often contribute to their distress. For many struggling with chronic anxiety, trauma, or depression, it becomes a long-term maintenance plan rather than a path to resolution. In this sense, CBT often operates more like palliative care for the psyche than a truly transformative treatment. That is the point of it, certainly: to satiate rather than alter.

That said, there is one domain where CBT's mechanistic, behavior-focused design

consistently delivers: *insomnia*. Cognitive behavioral therapy for insomnia (CBT-I) is an approved treatment of insomnia that can be used as a stand-alone or alongside sleeping pills that aims to identify the underlying cause of insomnia and treat it based on improving habits and sleep hygiene, while also incorporating numerous other methods. The exact protocol is typically tailored around the individual. CBT-I was found roughly as effective in treating insomnia as sleeping pills, taking a bit longer to work, but showing a higher rate of deep sleep and better long-term results after treatment when directly compared to a hypnotic sleeping pill (Cervena et al., 2004; Trauer et al., 2015).

In fact, when CBT-I was compared as a stand-alone treatment against CBT-I and Ambien, the results were not statistically different. CBT-I worked as well on its own (Morin et al., 2009). The efficacy of CBT-I has been established in numerous other trials and confirmed by meta-analysis (Trauer et al., 2015), although another meta-analysis notes that while effective, there is room for improvement (Hwang et al., 2025; Soh et al., 2020). Further, it is the preferred treatment by experts previously discussed in this series, such as Matthew Walker and Donn Posner. The efficacy has also been supported by Dr. Daniel Buysse, who, in a review he co-authored with colleagues, supported behavioral therapy while maintaining a neutral position on sleeping pills (Buysse et al., 2011).

Even fully automated, computer-generated CBT-I protocols have shown significant clinical benefits and cost-effectiveness. While they may be somewhat less effective than treatments delivered by therapists, their accessibility and scalability make them a valuable resource for reaching more individuals who might not otherwise receive care (Hwang et al., 2025). Based on the efficacy of CBT-I and the potential dangers of sleeping pills, it is likely preferable to attempt CBT-I first. That said, if CBT-I does not work for you, and the different strategies we discuss next week fail to find a benefit, it may be time to speak to your healthcare provider about temporarily trying sleeping pills.

Strategies for Aiding Sleep

The following are strategies I have personally employed, and can attest to have made a significant impact on my own sleep hygiene. I want to caution that these may not be effective in patients with clinical insomnia, however, each reader needs to be honest regarding their diagnosis. If you are currently on a prescribed medication or protocol, and hoping to stop, I caution you to speak to your practitioner first. For supplements I take to help promote sleep, please see the next chapter, in which I discuss all of my supplemental, experimental drug, and hormonal protocols.

Reading

For myself, depending on what I am reading, I find it either an incredible sleep aid, or an unmitigated disaster, with my interest in the content driving self-induced acute

insomnia. This observation is by no means related to how interesting I find the subject. In fact, some of the writing I find most interesting puts me to sleep the quickest, particularly heavy reading such as philosophy and science. In the past, I've mused heavy reading in bed may increase strain on my mind, which is already running on fumes by the end of the day, and everything I have learned over the years seems to confirm this. Easy to follow "pop science" books or articles, or even journal articles in subjects I am well acquainted with and can breeze through, and especially enthralling literature grounded in storytelling, whether it be fiction or nonfiction, will keep me turning the page all night.

Reading scientific publications on a subject I am struggling to understand, or reading and re-reading pages of philosophy that I need to think deeply on, will put me to sleep quite quickly. I've used a strategy where my end of night reading is the heaviest subject matter of the day for over a decade, and it still works like a charm. I begin reading roughly 20 minutes after taking my melatonin, and other sleep aid supplements, and rarely make it through more than 10 pages. Like clockwork, my eyes begin blurring and my head begins nodding, rendering my ability to find what sentence I last read nearly impossible. When this occurs, I close the book, shut off the reading light, and am typically asleep within minutes, resigned to re-reading the page I ended on the next day.

In addition to the benefits of driving sleep pressure, knowledge is its own reward. Reading is something I would encourage for everyone. We train our bodies and it should be a personal obligation to train our minds, the entire purpose of the companion book on *The Mind* in this volume. I recommend finding a subject you want to learn, one that you have a hard time following late at night, and giving it a try. It may improve your sleep while helping pursue new thoughts and knowledge. Keep the page-turners for earlier in the day.

Guided Hypnotherapy Recordings

The potential benefits of guided hypnotherapy go well beyond aiding in sleep. Unfortunately, many proponents of hypnotherapy have injected tremendous amounts of quackery, negatively impacting public perception. Guided hypnotherapy recordings used to be a frequent crutch of mine, although I rarely need to utilize them now, having dramatically improved my sleep hygiene, and stress management techniques during the day. That said, I still utilize it any time I lay in bed for more than 45 minutes without falling asleep, which at one time was every night. Hypnotherapy isn't a magic fix, and you have to allow yourself to be relaxed into the sleep state. Sometimes it fails, but often it becomes my saviour. When it works, I fall asleep quite quickly and tend to sleep through the night. I personally use, and highly recommend, the free recordings of Dr. Paul Ogilvie, a medical doctor from the UK, at liberationinmind.com

Cold Room

It's widely reported that sleeping in a cold room helps you fall asleep quicker, and sleep better, with many sleep scientists recommending you sleep in a slightly cooled room (Togo et al., 2007; Kräuchi et al., 2000). Personally, barring massive protest from my fiancée, I run my A/C all spring, summer, and fall, and would in the winter if I was allowed to. In the past, during the winter I would turn off my heat and crack my window open to allow cool air to come in. That said, I live in the Greater Vancouver area of Canada, and as such it rarely gets as cold as many others would identify as "cold." While sleeping in a cool room may come with sleep benefits, and potentially other health benefits, make sure not to be "too cold" for too long, otherwise, it could come with negative health implications, some of which I discussed in Chapter 1.

Recommendations I Can Anecdotally Support

- Don't eat within a few hours of sleeping.

Again, I have seen this recommended everywhere from various sleep experts to researchers supporting time-restricted eating to support our circadian rhythm. Anecdotally, if I eat a heavy meal within 3 hours of sleeping I feel hungover the next morning. It hits me like a ton of bricks, without fail, any time I break this rule.

- Don't work out too close to bedtime.

When we train hard, we get a rush of various endorphins. I know that when I work out too close to bed, I am "wired" and cannot fall asleep, and the sleep I do get is fractured and of suboptimal quality. Avoiding late-night exercise is advice I have seen posted by sleep experts, and it seems prudent. That said, if you do not currently exercise, there are few situations where starting a fitness program immediately wouldn't be beneficial. Unless you have a debilitating condition or disability preventing movement, take up an exercise program without delay. Expending energy may help your body relax and sleep better. Becoming healthier and more physically fit may help resolve some of the issues driving poor sleep. Further, physical fitness is one of the most important arsenals in your health routine. Even if it doesn't help you sleep, incorporating a moderate exercise plan is ideal.

Other Recommendations I Haven't Found to Work, but Others May (and Have Been Discussed by Sleep Experts) -

- Turn off half the lights a couple of hours before bed.

During bouts of poor sleep, I tried this years ago and personally, didn't find any benefit. For others, it may be worth a try.

- Early morning sunlight.

When I installed a Thai bag on my patio in my mid-30s, at a time I was suffering from chronic sleep issues, I started the habit of waking up each morning to do a round or two on the bag outside in the early morning sunlight. I then sat and worked beside an open window getting direct sunlight (that I kept open for my cat). I didn't find this practice, which I began for other motivations, to have any bearing on my sleep. For others, it is easy and may be worth a try.

- White noise

According to Matthew Walker, white noise is at least not harmful. I recently was given a HATCH sleep aid, which offers options of a dull light, and sound. I typically rotate between waves crashing, and the sounds of a rainstorm. Both I find extremely soothing, and help me relax and sleep. In the past, while on vacation, I have been stunned by the soothing and restorative aspects of the sounds of nature. In particular, running water, and the sounds of the forest, have always touched me to the core of my being.

Recommendations Others Have Spoken About That I Haven't Tried

- Rhythmic sound has limited evidence.
- Electrical stimulation can be harmful or effective. Do not try at home, Matthew Walker does this in his lab.
- If you have issues with your partner, consider sleeping in different beds.

My Lessons in Sleep & Self-Discipline

I get it better than most: winding down isn't always easy—especially if your work, like mine, lives across time zones. Many of my contacts are overseas, and not just in a single continent. I do business, and collaborate on research, worldwide: from North America, to Europe, to Australia, to Russia and East Asia. Not a day goes by where I am not receiving emails past 10 or 11 PM, often after midnight. Even people on the West Coast send last-minute messages while clearing out their inboxes, and I've always been one of those rare types who tries to hit inbox zero every night. It causes me great pain to make others wait—especially when I'm waiting on them. I'd rather lose sleep than delay momentum. I talk about this philosophy in *The Mind* on the compounding nature of time.

That mindset, while productive, was destroying my sleep. I used to wake up multiple times in the middle of the night to check my inbox. Not occasionally—*routinely,* as a habit. If someone emailed at 1 AM, I'd respond by 3 AM. If they emailed at 3:30 AM, I'd typically respond by 5:30 AM. During these days, come ~7 AM, I'd be in and out of light sleep for hours. Waking up every 15-20 minutes to respond to an email or two, before dozing back to sleep. It was unhealthy and unsustainable.

That's changed. I still haven't followed the advice to shut my phone off at night—I know myself, and I'm not there yet—but I've made meaningful changes:

- I don't check email in the middle of the night.
- I don't schedule early morning calls.
- I don't open my inbox until I've decided it's time to wake up.

It's a small shift, but the impact has been real. I'm sleeping deeper, waking clearer, and most importantly, I'm not starting each day in a mental haze and metabolic distress.

Customize, Don't Copy

This is the part most sleep advice gets wrong: discipline isn't one-size-fits-all. Not everyone can turn off their phone at 9 PM. Not everyone has the luxury of ignoring international clients or setting rigid digital boundaries, and that's okay. What matters is this: build a system that works for you—and one you can *actually* stick to.

For me, that meant modifying my environment and expectations *without pretending* I'd become someone I'm not. The result is a routine I'm maintaining effortlessly, and a sleep routine that's no longer compromised by obligation. Your path will look different, and that's not just ok—it's the point.

The goal isn't perfection. The goal is progress you can sustain. Find what works, refine it, and honor it. Your resilience depends on it.

Interlude: The Edge of Sleep

My fiancée has told me that she used to hate English. Not mildly, viscerally. She came from Russia at eleven, not by choice, and to her, the language felt clunky, ugly, and stupid. Every word grated on her, a reminder of the friends and home she was torn from. It wasn't just foreign, it was hostile. Even before departing Russia, not knowing her family was emigrating, she had opted to learn German in school instead of English, holding a negative view of the language prior to being thrust into it.

She's spoken to me about the turning point for her, when her anger subsided and her desire to master English began. A few months after moving to Canada, something changed: she started dreaming in English. She wasn't just understanding the language, she was thinking in it, and feeling it. Her mind had crossed over, and that changed everything for her. Her anger dissolved, replaced by confidence, pride, and curiosity. She describes feeling elated, thinking to herself "*I am getting really good at English*," and that positive feeling, mixed with the pride of her accomplishment, drove her to continue refining, and perfecting the language. She may not have chosen to come to Canada, or to learn English, but as soon as she felt accomplished at her forced situation, her emotions changed.

Now she speaks three languages: Russian, English, and French. The point is that fluency doesn't start with grammar. It starts when the resistance breaks, when something clicks, when it's not about memorization, but embodiment: when a foreign system starts to feel like home. In this instance, that "click" first revealed itself in a dream, during sleep. Perhaps it means nothing and is just a coincidence, or perhaps our dreams are able to shift our perspective, and give us hints to what we are missing in our conscious reality. I've covered a lot in this chapter—circadian rhythms, sleep stages, chronotypes, wearable tech, behavioral strategies, and even the pharmacological edge cases where sleeping pills might make sense. We've talked about inflammation, glymphatic clearance, cognitive repair, hormonal regulation, all of which are real, measurable consequences of how and when we sleep. But there's something we haven't talked about at all: dreams.

What do they mean? Do they mean anything? Are they psychic detritus—your brain's nightly purge of undigested thoughts? Are they wish fulfillment, ancestral memory, emotional sorting algorithms? The neuroscience isn't settled, and the psychoanalysis never was. I'm not going to pretend to have an opinion on this topic, as I simply do not know enough about it. From what I have gathered, what we *do* know is that dreams are not random. They're structured, personal, and recurring. Sometimes, dreams are lucid. Sometimes they're terrifying, and occasionally, they reach into something so deep, so unnameable, that we wake with our hearts pounding—not necessarily from fear, but perhaps from the sense that we've just brushed up against *something real*. The most vivid dream I can recall felt exactly like that.

I was dreaming, and I *knew* I was dreaming. It was a lucid dream, a practice I kept at the time. I was in full control, until I wasn't. A girl appeared, and I don't remember what she said, only that something shifted. She felt foreign to *my* dream world, as if she wasn't invited, wasn't playing the role I wanted her to play. I seem to recall some confrontation took place, me being arrogant and dismissive, believing I was in full control. Then, she turned sinister, and told me she was in control, not me. The world around me changed in a concerning manner. I remember it became dark, foreboding, and vaguely remember a red glow. Almost like the common perceptions of hell on Earth, or something to that effect. No longer enjoying the experience, I forced myself to wake up. I didn't wake in my bed, and remember being confused by that, I woke on my couch, or something that *looked* like my couch. The radio was on, and to clarify I didn't own a radio, and it was describing my waking, live, as if I were listening to a broadcast of myself. Then I saw it: a dark figure, shadowed, and demonic: advancing toward me with a weight and force in every step that reverberated through my body, and shook the house.

I tried to rise and fight, but my body didn't work. I fell, paralyzed, sinking into the floor. Then I really woke up, still on the couch. Perhaps I had sleepwalked to the couch in the middle of the night, or gone for a glass of water and passed out on the

couch, forgetting I had moved from my bed. I was 20 years old when this happened, and likely due to my frequent use of drugs, perhaps amplified by my severe caffeine addiction and intermittent battles with insomnia, sleepwalking wasn't unusual. This dream motivated me to scour the internet for others who had experienced the same thing. This was no "Hat Man," but the figure had horns—almost like a corporeal version of Sauron, etched into my brain from a *Lord of the Rings* movie. What exactly occurred? A glitch in consciousness? Sleep paralysis? A spiritual encounter? I don't know, but I know this: we sleep for a reason, and the veil between sleep and waking is thinner than we like to believe. Whatever that experience was, it shook me, and while I still suspect we'll one day have a coherent scientific explanation (maybe something about REM intrusion, thalamocortical disruption, or another elegant mechanism), I can say with full conviction: none of that mattered at the moment. My emotional state was real, more real than any rational framework I could retroactively impose on it. The fear wasn't imagined; the disorientation didn't feel neurological—it felt existential.

That moment left a mark, and I haven't practiced lucid dreaming since. It's been nineteen or twenty years. You can call it avoidance, maybe it is, but that choice wasn't driven by superstition, it was driven by respect; for the weight of altered states, for what the mind can generate when left untethered, and for how easily curiosity can become chaos when you mistake lucidity for control. Dreams remind us that sleep isn't just repair—it's revelation—and perhaps what we fear most isn't sleep deprivation, but the truths that wait when we surrender to the night. You can track your REM cycles, optimize your temperature, cut caffeine, and take your magnesium. You can hit seven hours on the dot and still wake up haunted. Because the moment you close your eyes, you surrender control.

Ask anyone who's experienced sleep paralysis. You're frozen—your mind awake, your body immobile, as if some ancient predator sits on your chest. You try to move, you try to scream, you feel a presence: not a metaphor, not a dream, *presence*.

Friedrich Nietzsche (1901/2006) wrote that the will to power is the engine of life, but what happens when that will is severed from the body? When your sovereign command is rejected by your limbs? The experience shatters the illusion that you are the master of your own house. Fyodor Dostoevsky (1864/1994) called it the underground: the place in the psyche where repression breeds revolt, the place where dreams become stage-plays of inner torment. Where guilt, fear, rage, longing—all the things you swore you had under control—return in costume. Finally, Carl Jung (1964/2023) reminds us: dreams are not random. They are mythological, archetypical, and symbolic. The serpent, the flood, the chase, the shadow—these are not just personal, but cultural. Your dreams are stitched from the stories your ancestors survived. A man in Finland and a woman in Nigeria might both dream of a great beast, but its face will differ.

So what is resilience here? Not lucidity, not dream hacking, not control, but the willingness to face what you can't explain, to enter the cave, to let go, and still come back with something true. Sleep is not the absence of life, it is where life reconfigures, and it is not weakness to descend, rather, it is strength to return.

Conclusion: The Case for Sleep

As you've learned over the course of this chapter, sleep is not optional. If exercise, proper nutrition, and the various hormetic stressors discussed in this book are the pillars of your temple, sleep is the foundation. In the quest for good health, sleep optimization is perhaps the single most important intervention for physical repair, emotional resilience, hormonal balance, metabolic stability, immune function, and cognitive clarity you can take today. Sleep is not hormetic, however, though it shares one thing in common with the various forms of hormesis discussed in this book: dose is critically important. Too much, or too little, and you will suffer grave consequences. There is no benefit to missing out on sleep when your body craves it, even if you think it gives you an edge. To repeat, sleep deprivation does *not* make you sharper, even if you delude yourself into thinking this is the case.

Deprive the body of sleep and it begins to unravel. It does this quietly at first, and then system by system: inflammation rises while hormones misfire and telomeres fray, while your fatigue deepens into depression. No supplement, medication, or optimization hack can replace the biological intelligence built into a full night of deep, uninterrupted rest.

If you do nothing else, if you change nothing else, fix your sleep. Protect it like your life depends on it, because it does. Everything else you do builds on the restoration sleep provides.

Picture 34. Las Vegas, NV, before the first UFC Noche

Our night ended with tables set up in the kitchen of a Mexican restaurant so Loopy wouldn't get mauled by fans after her dominant win.

CHAPTER 11:

Advanced Recovery Techniques

Introduction

The irony of the title *StressHacked* isn't lost on me. Strength, which is to say real, integrated, physiological, and mental strength, isn't a hack: it's a system driven by a guiding philosophy. It's a way of life that requires consistency, exposure, discipline, recovery, adaptation, and time. If I've learned anything from my years of self-experimentation, it's that you can't shortcut a system; you can only understand it better and work with it more precisely. The term "hack" is sexy, and so it sells, but hacks are often about skipping the hard stuff, and resilience doesn't come from skipping. It comes from stress, which is judiciously applied, carefully observed, and earned.

That's why, despite the clickbait title, *this isn't a book about hacks*. It's a book about systems thinking applied to hormesis: about approaching your body and brain like a scientist—like a biohacker in the original sense of the word, the one rooted in hacker ethics. Not the influencer-driven, fear-based culture that biohacking has largely become. After all, when techniques move away from science to fit a narrative, it destroys the entire benefit of biohacking. This book is about reclaiming that benefit. It's about understanding that progress takes work, and that strength is built, not bought. It's not a stack and it's not a patch, and it certainly isn't something you saw on TikTok in a 15-second clip.

Everyone's system is going to look a bit different, and that's why this book is intended to lead to an understanding of what each of us needs to accomplish, and how we can diligently seek knowledge and truth about ourselves to create a system that works for each of us individually. If this is too much for you, that's fine; no one is stopping you from parroting someone else's cookie-cutter protocol. Just know that your results are likely to suffer, as we are not homogeneous beings; what works for others may not be effective for us—and in fact, may harm us. While previous chapters explored the deliberate application of stress for building resilience, this chapter focuses exclusively on advanced recovery methods designed to accelerate tissue regeneration, systemic rejuvenation, cellular repair, and cognitive recovery. These techniques aren't intended to induce hormetic stress themselves, but rather to optimize the recovery phase where adaptation actually occurs. Think of them as complementary tools supporting optimal adaptation following your strategic stress exposures. I want to state emphatically that the following are protocols that I personally use and have incorporated for a specific purpose. Everyone's unique needs will differ, so do not take my protocol as a definitive blueprint for your own supplementation routine.

Advanced Supplemental Strategies for Deep Recovery

Recovery isn't just rest, it's reconstruction, and that process utilizes substantial raw materials: amino acids, co-factors, anti-inflammatory signals, and mitochondrial substrates. If you're applying stress deliberately, whether it be fasting, training, cold exposure, or any other mechanism, your body needs targeted inputs to rebuild at the cellular level.

The goal here isn't just to "feel better," it's to recover better. You've put in the hard work, now it's time to assist your body in repairing tissue faster, sleeping deeper, and facilitating a more efficient metabolic reset. Below are compounds with solid mechanistic rationale and emerging human data behind them, which I am currently taking, or am intermittently utilizing. These aren't cure-alls, but they are potentially useful tools when used in the right context, for the right reason. The following are the doses I am taking, at the time of writing, and should not be used as a definitive guide for the dosing your body, and its unique needs, may require.

Glycine (10g/day)

Glycine isn't just for joint health, it's a *recovery essential*. It drives collagen production, supports connective tissue repair, and lowers systemic inflammation. Crucially, it also helps drop core body temperature and improve slow-wave sleep, making it one of the rare supplements that accelerates both physical and neurological recovery (Ramos-Jiménez et al., 2024).

Personally, I split my Glycine into 5g in the morning, and 5g 30 minutes before bed.

N-Acetyl Cysteine (NAC) (1.2g/day)

N-Acetyl Cysteine (NAC, 1.2g/day): As a precursor to glutathione, which is your body's master antioxidant, NAC significantly mitigates oxidative damage following intense hormetic stressors. Research in the *Journal of the International Society of Sports* Nutrition demonstrates NAC's ability to reduce exercise-induced muscle damage and accelerate recovery times by up to 30% (Fernández-Lázaro et al., 2023; Devrim-Lanpir, 2021).

A glutathione precursor and oxidative stress shield. NAC helps neutralize the inflammatory aftermath of training, fasting, or illness.

I personally take 1.2g every morning, as my stress load is substantial.

Essential Amino Acids (EAAs) – 5g/day

Essential Amino Acid Blend (5g/day): Complete EAA formulations dramatically accelerate muscle repair post-exercise.

Unlike isolated BCAAs, which only cover a fragment of the muscle protein synthesis pathway, a complete EAA formulation delivers the full amino profile required for real structural repair, especially critical during fasted training or post-load glycogen recovery (Wolfe, 2017).

Personally, I use either Longevity EAA by Unmatched Supps, or PerfectAmino by BodyHealth, depending on what I get my hands on when I come to the USA.

Full disclosure: Kris Gethin, the owner of Unmatched, is a personal friend and has endorsed my technologies publicly. Dr. David Minkoff, the formulator and owner behind Perfect Aminos, is a customer of mine. That said, if I didn't believe they were the best options available, I wouldn't take them, let alone recommend them here.

I personally just take them first thing in the morning, so as not to forget, although I believe the suggested use is after exercise.

Omega-3 Fatty Acids (EPA/DHA) (5.12g/day)

Omega-3 Fatty Acids (5.12g/day): High-dose, high-purity EPA/DHA supplementation provides critical support for inflammation resolution and tissue repair without blocking beneficial inflammatory signaling. This precise dosing optimizes recovery while supporting cardiovascular and neurological health (Calder, 2017).

Personally, I supplement with Nordic Naturals. I try to take the softgel capsules with my meals, however, occasionally I take them in the morning with other supplements on days I know I may forget, i.e., when travelling or not eating at home. I take a larger dose than recommended as I have chronically low cholesterol, sometimes sub 100 mg/dl, despite eating substantial animal fat. Omega 3s help me optimize the ratio of HDL to my TC.

Vitamin D (10,000 IU/day) + Vitamin K2 (1.2mg/day)

Vitamin D (10,000 IU/day) and K2 (1.2mg/day): This synergistic combination supports hormonal optimization, bone health, and cardiovascular recovery systems while enhancing immune function during intensive recovery phases. K2 is critical to consume in combination with D3, as it ensures that elevated D3 doesn't misdirect calcium deposition (Gasmi et al., 2021).

I personally take this dosage first thing in the morning. Despite working beside a window and spending considerable time outdoors, I measure chronically low Vitamin D levels when I am not supplementing. I seem to need 8000-10,000iu daily to maintain a normal range. Before I started this protocol, I suffered from seasonal affective disorder (SAD), a common occurrence for people in northern climates. I'm particularly

at risk, also having ADHD. Low vitamin D levels are correlated with SAD, however, it is unclear if Vit D supplementation reduces the symptoms of SAD. Regardless, I have not had issues in years, since beginning my supplementation protocol.

TUDCA (500mg/day, cycled) + Milk Thistle (3g/day, cycled)

TUDCA (500mg/day, cycled) & Milk Thistle (3g/day, cycled): These hepatoprotective compounds provide critical liver support during intensive supplementation periods. TUDCA supports bile flow and endoplasmic reticulum stress response; milk thistle supports hepatic regeneration (Abenavoli et al., 2018; Ben Mosbah et al., 2010). The liver plays a central role in recovery and adaptation processes; supporting its function enhances overall recovery capacity.

Typically, I will use both for 1-2 months, every 4-6 months, depending on my lab reports.

Chromium Picolinate (1mg/day)

This trace mineral supports glucose metabolism and mitochondrial function, particularly following fasting or metabolic stressors. Its insulin-sensitizing effects enhance nutrient partitioning, as well as mitochondrial efficiency, during recovery phases (Dubey, Thakur, & Chattopadhyay, 2020).

Personally, I take my chromium picolinate through a custom hydrogen tablet I developed for Water & Wellness, called Active H2 Chromax. I take one daily, during the time I have allotted to consume my HRW.

Molecular Hydrogen

Hydrogen Tablets for Hydrogen Rich Water (HRW) (4-6/day)

Hydrogen Inhalation from InhaleH_2

Hydrogen Bathing, via Hydrogen Bath Tablets

My HRW protocol is constantly adapting. As I detailed in Chapter 8, molecular hydrogen (H_2) shares some overlap with hormetic stressors, but is not a stress in and of itself—at least not one that risks toxicity or harm. As for protocols, I recommend alteration every 3-6 months, with a 1-week washout period in between. Two common protocols I utilize are as follows: 4-6 of my patented open-cup hydrogen tablets, available through numerous brands, dropped in 1L of room temperature water, either first thing in the morning or immediately before exercise, on an empty stomach. It is important to chug the water as fast as you can, do not sip. Most cannot chug 1L, so this protocol is only ideal for those that can, and who have considerable physiological stress.

Other times, I will split my dosage into 2 or 3x a day, first thing in the morning, around 1 pm before I exercise, or late afternoon for a pick-me-up. When I do this, I

will add 2 tablets to 500mL of water, consuming 2-3x daily. It is imperative to drink on an empty stomach, as various fibers are able to stabilize and retain bubbles of H_2 gas in aqueous solutions. This could lead to dramatically slowed and impaired circulation through your system, hindering any therapeutic adaptation.

For hydrogen inhalation, I utilize mine and Dr. LeBaron's patent-pending design, InhaleH_2. We spent 7 years on the R&D for this machine, leading to the first safe and effective unit on the market, the first unit that is able to perfectly mimic large-scale clinical trial use, and control the precise concentration of each breath, without any risk of flammability or explosion. I typically inhale for 30-90 minutes, late afternoon or early evening, while either watching a show with my fiancee or working on my laptop.

For hydrogen baths, I utilize the hydrogen bath tablets I produce for various brands. I typically bathe in hydrogen-rich water only when looking to recover from acute muscle stress, such as after injury or intense exercise.

My hydrogen protocol is substantial and constantly adjusting based on my needs. There is no one size fits all approach for any therapy, and this includes molecular hydrogen.

AGEless Defense (DrinkHRW)

AGEless Defense is the first product I have had a hand in formulating, other than the hydrogen tablets. I formulated it not for monetary demand, but because I wanted a solution for myself and nothing existed on the market to accomplish the intended outcome: inhibition of advanced-glycation end product crosslinking (AGEs). In short, AGEs are one of the pillars of aging itself. Particularly, the crosslink glucosepane builds up in collagen-rich tissues as we age. Because collagen in tissues like tendons doesn't get replaced much, glucosepane stays in the tissue for a long time and causes changes in how tissues work as we get older (Nash et al., 2019).

AGEs lead to the slow deterioration of our system, the result of sugar molecules binding to lipids and proteins in our body. Imagine a brand new set of sheets, strong and malleable. Then, imagine an old and starched-out set of sheets. Frail, full of wrinkles, and ready for the garbage bin, or to be cut to rags. That's what happens to our tissue.

AGEless Defense is a combination of the natural ingredients with the most promising evidence on inhibiting AGE formation, or protecting against the effects of AGEs once formed.

It incorporates key ingredients such as:

- 300mg of Nicotinamide Riboside, which converts to NAD+ which is crucial for the production of cellular energy

- 100mg of Sirtmax, a patented ingredient that activated SIRT1 and has demonstrated a reduction of AGEs in clinical research.
- 100mg of Rosmarinic Acid, an ingredient that has outperformed the best pharmaceutical candidates for AGEs in head-to-head research

As well, 600mg of Alpha Lipoic Acid, 500mg of L-Carnosine, 300mg of Bilberry powder, 250mg of Benfotiamine, 100mg of Vitamin C, 100mg of L-Taurine, 100mg of CurcuWin—a bioavailable form of curcumin showing 46x increased availability—100mg of cinnamon powder, 100mg of clove powder, 30mg of Vitamin B6, and 30mg of resveratrol are included.

I take my AGEless every day. I have family members who are more religious with it than with their hydrogen tables, including my fiancée. Best taken with food.

Radiant (Collagen + support, Drink HRW)

RADIANT was formulated as a complete regenerative collagen and skin health supplement. Eight grams of collagen per scoop are supplemented with 240mg of Hyaluronic Acid, 200mg of Vitamin C, 160mg of Lustriva (Inositol-stabilized arginine silicate and magnesium biotinate), and 75mg of ElastaGLO (hydrolyzed elastin)

I take RADIANT days I remember, and ideally would try to remember every day. My fiance never forgets. I tend to drink my RADIANT after a workout, or on an empty stomach.

PRIME (Creatine + GAA + Cofactors)

An advanced formulation that combines 3 grams of creatine monohydrate and 2 grams of guanidinoacetic acid (GAA) for muscle saturation, ATP regeneration, and brain energy support. GAA is a precursor to creatine, converting inside our bodies. It helps increase creatine levels in the brain more effectively than creatine supplements, though it's unclear how much GAA itself crosses the blood-brain barrier versus being converted outside the brain first (Ostojic et al., 2016).

Additionally, PRIME contains hydroxymethyl butyrate, or HMB. HMB does little for young men, but is pivotal for men over 30. It works by increasing anabolism, while decreasing catabolism. We lose, on average, 1% of our muscle per year after age 30, all things remaining equal. HMB can slow this deterioration (Su et al., 2024).

PRIME contains other pivotal ingredients such as 2.5g of beta-alanine, 2.4g of betaine anhydrous (TriMethylGlycine), a patented testosterone support called Tesnor, as well as boron and zinc.

I take my PRIME daily, typically about an hour after waking.

Taurine (3g/day)

Essential for cell volume regulation, antioxidant buffering, and cardiovascular protection. It may support sleep by acting on GABA and glycine receptors, and has emerging links to longevity (Schaffer & Kim, 2018). Taurine has also been shown to inhibit fructose driven AGEs (Nandhini, Thirunavukkarasu, & Anuradha, 2024).

I believe it is best to take it in the evenings; for myself, I add it to my PRIME in the morning, for convenience.

Nitric Oxide (NO) Support (varied dosing)

Whether through beetroot extract, citrulline, or advanced NO blends, supporting endothelial function and vasodilation boosts oxygen delivery, mitochondrial efficiency, and recovery (da Silva, 2023; Larsen et al., 2012).

Personally, I am taking Nitrosigine with my H_2 and caffeine, a patented form of arginine, then prior to exercise I take citrulline malate for an extra hit.

Caffeine (200mg/day)

Used with precision, caffeine can enhance performance, mood, and even mitochondrial biogenesis (Yamada et al., 2022). For general use, up to 400 mg spread across the day (about 5.7 mg/kg for an average adult) is considered safe. For physical performance, doses around 3 mg/kg taken less than two hours before intense exercise pose no safety concerns for most healthy adults under normal conditions. Caffeine also shows broader utility when used strategically: it supports metabolic, hepatic, and cardiovascular health, and has been linked to reduced risk of several major diseases. One umbrella review found coffee consumption associated with lower rates of liver disease, stroke, heart disease, some cancers, and all-cause mortality—without evidence of harm at typical intake levels (Poole et al., 2017). Finally, caffeine, at the right dose, has been shown to reduce neurodegeneration, as well (Socala et al., 2021; Ikram et al., 2020). Timing and dosing are critical, however: research shows that excessive or late-day intake compromises sleep architecture (Gardiner et al., 2025; Yamada et al., 2022). The European Food Safety Authority (EFSA) notes that single doses of around 100 mg—or roughly 1.4 mg per kilogram of body weight—can already affect sleep in some adults if taken too close to bedtime. For these reasons I stick to caffeine only in the morning, with rare exceptions, to avoid disrupting sleep.

I only take my caffeine with HRW and Nitrosigine, within 1hr of waking.

5-Methyltetrahydrofolate (Variable)

A bioactive form of folate is involved in DNA repair, methylation, neurotransmitter synthesis, and male reproductive health. This is especially important for those with MTHFR polymorphisms (Vidmar Golja et al., 2020). Dosing is important, and can

change drastically depending on the circumstances. For short-term use, methylfolate has been shown to increase male fertility at high doses, up to 5mg per day. However, this dose, chronically administered for the medium or long term, will invariably lead to increased homocysteine—especially if other b vitamins are not taken in adequate amounts.

I take it in the morning. Currently, 1mg per day, but previously I was taking 5mg a day to restore my fertility after discontinuing testosterone replacement therapy.

Zinc (15–30mg/day), Copper (1–2mg/day), Selenium (200mcg/day)

This triad supports antioxidant defense (via SOD and glutathione pathways), testosterone production, immune regulation, and thyroid function (Wang et al., 2023; Olechnowicz et al., 2018; Mertens et al., 2015; Klotz et al., 2003). Always balance zinc and copper intake .

I believe it is ideal to take with meals; however, I take it first thing in the morning with other supplements for simplicity and ease.

Low-Dose Tadalafil (Cialis, 2.5–5mg/day)

Primarily prescribed for erectile dysfunction and pulmonary arterial hypertension, tadalafil is increasingly used off-label for its broader vascular and longevity-related benefits. At low daily doses, it supports endothelial function, enhances nitric oxide signaling, and improves circulation to all tissues—including the brain (Ölmestig et al., 2024; Amano et al., 2017; Ozcan et al., 2017). Recent longitudinal data show that regular use of PDE-5 inhibitors like tadalafil is associated with significantly reduced all-cause mortality, cardiovascular events (including stroke and myocardial infarction), venous thromboembolism, and even dementia risk (Jehle et al., 2025). These protective effects were strongest for tadalafil, especially among men treated for erectile dysfunction or lower urinary tract symptoms. While traditionally framed around sexual health, its long-term vasodilatory and anti-inflammatory properties make it a promising agent for healthy aging.

I take 2.5mg a day, first thing in the morning with the bulk of my supplements and off-label drugs.

Telmisartan (variable dosing)

An angiotensin receptor blocker (ARB) with PPAR-γ agonist activity. Used to control blood pressure and support metabolic flexibility (Benson et al., 2004). Dosed to maintain ideal BP (110/70), especially in muscular individuals.

I personally measure my blood pressure weekly, and adjust the dosage to try to maintain around 110/70. Since I am a larger man with significant muscle mass, any lower and I run into some issues. Often I do not need to take any telmisartan, and only take it if my BP is creeping up.

5-Amino-1MQ (10–20mg/day)

A potent small molecule that inhibits NNMT, boosting NAD+ and enhancing metabolism, insulin sensitivity, and potentially body recomposition. Experimental—but promising (Babula et al., 2024; Kannt et al., 2018).

I am taking 20mg a day, first thing in the morning

Electrolyte Management

Proper electrolyte balance is fundamental to recovery, yet often overlooked. This becomes especially critical when using hormone replacement therapy or peptides, which significantly alter fluid and electrolyte dynamics. When using hormone replacement therapy (HRT) or peptides, adding approximately 1g/day of additional potassium and modest increases in sodium intake (primarily through lightly salting foods) helps prevent electrolyte imbalance, water retention, muscle cramps, and fatigue.

Personally, I take 1g of Potassium in the morning, and also let my body tell me if I need more. I keep bananas and coconut water in the house, and if they feel appealing to me, I consume them. For salt, I let that guide me, as well.

Hormonal and Peptide Protocols for Enhanced Recovery

Unlike hormetic stressors that deliberately challenge your system, these protocols are strictly regenerative, and designed to enhance the body's natural recovery processes. Ideally, doses of these various hormones and peptides are taken at levels to maximize your system to the upper end of what is healthy and normal for humans—not to exceed supra-human levels, which often come with deleterious downstream effects.

The following dosages are the ones that I personally take, optimized for my unique physiology based on extensive blood testing, and subjective monitoring of my performance and overall wellbeing. Exact dosing will vary greatly between individual needs.

It is also important to note that these doses often vary when significant changes happen in my lifestyle. This is why constant blood monitoring, as well as taking careful note of subjective well-being, is critical for each individual undertaking these performance and health-enhancing solutions.

Hormone Replacement Therapy (HRT)

Low-dose Testosterone Cypionate (100mg/week) & Primobolan (100mg/week): This combination supports accelerated muscular regeneration, reduces systemic inflammation, and improves protein synthesis. Unlike supraphysiological doses used in bodybuilding, these therapeutic levels enhance recovery without overwhelming the system.

A testorenote-replacement therapy (TRT) protocol is especially important for men the older they get. Additionally, the modern lifestyle and endocrine-disrupting toxins in our environment have led to substantially lower testosterone levels today than previously measured.

TRT can lead to substantial increases in quality of life, including desirable effects on mood, energy levels, sense of well-being, sexual function, lean body mass, muscle strength, erythropoiesis, and bone mineral density (BMD), cognition, and some benefits on cardiovascular risk factors (Bassil, Alkaade, & Morley, 2009). Additionally, testosterone replacement therapy has been positively linked to fat loss (Traish, 2014), and has also been shown to mitigate many age-related declines in male vitality in elderly cohorts (Rodrigues Dos Santos & Bhasin, 2021). It could be argued that a large majority of young men could benefit from testosterone therapy, as levels of testosterone have steadily, and significantly, declined over the last several decades, even when corrected for confounding variables such as excess body fat (Lokeshwar et al., 2021).

Adverse effects of testosterone replacement therapy are minimal to non-existent. Previous reports of various increased risks relied on poor methodology and have since been challenged (Morgentaler, 2016). A recent meta-analysis reports that harm evidence reported in the research is of insufficient or low certainty (Diem et al., 2020), while another review by Giovanni Corona and colleagues concluded that the cardiovascular risks often attributed to testosterone therapy stem primarily from a small set of observational and clinical trials with notable methodological limitations. When testosterone treatment is appropriately administered to patients with confirmed hypogonadism, no significant risks to cardiovascular or prostate health have been observed (Corona et al., 2017).

I highly recommend utilizing subcutaneous injections into the upper glute, 2-3x weekly, rather than intramuscular. You will need to ensure the oil carrier is compatible with this method, and also, the primobolan should not exceed 100 mg/mL concentration, otherwise it will lead to painful nodules. Subcutaneous injection reduces the accumulation of scar tissue, and also leads to slower absorption and distribution, meaning hormone levels will stay more consistent between doses (Piatkowski, 2025; Figueiredo, Gagliano-Jucá, & Basaria, 2022).

Enclomiphene *(25mg/day)*

Enclomiphene (25mg/day): Preferred over Clomiphene for its more selective estrogen receptor modulation, Enclomiphene supports endogenous testosterone production and fertility. Research in the *Journal of Clinical Endocrinology and Metabolism* demonstrates its efficacy in maintaining natural production alongside exogenous testosterone (Wiehle et al., 2014).

I personally take Enclomiphene daily, which helps keep natural testosterone intact while on 100mg/week of testosterone cypionate. I routinely measure my total testosterone in the ~9-11 ng/mL, the upper of what a young male athlete would measure. Incorporating enclomiphene keeps my follicle stimulating hormone and luteinizing hormones within the low-normal levels, maintaining fertility—and making it fast and easy to regain high fertility levels with a protocol shift.

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hCG (Human Chorionic Gonadotropin) – 500 IU every other day

When my total testosterone, or, FSH and LH fall lower than ideal, I incorporate hCG (500 IU every other day): This peptide preserves endogenous hormone production and fertility when using exogenous testosterone. By mimicking luteinizing hormone, hCG prevents testicular atrophy and maintains natural regulatory functions. It also plays a critical role in preserving fertility, testicular volume, and hypothalamic-pituitary signaling in men on HRT (Hsieh et al., 2013).

I opt to avoid chronic use of hCG as I typically see a considerable rise in aromatization, converting testosterone to estradiol, a potent estrogen—hCG can significantly increase estrogen levels, especially in individuals with higher body fat percentages, where aromatase activity is elevated (Swerdloff & Ng, 2023; Foster, 2022). When this is happening to a considerable extent, it becomes difficult to mitigate the high estrogen, even with added aromatase inhibitors. While on any TRT protocol it is important to routinely measure estradiol, as well.

I'll add that a much more powerful, but substantially more expensive, solution is HMG. HMG can be taken at shorter intervals to restore fertility and natural production of key hormones like LH and FSH, especially when hCG (human chorionic gonadotropin) doesn't get the job done. HMG supports testicular function by providing both FSH and LH activities, promoting testosterone production and spermatogenesis in a manner that aligns more closely with natural gonadotropin regulation than hCG alone (Wu & Sung, 2024; Ezcurra & Humaidan, 2014). It's not universally effective, but in many cases, including my own, it works where hCG fails. When I was trying to regain fertility, the combination of hCG and clomiphene had limited effect, stagnating at the low end of "fertile," with impaired testosterone, 6 months after initiation. However, within 10 days of starting HMG, my hormone levels rebounded dramatically, and my fiancée and I conceived roughly 90 days later; roughly the time for new sperm to reach maturation. That said, because of its high cost, HMG is rarely a first-line treatment, it's usually reserved for cases where other strategies don't work.

These tools aren't mandatory, and they're not for everyone, but for those operating at the edge of recovery—high-stress lives, aging systems, deep training blocks—they offer targeted physiological support when natural output falls short of the recovery demand. This isn't hormone chasing for vanity, this is rebuilding with intelligence.

Advanced Regenerative Peptide

Peptides offer precise signaling mechanisms to enhance specific recovery pathways:

Where traditional recovery focuses on inputs like sleep, nutrition, and time, peptides offer something more surgical: precision signaling. These molecules don't force adaptation, instead, they *guide* it by activating repair cascades, modulating immune response, and accelerating regeneration in ways nutrition and rest alone can't (Hao et al., 2024).

They're not a miracle, but they can be profoundly therapeutic and beneficial in your quest for accelerated recovery, delayed aging, and improved performance.

BPC-157

BPC-157: BPC-157 is perhaps the most well-known tissue repair peptide. Originally isolated from gastric juice, BPC-157 supports rapid healing in connective tissue, muscle, and even the gastrointestinal tract. It accelerates angiogenesis, improves collagen synthesis, and calms localized inflammation (Gwyer, Wragg, & Wilson, 2019). BPC-157 offers remarkable tissue regeneration properties, particularly for gastrointestinal healing and tendon/ligament repair. Studies show accelerated wound healing and reduced inflammation in multiple tissue types.

I personally administer BPC-157 in conjunction with TB-500 and TA-1 once or twice a year, or whenever my body is feeling especially run down. If dealing with an acute injury, I will turn to a cycle of BPC-157 and TB-500, specifically. BPC-157 is injected subcutaneously, although, I believe there is some evidence for oral administration, especially for gut health.

Thymosin Alpha-1 (TA-1)

Thymosin Alpha-1 helps the immune system do what it's supposed to, without doing too much. Unlike generic "immune boosters" that market stronger immune responses as a virtue (which, if they actually did this, would translate to more inflammation, worse symptoms, and a more rapid decline in immune function as we age), TA-1 modulates the system intelligently. It reduces unnecessary systemic inflammation while preserving immune vigilance, helping the body respond effectively without overreacting. Since the thymus is where our T-cells learn to respond to new threats, preserving its function isn't just about avoiding illness now, it's about long-term immune adaptability.

If you're in a recovery phase post-infection, post-stress, or post-injury, TA-1 helps bring the immune system back online without overactivation. It reduces systemic inflammation while preserving the ability to mount an adaptive immune response, which is essential during deep repair (Tao et al., 2023; Wei et al., 2023).

I cycle through TA-1 once or twice a year to keep my immune function optimized, preventing thymic involution. I inject subcutaneously, as is the protocol for the majority of peptides, but not all.

Thymosin Beta-4 (TB-500)

Thymosin Beta-4 (TB500): Specifically effective for soft-tissue and joint repair acceleration, TB500 enhances actin regulation and tissue migration. Recent research demonstrates its ability to accelerate recovery from connective tissue injuries by up to 60% (Goldstein et al., 2011).

I administer TB-500 via subcutaneous injection, the method that has the strongest evidence. I inject as close to whichever damaged tissue I am able to.

MOTS-c

This mitochondrially-derived peptide optimizes mitochondrial function, metabolic flexibility, and overall endurance recovery. Studies in Cell Metabolism show MOTS-c supplementation enhances recovery from metabolic stressors and improves mitochondrial adaptation (Kim et al., 2018; Lee et al., 2015). By now, you know how critical mitochondrial health is for our function, resilience, and longevity. I take my mitochondrial health seriously, which is why I utilize MOTS-c.

I cycle through MOTS-c at a dosage of 10mg/week, split into two subcutaneous injections, for 7 days. I do this once every few months.

Epitalon

Epitalon is a synthetic peptide derived from epithalamin, a naturally occurring compound secreted by the pineal gland. It has been studied for its ability to upregulate telomerase, an enzyme that maintains and extends telomere length (Khavinson, Bondarev, & Butyugov, 2003). Since telomeres shorten with age and cellular replication, preserving their length has become a focal point in the science of longevity.

In animal models and limited human trials, Epitalon has been associated with lifespan extension, improved sleep regulation via melatonin normalization, and markers of slowed cellular aging (Araj et al., 2025; Khavinson, 2002; Khavinson et al., 2000, Khavinson, Bondarev, & Butyugov, 2003). It appears to act, at least in part, by supporting circadian rhythm alignment and reducing oxidative stress at the cellular level.

I take Epitalon at a dose of 10 mg daily for 20 days, cycling it twice a year. Administration is via subcutaneous injection.

While the telomere effects are still being explored in human studies, the existing data, and its long-standing use in Eastern European longevity clinics, make it worth including in my semiannual peptide stack. For me, it's part of a broader protocol aimed not just at living longer, but living *better*, longer.

Optimized HGH and Peptide Protocol

If testosterone is the architect, growth hormone is the builder. It drives tissue regeneration, collagen synthesis, fat metabolism, and cellular turnover, but like any powerful tool, timing and precision matter more than volume.

Exogenous HGH has always led to substantial side effects for me, such as fatigue, high blood pressure, and abysmal recovery data; including elevated heart rate, frighteningly low HRV, and impaired deep and REM sleep. Motivated to resolve this challenge, I designed a low-dose morning protocol: starting at 1 IU per day and scaling up to 2 IU over several weeks to mimic natural circadian rhythms and avoid the downsides of supraphysiological dosing: edema, insulin resistance, circadian disruption (Cummings & Merriam, 2003; Yuen et al., 2002). Concurrently, in the evenings I was utilizing CJC-195 and Ipamorelin in order to stimulate natural production of growth hormone as I slept. The idea was simple: to restore, supplement, stimulate, and not override or replace. For a while, it worked.

However, eventually, the side effects crept in: water retention, joint stiffness, headaches, atrocious recovery data, and disrupted sleep. No matter how much potassium and magnesium I consumed, I found no relief. I've since come to accept that, at least for me, exogenous HGH doesn't deliver a net benefit, so I no longer include it in my protocol. That doesn't mean it has no place in a recovery or longevity stack, but it means that precision, self-monitoring, and biochemical individuality matter more than hype or population-wide data.

Cognitive and Neurological Recovery Enhancement

Cognitive stress isn't always loud. It doesn't always leave soreness, or swelling, or obvious fatigue, but it burns through neurotransmitters, taxes memory circuits, and depletes attention like a muscle group that's been worked to failure.

If you're doing deep creative work, decision-making under pressure, high-stakes problem solving—or just operating in a world that pulls your attention in a hundred directions—your brain needs a recovery protocol too.

This section isn't about overclocking cognition. It's about restoring balance after sustained mental output.

Tesofensine *(500 mcg/day)*

Tesofensine, which is a triple reuptake inhibitor for serotonin, dopamine, and norepinephrine, began its journey as an experimental drug for Alzheimer's and

Parkinson's, yielding significant, albeit clinically limited, results in phase II clinical trials (Axel, Mikkelsen, & Hansen, 2010; Lehr et al., 2010; Astrup et al., 2008b). These trials revealed an unexpected "adverse side effect": participants lost considerable amounts of weight, particularly those who were overweight and obese. (Seeing as how these trials were in models of cognitive decline, many of these patients were bound to be elderly, frail, and already prone to malnutrition. Further weight loss, if the weight loss includes lean body mass, exacerbates frailty, increases morbidity risk, and negatively affects their quality of life, making this an undesirable outcome. The studies did state that participants who were overweight or obese had more substantial weight loss, however, no analysis was done regarding what type of weight was lost, namely, adipose tissue or muscle mass).

Research direction quickly pivoted, with the pursuit of tesofensine for AD and PD being discontinued, and trials targeting obesity beginning. An impressive phase IIb clinical trial saw substantial effects, with an average of 9kg weight loss over 24 weeks (Astrup et al., 2008a). Importantly, tesofensine works in a manner that is not similar to the popular class of GLP-1 mimetics like semaglutide, altering the reward center in the brain rather than artificially driving a feeling of "fullness" (Perez et al., 2024). This alteration changes a food addict's emotional drive towards unhealthy eating habits, assisting to break the addictive reliance, rather than simply mitigate the volumes. For this reason, tesofensine could also be promising for other addictive behaviors, whether it be alcohol consumption, illicit drugs, gambling, or any other emotional crutch that ails an individual (McMillen et al., 2007).

A prevailing notion exists for many that they immediately reject any drug or supplement with the capability of altering neurotransmitter levels. The thought goes that altering mood, and potentially personality, changes who we are. That's true, but so does starting an exercise protocol, adjusting our sleep schedule, or picking up a new hobby. Stagnation is almost impossible—we are constantly changing and adapting to our environment and the experiences it provides. That goes for our body, and also our mind. Drugs like tesofensine are powerful because the change becomes *intentional*. Rather than being a passive observer in life, allowing the chaos of your surroundings to shape you, options exist to utilize tools like this to create the change you desire. There is nothing embarrassing about that, nothing warranting stigma. What we should be ashamed of is the crippling insecurity that leads us to the belief that we should be embarrassed to improve, to grow, and to relentlessly pursue who we want to be, not who we currently are.

Personally, I take 500 mcg a day of tesofensine, and my urge to consume alcohol has all but disappeared. My cravings have changed, too. While still imperfect, I have less of a desire to eat many of the calorically dense "comfort foods" I typically turn to in times of heightened emotions. Stress, happiness—it didn't use to matter. If anything in my day drove me to deviate from the norm, I turned to food, whether to celebrate or to comfort myself. This reliance is now all but broken, thanks in large part to tesofensine.

Peptide Stack for Neural Repair

Each of these targets a specific aspect of brain recovery:

- **Semax**: Increases BDNF, supports executive function and focus under stress (Gusev et al., 2018).
- **Selank**: Regulates anxiety through GABAergic and serotonergic pathways (Vyunova et al., 2018).
- **Dihexa**: Enhances synaptogenesis and neural plasticity, potentially improving learning and memory consolidation (Wright & Harding, 2015).

I noted above the double standard with our conception of improving the mind as compared to the body, and how we define what it means to be "us." This stigma extends to interventions intended to replenish what our mind needs to perform, as well, but to a lesser extent. If you mentioned to even the most dogmatic anti-supplement proponents that you are supplementing with, say, iron or vitamin D due to low serum measurements, they would almost be guaranteed to give you a pass. Same goes for electrolytes during increased demands, such as exhaustive exercise. Jump into the performance category, and mention you are taking creatine, or beta-alanine, or other clinically validated ingredients and they may raise an eyebrow, but they're still unlikely to ridicule you.

Shift to supplements for the brain, such as various B vitamins with the express purpose of replenishing neurotransmitters, and even many normal supplement users will think you're too obsessed. Now, move to peptides with the intent purpose of improving brain function, and few outside the biohacker and longevity space will hesitate to judge, let alone demonstrate any interest. This is tragic, as we are the totality of our being; our mind, body, and spirit, or rather, the purpose that drives us forward. To be truly strong, and impactful to those around us, we need to be complete. I detail this rationale in the epilogue, but for now, if you were previously wary of supplementing the mind, I hope by the end of this book you will alter your perspective. We need as many people as sharp and focused as possible, to drive the future our future progeny deserve.

Complementary Cognitive Supplements

Several compounds provide powerful cognitive recovery support:

Not every recovery needs calls for a peptide or prescription compound. Some of the most effective tools for cognitive reset come from well-studied, naturally derived compounds—quiet, reliable allies in the background of your brain's repair cycle.

These aren't "productivity boosters." They're recovery amplifiers: neurochemical support systems that smooth the descent from high-load mental stress, helping recalibrate emotional stability, cognitive clarity, and neurotransmitter balance.

If you're using neuropeptides like Semax, Selank, or dihexa alongside these compounds, monitor for symptoms like headaches or dizziness, and adjust accordingly. Stacking the wrong tools too aggressively can overstimulate neural circuits when you're trying to calm them.

Here's what I personally take before bed, most nights:

- **5g glycine –** promotes sleep by shortening sleep latency, regulating body temperature and modulating neural activity (Kawai et al., 2015)
- **440mg tryptophan –** serotonin precursor, reinforces natural sleep cycles (in times of high stress, I have taken this in the morning)
- **3mg melatonin –** keeps circadian signals aligned
- **100mg L-theanine –** reduces anxiety and smooths mental chatter (I take this in the morning [sometimes] if my various neurological protocols have me firing too hot or waking up with my mind racing; I often get headaches, and morning theanine and some of the others on this list prevents this. I always take it at night)

This stack isn't sedating, it's stabilizing. It supports natural neurotransmitter cycling without shutting the system down. And it's modifiable depending on your stress load, peptide use, and recovery state.

Here's what works.

Apigenin (~50 mg, evening)

Apigenin (~50mg evening): Found naturally in chamomile and parsley, apigenin supports neurogenesis, provides anti-anxiety effects, enhances cognitive clarity, and promotes neuronal recovery through GABA modulation (Olasehinde & Olaokun, 2024; Gao et al., 2023).

The timing for agipenin really depends. Originally, I was taking it in the morning as I was waking up wired with a headache. Now that I am in a constant state of sympathetic stress, due to having a 3 week old infant with colic, I take it in the evening.

Tyrosine (~500–1000 mg, morning)

Tyrosine (~500-1000mg morning): As a precursor to dopamine and norepinephrine, tyrosine supports neurotransmitter synthesis, enhances cognitive performance under stress, and accelerates recovery from cognitive demands (McAllister et al., 2024).

I specifically take 500mg because going to 1000mg gave me headaches, from my recollection.

L-Theanine (~200 mg, morning)

L-Theanine (~200mg morning dose): An additional morning dose supports cognitive function, reduces anxiety, and promotes clarity without sedation, making it particularly effective for recovery from mental stressors.

Often paired with caffeine, Theanine works just as well—if not better—as a standalone cognitive stabilizer during recovery. It promotes calm alertness, reduces cortisol, and protects against overstimulation without impairing clarity (Giles et al., 2017).

I take 200mg in the morning and 100mg in the evening.

Phosphatidylserine (~300–600 mg, evening)

Phosphatidylserine (~300-600mg daily, evening): This phospholipid lowers cortisol levels and supports hormonal regulation, improves memory and enhances cognitive recovery, especially during periods of high stress or demand (Richter et al., 2013; Starks et al.,2008).

Same as agipenin, I now take phosphatidylserine in the evenings, compensating for the same chronic stress I am enduring with a newborn, but I was previously taking it in the mornings—and hopefully will again soon. Context matters.

Alpha-GPC (~300–1200 mg, morning or pre-cognitive effort)

Alpha-GPC (~300-600mg morning/pre-cognitive load): By enhancing cholinergic function, Alpha-GPC improves cognitive clarity and memory recall during recovery phases (Sagaro, Traini, & Amenta, 2023). Research demonstrates its ability to enhance acute mental performance and speed up recovery of cognitive function following neurological stress (Kerksick, 2024; Barbagallo Sangiorgi, 1994).

Supplement manufacturers recommend 300-600mg of Alpha GOC per day, which is what some research on improving exercise performance has utilized, however, the largest clinical studies on cognitive decline often use up to 1,200mg daily. Personally, I'm splitting the difference, supplementing with 600mg in the morning, and 300mg in the evening. This is above the lower end, as I believe my cognitive demands exceed that of the typical person, but lower than the upper end as I'm not treating cognitive impairment. I'm just aiming for a maintenance dose that keeps me sharp, without overdoing it.

Each of these compounds supports a different axis of neurological recovery. You don't need them all, and you don't need them always, but when used strategically, they can reinforce the brain's own repair systems and help you come back sharper, sooner, and more grounded.

Recovery is not just how you sleep. It's how you think the next morning.

Regenerative Medicine and Biological Therapies

Sometimes, the substrates and building blocks we need to regenerate and heal cannot be administered via pill, powder, or even intramuscular or subcutaneous injection. Sometimes, they require more advanced delivery methods, intravenously or deep into our joints, necessitating qualified medical supervision.

Stem Cells, Exosomes, PRP, NAD+, and Ozone

Intravenous administration of stem cells, exosomes,[9] ozone therapy, NAD+, and targeted PRP injections provides comprehensive support for systemic rejuvenation, reduced inflammation, and tissue regeneration. These therapies deliver concentrated signaling molecules and regenerative factors either directly to damaged tissues, such as via targeted injection into a joint cavity, or systemically through IV infusion, creating an optimal environment for repair.

PRP (Platelet-Rich Plasma) concentrates your own body's growth factors and delivers them exactly where healing is needed: tendons, joints, ligaments (Jain & Gulati, 2016).

Exosomes act like encrypted messages between cells, triggering tissue-specific repair cascades (Zhao et al., 2020).

Stem cell infusions may help systemically restore inflammation resolution and repair capacity, though the quality and source of cells is a critical variable (Epstein, Lipinski, & Luger, 2018). Intravenous administration is common for systemic effects but results in pulmonary trapping and limited redistribution, while targeted injections, such as intra-articular or intrathecal delivery, maximize local concentration for tissue-specific regeneration (Acharya et al. 2025, Fischer et al., 2009). Beyond this, stem cells also contribute to immunomodulation, angiogenesis, and the regeneration of damaged or senescent tissues through paracrine signaling (Han et al., 2022; Baraniak & McDevitt, 2010). Their secretome, which is rich in

[9] While the term "exosome" is widely recognized and commonly used in clinical and commercial contexts, its unqualified usage has drawn increasing scrutiny from the scientific community. The International Society for Extracellular Vesicles (ISEV) has issued updated MISEV guidelines cautioning against the indiscriminate use of "exosome," recommending instead the broader and more accurate term extracellular vesicles (EV), unless rigorous and validated methods have been used to confirm endosomal origin. Many commercially marketed "exosome" therapies may contain a heterogeneous mix of vesicle types or even non-vesicular contaminants due to current limitations in isolation and characterization techniques. Moreover, safety concerns persist, particularly with human-donor-derived exosome products used in unregulated aesthetic treatments, raising ethical and biomedical red flags in jurisdictions with lax oversight. Despite this, the term "exosome" continues to be used here for accessibility, though readers and practitioners should remain cautious and demand transparency regarding the biological identity, purity, and quality control of such interventions.

growth factors, exosomes, and anti-inflammatory cytokines, can influence systemic healing responses, support neurogenesis, promote vascular repair, and even modulate metabolic function in aging or chronically stressed systems (Zhidu et al., 2024; Li et al., 2023; Zhang et al., 2022; Konala et al., 2016).

NAD+ IVs are widely marketed as mitochondrial restoration therapies, but the actual clinical evidence behind them is underwhelming. A recent systematic review found only *speculative* benefits for select conditions, such as psoriasis and muscle performance, and a placebo-controlled randomized controlled trial (RCT) showed no difference in vital signs between NAD+ IV, NAD+ oral, and plain saline (Hawkins et al., 2024; Radenkovic, Reason, & Verdin, 2020). So why are they everywhere? Because once one clinic added NAD+ IVs and marketed them as essential, others had no choice but to follow suit; otherwise, customers would start to assume something was missing. It's like a crowded movie theater: if someone in the front row stands up, everyone behind them has to stand just to see the screen. In this case, it's not about what's best, but what's expected.

Ozone therapy is controversial but mechanistically interesting—enhancing oxygen utilization and modulating immune signaling (Valdenassi et al., 2016; Bocci, 2007). As a hormetic intervention, it fits the mold perfectly: a reactive oxygen species used in small, controlled doses to provoke adaptive healing responses. Recent small-scale RCTs have reported promising outcomes for conditions ranging from chronic pain to autoimmune disease (Arjmanddoust, Nazari, & Moezy, 2025; Cabioglu et al., 2023; Sconza et al., 2023; Machado & Contri, 2022; Andrade et al., 2019). That said, the overall evidence base remains limited and carries risk of bias. I'm especially cautious about claims that ozone enhances other regenerative therapies like stem cells. While the logic is appealing, both appear to promote repair, there's no published data supporting synergistic or complementary use. In some instances, two therapies that appear complementary on the surface could in fact interfere with each other. Until combination trials emerge, any claimed synergy is speculative at best.

My personal interest centers on the trio above (PRP, exosomes, stem cells), however, virtually every cutting-edge clinic today incorporates NAD+ and ozone into their regenerative protocols. They've become staples, not necessarily because their evidence is strongest, but because the mechanisms are compelling and consumers will almost exclusively opt for protocols with additional marketed treatments over those with less.

Autophagy & Senescent Cell Clearance Protocols

While these protocols induce acute stress, they differ fundamentally from traditional hormetic stressors. They create short-lived stress specifically to facilitate significant cellular repair, rejuvenation, and renewal—functioning more as "deep cleaning" than progressive adaptation triggers.

Not all stress leads to growth. Some stress, if applied briefly and precisely, is used

not to adapt, but to *clean house*. These protocols are different from the hormetic tools in this book. They're not meant to build toughness, they're not progressive, they don't scale.

As I've described in this book, autophagy is the body's way of breaking down old or damaged cell parts and reusing them. Mitochondria, which are tiny structures inside most cells that help make energy, can be cleared out and recycled during this process. In fact, the significance of autophagy was globally recognized in 2016, when the Nobel Prize in Physiology or Medicine was awarded to Yoshinori Ohsumi for his pioneering discoveries uncovering the molecular mechanisms behind autophagy. His research demonstrated how nutrient starvation triggers this self-cleaning process in yeast cells, laying the foundation for understanding how our bodies recycle cellular components to maintain health and resilience (Nobel Prize Outreach, 2016). Used infrequently but strategically, interventions promoting autophagy can dramatically increase recovery efficiency, metabolic resilience, and systemic clarity.

Weekly Autophagy Activation Protocol

This protocol combines three powerful autophagy activators on the same day, weekly:

This protocol induces a transient cellular stress signal, turning on internal recycling systems and supporting long-term metabolic rejuvenation.

It combines three compounds into a single *autophagy reset day* each week:

Rapamycin (5–7 mg, once weekly)

Inhibits mTOR, triggering deep autophagy and the clearance of damaged proteins, organelles, and dysfunctional cells (Civiletto et al., 2018; Ravikumar et al., 2006).

Metformin (500 mg)

Activates AMPK, improves mitochondrial efficiency, and supports autophagy. When used with rapamycin, the effect is synergistic, not simply additive (Ma et al., 2023; Bharath et al., 2020; Wang et al., 2019).

24-Hour Fast (water-only or with electrolytes)

Completes the trifecta by suppressing insulin, deepening the autophagic state, and enhancing metabolic flexibility (Washburn et al., 2019).

Following this intense autophagy protocol, enhanced hydration, electrolyte replenishment, quality sleep, and gentle recovery activities are essential to maximize benefits while minimizing stress impact. In short, this combination produces a powerful cellular spring cleaning effect by removing waste without damaging functional tissue.

This is why post-protocol support is critical:

- Increase hydration
- Replenish electrolytes
- Eat nutrient-dense, anti-inflammatory meals
- Avoid training for 24–36 hours
- Prioritize deep sleep and circadian alignment

Think of this like *internal housekeeping*: not exciting, not heroic, but essential.

Quarterly Senolytic Protocol

Senescent cells are like toxic coworkers: no longer doing their job, and actively interfering with the people who are. Clearing them out is essential for restoring signal clarity, especially as we age or accumulate chronic stress (Sun, Li, & Kirkland, 2022).

This protocol is run once every 3–4 months. It's brief, intense, and highly targeted.

Fisetin (1–2 g/day for 3 days)

Flavonoid shown to trigger apoptosis (programmed death) in senescent cells. High doses needed for senolytic effect—this is not a daily supplement (Zhu et al., 2017).

Quercetin (500–1000 mg/day for 3 days)

Works synergistically with Fisetin to enhance senescent cell targeting and improve systemic inflammatory resolution (Wang et al., 2021; Zhu et al., 2017).

Dasatinib (50–100 mg/day for 1–3 days) (optional)

Pharmaceutical-grade senolytic. Extremely potent. Requires medical supervision and careful post-protocol support. May not be suitable for everyone (Xu et al., 2018; Zhu et al., 2015).

Acute stress is part of the process. During and immediately after this protocol, it's common to experience temporary fatigue, mental fog, or muscular soreness, which are signs your system is integrating the challenge. You may notice increased thirst as your body works to flush out metabolites and manage fluid balance. Don't be surprised if your training capacity dips for a few days; it's a natural rebound effect while resources are redirected toward recovery and repair. This isn't backsliding, it's rebuilding.

This quarterly protocol requires significant recovery support afterward, including increased hydration, nutrient-dense meals, temporary reduction in training intensity, and prioritized sleep for 3-5 days following completion

Plan your calendar accordingly. This is not a protocol to run during high-output weeks.

After the protocol, the emphasis shifts toward deliberate recovery. Prioritize deep, restorative sleep to consolidate adaptations and rebuild. Replenish electrolytes through hydration, food, or supplementation. Choose protein-rich meals to supply the raw materials for muscle regeneration and immune support. Perhaps most importantly, don't stay still; light movement such as walking, as well as sauna, and breathwork, helps flush metabolic byproducts, maintain circulation, and ease your system back into homeostasis.

These protocols are not for daily use. They are periodic interventions—like changing your oil or upgrading your firmware. They don't make you stronger, they make you cleaner, and sometimes, that's what recovery really needs.

Conclusion: The Personalized Recovery Framework

While hormetic stressors provide the stimulus for adaptation, these advanced recovery protocols create the optimal environment for those adaptations to fully manifest. By strategically implementing personalized, strictly regenerative recovery techniques, you can dramatically enhance adaptation, systemic rejuvenation, and long-term functional improvements. These methods represent the complementary half of the hormetic equation—where stress provides the signal, but recovery delivers the results. As with all aspects of resilience building, individualization remains paramount. Start with foundational recovery techniques and progressively incorporate these advanced methods as your experience and understanding deepens.

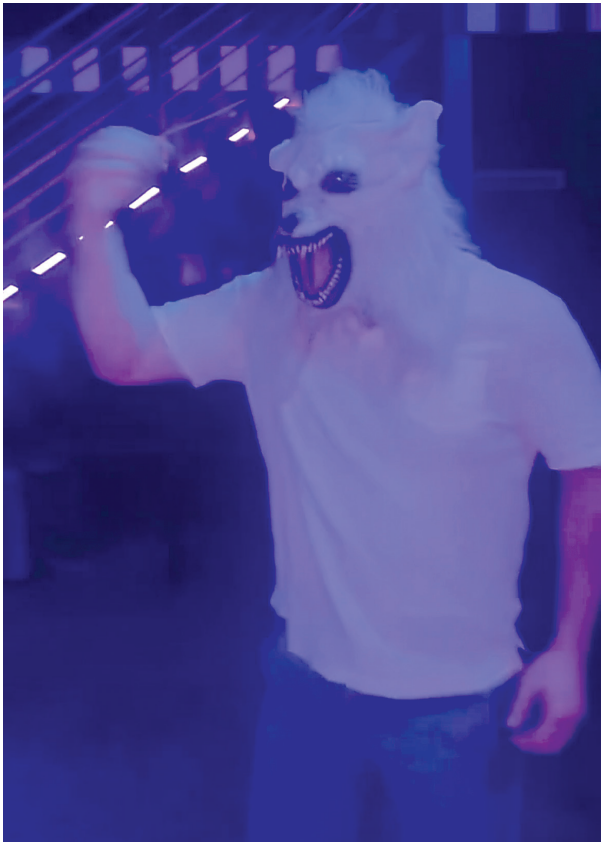
When used strategically, these protocols act as accelerants for the body's repair and renewal systems. They help regenerate tissue; they don't just do this superficially but deep within joints, muscles, and connective structures. These protocols support mitochondrial repair, restoring the engines that power every cell. If used correctly, your mental fog lifts, and it's replaced by sharper cognitive clarity. Hormonal systems stabilize by smoothing out erratic fluctuations that otherwise compromise energy, mood, and performance. Over time, these effects compound, and they build a more resilient baseline that's not just reactive, but robustly prepared for the next challenge.

Here's an important principle to think about, however: not every advanced recovery tool is meant for you—not yet, at least. What does recovery mean? It's not about chasing the latest biohack or stacking protocols on top of a shaky foundation. Rather, recovery is a layered system, and like any structure, it needs to rest on solid ground. That means starting with the basics. Sleep isn't just a nightly reset, it's the cornerstone of repair. In practical terms, this means that electrolyte balance sustains cellular function and keeps the nervous system firing smoothly, all while deep nutrition fuels regeneration at every level; and, breath and stillness give the body

space to recalibrate. These aren't optional add-ons but the base layer on which everything else depends.

Then, as your stress load increases and your capacity deepens, you can begin to scale recovery in proportion. Add peptides, introduce mitochondrial support, cycle in autophagy protocols, and layer as needed. This isn't a prescription. It's a menu, and it's up to you to choose wisely—and test carefully. Because in the end, recovery isn't passive. It's not rest; it's a skill, a discipline, and a form of *engineering*. Stress is your catalyst. Recovery is what makes it real.

Picture 35. Small rock concert in Austin, TX. Three days before recording The Skinny Confidential.



Perhaps the band giving me their werewolf mask was a sign that the night was getting a tad bit out of control.

CHAPTER 12:

Measuring and Tracking Your Progress

Introduction: Why Measurement Matters for Hormesis

"*Man is the measure of all things*," Protagoras wrote (Bonazzi, 2024).

On the surface, this idea sounds empowering. Human perception serves as the reference point, and reality is inherently contextual. This thought confronts the stark and unforgiving truth of our physiology when entering the realm of stress and adaptation, and its limitations become obvious, because if *man is the measure*, then the measure had better be precise.

I'm reminded of a dose of realism I was once doused in, which forever changed my perspective. I was watching the UFC fights in my boss and mentor-at-the-time's living room, conversing with some of my colleagues and one of his acquaintances. I remarked on some new endeavour I was attempting, or some skill I was trying to learn. I can't exactly remember what it was, but that isn't the point. When questioned on whether I knew what I was doing on this new project, I remarked, "*Anything worth doing is worth doing poorly, until you learn to do it well*." A piece of motivational philosophy I had picked up from Zig Ziglar, and one which I often think back to, despite its limitations. My boss's friend leaned over and said slowly, "*That strategy would be ill advised if you're learning to rewire your home, or a broken dryer*." Everyone laughed, and I thought about what he'd said, not because I was upset, but because it was true.

There are certain tasks that are inherently dangerous, period. Doing them poorly for the sake of some notion of optimism would be foolish and potentially disastrous. Many hormetic stressors fit into this camp, although the disaster you may subject yourself to through improper dosing and recovery is likely milder than if you shock yourself to death, or burn your house down from shoddy electrical work. Trusting your instincts alone might work in calm conditions, but under pressure, it's a gamble. This becomes especially true when you start walking the line between growth and collapse.

Intentional stress undertaken to drive adaptation—scientifically named *hormetic stress,* as you know by now—is a form of challenge designed to build capacity. Cold, heat, hunger, exertion: these aren't just discomforts, they're *signals*, sharpening tools, and catalysts for adaptation. However, there's a fine line between heroic and reckless, and between resilience-building and breakdown, and that's where the Greeks come in.

In the epic tales of Homer—*The Iliad*, *The Odyssey*—heroes are tested through ordeal. They suffer, endure, grow, and, if they're lucky, return stronger. These journeys were long, chaotic, and full of missteps. In the end, what defined the hero

wasn't just how much he endured, but rather, it was how well he *navigated* the stress and how well he recovered, and the substance of the lessons he brought home.

Hormetic stressors, in that sense, are *Homeric* in spirit: they push us into trials meant to transform, but unlike myth, your body doesn't come with a built-in narrative arc. There's no chorus, and there's no divine intervention when you've gone too far. The difference between a hormetic path and a Homeric collapse is dosage, timing, and self-awareness. The idiom "if you fail to plan, you plan to fail" rings especially true for hormesis. Also true always, but more so with hormesis: the plan needs to change on a moment's notice, when the situation changes.That means measurement, and not just once in a while, but always—and as much as your resources and capacity allows.

Without feedback loops (objective and subjective) you can't know whether your cold exposure is priming your immune system, or whether it is wrecking your sleep. Whether your training is driving strength and growth, or acting as a path to hormonal sabotage. The dose makes the poison, but with hormesis this is a constantly moving target, and you won't see the poison coming if you aren't tracking the effects of the dose in real time. In the world of hormesis, where the whole point is to introduce controlled stressors to drive adaptation, measurement becomes the difference between progress and burnout.

Perception, intuition and feeling go a long way in guiding your routine, and sometimes, they are all we have to go off. Measurements act as that safety net, for when your hunch is wrong. Measuring helps you make changes before you feel the ill effects, and it helps you reverse the course before you've crashed. Sometimes, the crash isn't sudden, it's gradual, like a frog slowly being boiled alive. Don't be the frog, don't slowly get used to your dangerous situation until it's too late. Measure, meticulously, and jump the hell out of the pot if the water temperature starts rising.

Hormesis is dose-dependent by definition. The same cold plunge that enhances immune resilience today might suppress thyroid function tomorrow if the context shifts, such as if you're under-slept, under-fed, or recovering from another stressor. While you might feel alert and energized, your biomarkers might tell a very different story: elevated cortisol, reduced HRV, declining sleep quality. That's why tracking in this day and age is not optional for anyone taking this seriously, it's foundational.

In this short chapter, I'll walk you through systems designed to quantify your adaptation curve, linking stress inputs to recovery outcomes. I'll combine objective data—sleep and recovery metrics, blood work, and urine analysis—with subjective metrics like focus, mood stability, training performance, and emotional regulation. This isn't about obsessive self-surveillance or data for data's sake. It's about pattern recognition: the ability to see, clearly and consistently, how your body and mind are responding to the loads you're placing on them.

Because without data, stress is noise. With data, stress becomes a signal. The goal isn't to optimize every variable; rather, it's to build a system that keeps you aligned with the process of adaptation, so that every stressor you choose contributes to resilience, not erosion. Because tracking isn't a crutch, it's the compass.

Wearable Technology

In 2019, I set out to investigate which of the leading wearables was the most accurate. I wore multiple devices for an entire month, diligently and meticulously tracking their accuracy through a series of both known measurements, such as distance walked or steps taken, as well as subjective ones, such as matching my sleep and recovery data to my subjective energy levels and overall physical state. Oura came out on top at the time, and it wasn't close. In addition, the bands on the WHOOP and the Biostrap caused substantial skin irritation for me, leading to raw and painful wrists. For those reasons, I have stuck with the Oura ring ever since.

A lot can change in 6 years, especially considering technological devices, and the technology and algorithms utilized to determine readings. I cannot honestly state that Oura is still the best, I would need to redo the experiment—a task which I lack the time, and motivation, to undertake. Many of Oura's competitors measure the same markers, and these are the markers I monitor with my Oura.

Biomarkers for Tracking Hormetic Adaptation

Cardiovascular and Autonomic Nervous System Markers

Heart Rate Variability (HRV)

HRV represents the variation in time between consecutive heartbeats and serves as perhaps our most accessible window into autonomic nervous system balance and overall resilience. Decreasing HRV typically indicates the accumulation of stress and insufficient recovery, while increasing HRV suggests enhanced resilience and adaptation (An et al., 2020; Roberto, Di Ionna, & Cavezzi, 2018).

High-quality HRV measurements reveal:

- Sympathetic vs. parasympathetic balance
- Recovery status from previous hormetic exposures
- Readiness for new hormetic challenges
- Long-term trends in autonomic nervous system adaptation

Most wearables now provide reliable HRV metrics, with morning readings offering the most consistent baseline. Tracking morning HRV trends over weeks and months can reveal whether your hormetic practices are enhancing or diminishing your resilience.

Resting Heart Rate (RHR)

While less sensitive than HRV, resting heart rate provides valuable complementary data. A decrease in your RHR over several months generally indicates improved cardiovascular efficiency and enhanced recovery capacity. Conversely, an elevated RHR (5+ beats above your baseline) suggests accumulated stress and insufficient recovery between hormetic exposures (Williams et al., 2017; Bosquet et al., 2008).

Sleep Tracking

- Deep sleep duration and percentage
- REM sleep patterns
- Sleep latency (time to fall asleep)
- Sleep efficiency
- Respiratory rate
- Body temperature variations

The Oura's "Readiness" score provides an algorithmic assessment of recovery status, helping determine when your system is prepared for new hormetic challenges versus when additional recovery is needed (Altini & Kinnunen, 2021).

"Stress" Tracking

I have been using the "Stress Tracking" feature through Oura since they launched it, and so far, it has been startlingly accurate, albeit delayed in uploading (typically 30-60 minutes behind). From Oura's website they state:

> *Previously, we have been able to measure stress through nighttime heart rate variability (HRV). Now we have a new ability to measure near-real-time stress and recovery using daytime HRV, heart rate, temperature trends, and accelerometer data to power the Daytime Stress feature and provide ongoing insights.* (Oura Team, 2024)

Blood Pressure Variability

Regular blood pressure monitoring can reveal how different hormetic practices affect your cardiovascular system. Track morning readings alongside evening measurements to assess how specific stressors impact your system and how effectively you recover (Kuksa, Shibaev, & Isaikina, 2019; Roeser et al., 2012). Because of this, evening monitoring may offer a more reliable snapshot of baseline physiological state and serve as the optimal window for detecting stress sensitivity, guiding recovery strategies, and timing interventions aimed at reducing cardiovascular risk. Personally, I only measure weekly, although I know others who measure as frequently as twice a day.

Recently, I have been seeing advertisements for a new wearable continuous blood pressure bracelet. I haven't purchased it yet, but I intend to, and I'm excited to explore the new data that will become available.

Blood, Urinary & Saliva Tests

I personally use Siphox for my blood testing, purchasing extensive quarterly tests, and I have begun taking additional annual tests for specialized measurements, such as my biological age and daytime stress range. This is substantially more intensive than most, however, substantially less than some others. For instance, some acquaintances of mine, such as Siim Land and Joe Cohen, hold retreats in India where they affordably mega-test their blood. In India, this can be done far more affordably than in the USA or Europe, and from my understanding, it almost leads to a "free trip," at least if you intend to conduct this battery of tests in the USA. To put it another way: if the combined cost of your flight, accommodations, and testing in India totals $10,000, and the same suite of tests would cost you $10,000 back home, then either the testing is free or the travel is. You're getting both for the price of one, so while technically you're still spending money, it's a net neutral exchange compared to domestic pricing, with the bonus of a trip attached.

In my quarterly test, the following measurements are taken:

- Liver Health: ALT, AST, Total Bilirubin
- Kidney Health: BUN, Creatinine, Cystatin C, eGFR

Heart Health: APOA1, APOB, HDL, LDL, Lipoprotein(a), VLDL, Total Cholesterol, Triglycerides, ApoB:ApoA1 ratio, Total Cholesterol:HDL ratio

Metabolic: Albumin, Morning Cortisol, Fasting glucose, HA1C, Insulin,Triglyceride:HDL ratio

- Thyroid: Free T4, Free triiodothyoronine, Free T3, TSH, TPOAb
- Hormonal: DHEA-S, Estradiol, FSH, LH, FAI, Prolactin, SHGB, Free Testosterone, Total Testosterone, Testosterone:Cortisol ratio
- Inflammation: hsCRP, Homocysteine,
- Nutritional: Vitamin D, Ferritin (iron)
- Prostate: Prostate-specific antigen

Additionally, if my fasting glucose is outside of the ideal range, I will test fasting glucose at home every 2-3 weeks as I make adjustments and attempt to drive it down.

When fasting, I utilize ketone urine kits to track ketone levels, along with fasting glucose, to ensure I am both in a safe range and am entering into ketosis. In the

future, I intend to add a continuous glucose monitor. Many friends and acquaintances have incorporated them, and I'm excited to play with them. Last I checked, I could not purchase one in Canada without a doctor's prescription for diabetes, so I will need to wait until this changes or until I depart the country.

I currently incorporate the Vivoo urine test strips, which measure a number of markers. Vivoo measures protein excretion, urine pH, hydration levels, ketones, calcium, magnesium, sodium, vitamin C, and oxidative stress (MDA). As of the last test, my "Wellness Score" is 10/10, receiving a "perfect" score in each measurement. Using the tests allows me to adapt my supplementation as my lifestyle and stress levels change.

Journaling

Finally, I advise those, especially those starting out, to keep a journal and track changes in their mood, energy, and performance. This is subjective, however, with careful thought and honesty, these observations can be illuminating. I used to extensively journal, however, I have mostly stopped the practices. There are numerous validated questionnaires for all sorts of outcomes, from day to energy, to fatigue, to sleep, to pain, that can be downloaded online. Alternatively, your own questionnaires that resonate with you, and your goals, can be manually created.

Conclusion: If You Can't Measure It, You Can't Master It

Without measurements we cannot reasonably conclude if our protocols are leading to improvement, decline or stagnation. Importantly, improvement in one area may lead to decline in another. By extensively tracking changes to our biomarkers, measurements, and our subjective existence, we can slowly adjust, tweak, and adapt our protocol to harmonize. The goal is improvement, and without measurement, we are shooting blind at a moving target—an all but impossible task.

CONCLUSION:

The War on Physical Sovereignty

This book was written at two different stages of my life. I wrote the original content for the most part in 2018 and 2019, post-collapse. This was shortly after the period of my life when my body was in the worst shape it had ever been, and my mind and spirit were degenerating. I was angry—angry at the cards I'd been dealt regarding my health, angry at the declining state of our society and the opportunities afforded to an entrepreneur without a trust fund, and I was drowning these angers in copious amounts of alcohol, but you already know that. This was written in the brief moments of clarity I felt as I was trying to find a path forward, a way to improve my body and to become who I believed myself to be.

So, I wrote a blog series tackling these topics. It was dry, overly technical, and lacking much of the personal narrative and reflection that now inhabit these pages. It was read by a few hundred—tops—and accomplished what I needed: I wrote the series to educate myself on the topics, so that I could channel these strategies to my own ends, to recapture my physical sovereignty. Many of these strategies gave me profound benefits, vastly improving my health, but still, I was not whole.

As my health returned, my mind became sharper, but I squandered this opportunity; with booze, the cynical derision of others, and extracurricular projects that would start, stall, and stumble. My mind was working better as my body became stronger, but I continued to sabotage it with my own personal insecurities, resentments, and emotional addictions. I kept it together to keep my business on track, publish the odd paper, but my full potential was nowhere close to being realized.

Then, something happened: a few lucky breaks, or rather, fortunate decisions and timing on the rewards I reaped for past work, all happened in succession. My fiancée found out she was pregnant, something we had been trying to achieve for the better part of a year, the delay mostly due to the extended time it took me to regain fertility after discontinuing testosterone therapy. Then, I stopped drinking. Finally, my work in the hydrogen industry started to gain more recognition, and more reward—both appreciation and financial. All these factors came together to reignite my inspiration, which started as boredom before moving on to new projects, and restarting all of my stalled projects from years past.

I started simultaneously working on all seven books I intended to write for years, including this one, all being at various stages in notes, content and coherence. As I dug into this book, which at the time was *only The Body*, I realized that the book was hollow and incomplete. My failing health led to a degenerating mind, but regaining my lost health didn't reignite the mind, and since the mind was stalled, the body had no direction to move. I started thinking about this deeply, concluding that this book could not be whole without equal consideration to the mind, and an underlying message about purpose, and how we direct our strength.

I go into these thoughts in greater detail in *The Mind*, but the dualistic philosophy of separation of body and soul is not only wrong, but demonstrably toxic. This concept was first proposed by Pythagoras, with the seminal propagation coming from Plato, and the final barrage that flooded the consciousness of the West, led by the final general in this philosophy of separation was René Descartes. This philosophy has often led to the destruction of personal responsibility. As a result, it is not only wrong but *dangerous*.

The official theology differs so dramatically from denomination to denomination within Christianity, let alone the other Abrahamic religions, that defining the difference between the body, mind and soul is a fool's endeavor. The problem is, however, that throughout virtually every official position I have looked into, the definition is largely incoherent. Friends and family members of mine that hold deep seated faith, with leadership positions within their respective churches, validate this. One stated, "*Neither practicing Christians, nor non-Christians, understand what the soul is for the most part*" (and of course, he means his denomination's definition). In practice, I've noticed that most individuals tend to think of their intellect and personality as one and the same as their soul. This fatal misunderstanding leads several denominations and many adherents within the monotheistic religions, even adherents of denominations whose scripture refutes this concept, to conclude that any introspection of our faults is akin to heresy, and any suggestion that our mind and spirit can be altered is an affront to who we are—a proposed assassination of what makes us "us." Most readers are likely to have heard someone avoid acknowledging a personality fault of theirs with the phrase "this is just who I am, it's how God made me." Of course, whether this is thought consciously or not is irrelevant, this pervasive toxin destroying the fabric of personal progress has infected our culture so thoroughly that the damage is done, whether we are aware of it or not.

In truth, our self is an ever-changing sphere made up of our experiences, genetics, hormones, and neurotransmitters—all of which change with our health, our actions, our stress, and even the thoughts we actively guide ourselves through. Some denominations support this concept, but their adherents may not know it. Most people are more likely to regurgitate a thought they heard in a movie or TV show, or saw an influencer post on social media, than they are to deeply think and educate them on a subject. This is even true for most individuals' most deeply held beliefs.

We are not the same person as we were yesterday, and we will not be the same person tomorrow as we are today. That is more than okay, as it gives us the opportunity, each and every day, to view ourselves as reborn, as capable of more. Each day we are granted the opportunity to seize the rest of our life and aspire to become the person we hope to be. If the soul was immutable, this aspiration would be rendered impossible. But, people do change, so, our self is not immutable, and as such if the soul does exist, it is not separated and it is not immutable, either.

Conversely, if you're an individual or within a denomination of an Abrahamic religion who believes that your soul is an immutable essence from God—meaning it is completely detached from all of your experiences and current situation, for better or worse— then what you change to the mind and body, therefore, will not have any effect on the soul. This is critical, as the lessons I am teaching are designed to better ourselves during this life. We don't need to believe in an afterlife to be better today, and the belief in an afterlife is not a justification to avoid improvement during life. Most people will find any excuse to avoid growth, the purpose of this thought experiment is to remove the excuse of dualism—because how many define it contradicts both scientific fact *and* the theological doctrines it is based upon, even at opposite ends of the spectrum of interpretation.

To become whole, in mind, body, and spirit, we need to understand who we are, first. We cannot reunite these components into their totality without reconciling what encompasses each component. We need to understand our limitations, our potential, and the preconceptions that are impairing our trajectory; whether they be our injuries that lead to excuses on why we *can't* be healthy, or our resentments and insecurities that convince us of why we can't improve our attitude, knowledge, and mental capacity.

So, these reflections led to the book doubling in size, but more than doubling in thought and consideration. Critically, it led to thoughts I believe can, and will, truly impact some of those who read it. It's often said that if even one person benefits, the effort was worthwhile. I don't agree with this, but if even one thoughtful person were to gain half as much as I did from writing this, and go on to better the world because of it, then publishing would have been worth the effort. For myself, I have already gained more from writing this than could ever be measured in financial reward; one of many reasons this will eternally remain free on my private domain, and that all profits from Amazon, Kindle, and Audible will be directed, in their entirety, to advertise the message to others.

So, if you haven't already, take the next step and explore your mind, letting the lessons and guidance I wrote in *The Mind* assist you on the journey. First, there are some critical interludes that outline the responsibilities you face with newfound strength. This book was written to help myself find my strength, and to let my purpose congeal into a vividly-mapped direction. It was published in the hopes that those of you capable of seizing strength for yourselves will channel that strength into a purpose that lifts others, that protects not predates, and perhaps if you're strong enough, that seeks to prey upon the very wolves that have encircled all that is good and true in society. But again, first you must unite; first yourself, and then with others who share the same vision. One step at a time.

Now Direct It

If you've used this book as a blueprint, which I hope you have, and moved through the chapters conquering each stress physically, as you learn to understand them mentally, you've now only just begun the battle. Indeed, there is a whole war to fight, a war for truth, decency, freedom, and autonomy. A war against wolves that, quite often, you may not even be aware exist. You've started your journey to condition your system. You've moved through discomfort; through cold, heat, hunger, fatigue, and friction. You've faced your limits and stepped past them. You've learned how your body responds under pressure, how it adapts under load, how it recovers, and all of this matters.

However, physical capacity alone is not enough. No—strength without direction becomes volatility. Resilience without clarity becomes reflex: hardwired, automatic, and blind. You've aspired to build something real in your body, but the question now becomes, what do you do with this newfound capacity? What purpose does your strength serve, and how will you ensure your direction is properly aimed and calibrated to your goals, to who you want to become?

You are no longer passive tissue reacting to stress. You are an organism capable of exerting force, withstanding strain, and regenerating under pressure, but that strength—the kind that lasts—needs orientation, it needs a target. In fact, the stronger you get, the more desperately a target is needed, because without a target, strength will fester, stagnate, and eventually explode at the wrong time, on the wrong target, with an outcome neither desired nor anticipated.

If you started with *The Body*, your journey doesn't end here. Because your next edge isn't physical, at least not primarily. The next phase unfolds within: in interpretation, attention, and belief. The next battlefield shifts from your muscle into your mind. It's in the stories you believe, the meanings you assign, the habits of thought that calcify into identity, and the ones that quietly erode it. If you don't direct your strength toward something chosen, it will be hijacked by whatever noise yells loudest.

So this is where the next discipline begins, not in pushing the edge—but in aiming it. Sharpen your focus, set your footing. You've trained the body. Now it's time to face the self.

Prelude to The Weight of Strength

Before We Begin

Let's get something clear—because if what follows is misunderstood, it will be twisted into the very thing it was written to resist. This is not a philosophy of domination.

It is not a justification for elitism, and it is *absolutely not* a blueprint for collectivism.

This is a call to strength for those capable of becoming strong, not just in body or mind, but in integrity, in restraint, and in wisdom, complete strength of being in its full form. This is the type of strength that is not built for personal glory, we do not seek it to isolate or rule, its purpose is to carry.

The strong exist *for the rest*: their burden is heavier not because they are worth more, but because they can bear more. Strength, rightly cultivated, is not about looking down, rather, it is about lifting up. A functional society requires a spectrum of strength and ability, not uniformity, and not forced equality. Hierarchy is inevitable—but it must be built on integrity and service, not inheritance or ego.

The weak are not useless; they are not to be discarded. They must be treated humanely, allowed to thrive, and given space to contribute in ways that align with their nature. The truly strong, they do not exploit the weak. They protect them—from the wolves: those with raw gifts but no grounding, who seek only dominance, applause, or chaos.

The wolves are not leaders, and they should not be idolized. They are cautionary tales that should invoke fear, anger, and finally action; action to understand who and what they are, and protect against the harm they can inflict. Action to educate others about the dangers. Today, we too often place the wolves on pedestals, and because of this, the wolves are winning. This book exists for those who are capable of suffering deliberately, transforming with discipline, and returning from that fire not embittered, but bound to serve. Because the test of true strength is not how high you climb, it's how much weight you can carry—*and still leave no one behind to the wolves*.

Picture 36. Me snapping a selfie at the hotel gym

Working on my core… values.

The Weight of Strength

Hierarchy is real, not as a political stance, but as a biological fact: *a functional reality*. Some people are stronger than others, mentally, physically, and emotionally. Pretending otherwise doesn't uplift the vulnerable, it serves to disarm the strong and dilute the truth.

For some of the strong, it can be tempting to default to elitism: to carry contempt for those with capabilities less than your own. It is only through a deep understanding of humanity, and the world we exist in, that we can shed this toxicity—or at least, work towards shedding it. The strong cannot reasonably take credit for the genetics they inherited, nor the experiences they accumulated, through luck and randomness, that helped build their resilience. In the same vein, those of lesser capabilities did not choose their genetic makeup, or experiences, either. The answer isn't unrestrained empathy, which can be toxic in of itself, but rather, learned patience, and understanding, leading to tempered empathy. This serves the strong, and society as a whole. We need people of varying capacities. That doesn't mean everyone deserves equal outcomes. It means we need those of us with strength to carry weight that others can't. For that to work, however—for strength to be functional rather than destructive—it must be integrated.

Aldous Huxley saw this coming: in *Brave New World*, when a society tried to fill itself with nothing but Alphas—the genetically "gifted," the engineered elite—it collapsed under its own weight. Why? Because when everyone is exceptional, no one can perform the essential, invisible and mundane work that maintains society. If someone of great talent is forced to perform this type of work, when their equal is able to conduct a fulfilling role? Then resentment will build, and attempts to undermine the hierarchy will intensify, they will be inevitable. Eventually, the system will collapse, as no system can sustain permanent struggle, and unrelenting subversion. A society can't run on dominance alone, you need variation, and you need depth. You need people pulling from different ends of the spectrum.

We are not built the same, and that's not a problem, it's the point. It's what makes society work.

Picture 37. Me after a hard sparring bout

Blood, sweat, and…yeah, mostly blood.

Equity, equality of outcomes, is not a viable solution, but integration is. Difference in capacity doesn't require flattening the world. It doesn't mean forcing equal outcomes. It means recognizing that strength comes with weight—and that those who carry more must also carry more responsibility. Here's the nuance: responsibility is not a burden when it's *integrated*, it becomes stability. It becomes *purpose*. Because unintegrated strength is corrosive.

When a sharp mind detaches from ethics or self-awareness, it turns cold and manipulative. When a strong body is built on insecurity rather than discipline, it intimidates rather than protects. When willpower outpaces clarity, it veers into delusion. These are not hypotheticals: you've seen them. *You've been near them*: the jacked guy at the gym who radiates fragility, the brilliant student who weaponizes language to feel superior, the self-proclaimed spiritual seeker who uses "non-attachment" to avoid hard truths and hide from conflict the man who utilizes his strength to prey on those weaker than him, or the socially adept woman who uses her intelligence to cut rather than connect. None of that is mastery, it's imbalance.

When strength—real or perceived—is channeled through imbalance, it doesn't just fail, it damages.

> *You're here because you know something. What you know you can't explain. But you feel it. You've felt it your entire life… That there's something wrong with the world.*
>
> – MORPHEUS

The thing about a weak body is that, eventually, it breeds a weak mind, not all at once, though. At first, it's just a skipped workout, no big deal, then another, then all of them. It becomes easier to justify the indulgence: the whole pizza, the nightly bottle of wine, the creeping return of whatever vice used to be dormant.

When I was at my worst physically—fifty, sixty, even a hundred pounds heavier than I should've been—my mind didn't feel broken. It was active, I was still thinking, still producing. I wrote, I debated, I executed, but everything I created in that state, no matter how forceful, lacked something I couldn't name at the time. Later, I could: clarity.

I was drinking hard. My nervous system was wrecked: overstimulated and under-recovered. I couldn't stop noticing my own body: the weight, the inflammation, the static in my chemistry. I could still function—outperform, even—but I wasn't seeing clearly. I didn't *understand* what I was seeing. I reacted instead of diagnosing. I wrote solutions, yes, but they were fragmented: surface-level, siloed, held together more by instinct than coherence.

That stretch of time produced ideas with mass, but not form. Weight without structure. Looking back now, I see it plainly: my weak body made my mind *fragile*. The raw cognitive horsepower was still there, but I couldn't go deep—not system-deep, not all the way to the root of anything. Because you can't see the full system when yours is flooded; by alcohol, by anger, by ego, or by unprocessed physical pain.

Today, I see more clearly, not because I got smarter, but because I got stronger. A strong body stabilizes the mind. A strong body brings the nervous system to stillness, clears the inflammation that warps perception, and quiets the noise that once made it impossible to hear your own internal signals—or to call out your own bullshit.

That's what clarity is: the absence of noise. That's what allows you to distinguish reaction from understanding. Once you can see the system, you stop trying to scream it into submission. You learn where to press, where to break, and where to rebuild. You stop chasing symptoms, and you start dismantling root causes. With this clarity the anger fades, and the precision sharpens.

You can't fix the matrix until you can *see* the matrix. You can't see it if your biology is scrambled. Strength gives you that silence, and silence gives you that sight.

You have to let it all go, Neo. Fear, doubt, and disbelief. Free your mind.
— MORPHEUS

This book isn't about domination, it's about structure. It's about what happens when the mind, body, and spirit aren't trained in isolation, but brought into conversation, into alignment.

You don't get stronger by over developing one axis and neglecting the rest. You don't build resilience by stacking skills like trophies on a shelf. You build it by integrating them, so they reinforce each other when life brings unexpected challenges, which it always will, eventually. A fragmented mind isn't fragile because it's weak, it's fragile because it's split: uncoordinated with the body that carries it and the values that ought to guide it.

You can't train the body and ignore the mind. You can't sharpen the intellect and neglect your nervous system. You can't chase spiritual clarity while avoiding physical capacity. Finally, you can't build power in one domain and expect it to hold when the others collapse.

That kind of asymmetry might feel like strength for a while, until it doesn't. Because if your power feeds your ego instead of your presence, you haven't built strength—you've built instability. You've forged a blade with no sheath, and when the time comes, you won't just cut others. You'll cut yourself, and you won't even understand why.

As Hannah Arendt (1951/1973) put it in *The Origins of Totalitarianism*:

> *What makes men obey or tolerate real power and, on the other hand, hate people who have wealth without power, is the rational instinct that power has a certain function and is of some general use. Even exploitation and oppression still make society work and establish some kind of order. (p. 5)*

If you're going to pursue wealth, you must pursue power, and, hopefully after reading this book, you will do it with the intent and purpose of being a shepherd—of bringing value to society. Of paying back in kind, contributing structure and foundation in return for the luxuries you've received.

The solution isn't to pretend everyone's the same. The solution is to carry the responsibility that comes with being strong—whatever "strong" means in your context. To honor that strength across *every* dimension, not as performance, not as branding, but as daily practice. When you do that—when strength becomes whole, not splintered—you stop using it to dominate. You use it to stabilize, to protect, to uplift, and to step into the room and lower the chaos, rather than adding to it.

Because that's what strength is for: divided strength becomes danger, while integrated strength becomes peace. You don't train so you can rise; you train so that *when* you rise, you don't leave wreckage behind. That is the weight of strength, and it must be carried with both hands. So now—flip the book, invert the lens, and start from the beginning.

Read it again, not just as mental preparation, but as the groundwork for physical expression. Mind and body are not separate systems. They're facets of the same structure, and they fail—or flourish—together. Start there, begin again, and this time, bring your whole self with you.

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