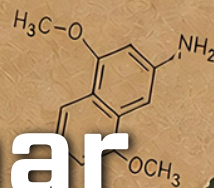
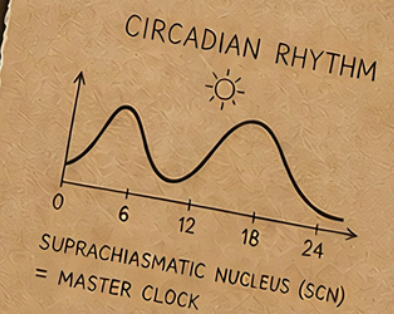
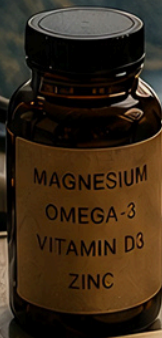


TRAVELING BIOHACKER

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A PRACTICAL GUIDE TO
STAYING FUNCTIONAL ON
THE MOVE

Michal Kodnar



Traveling Biohacker

by Michal Kodnar

Why This Book Exists

I travel a lot.

I move between places, stay for a while, then move again. New cities, new environments, different rhythms of life. I enjoy that part of it. Being somewhere else changes how I think, how I feel, how I pay attention. It wakes me up. It gives me a kind of energy that I don't get from staying in one place for too long.

But the act of traveling itself is something I've never really enjoyed.

Airports, long hours of sitting, broken sleep, constant adjustments. You arrive somewhere new, but your body is lagging behind. Your routine is gone. Your environment is unpredictable. Even simple things – food, light, space – are suddenly outside of your control.

And with all that traveling, I've spent years exploring what people call "biohacking".

I've experimented with food, different ways of eating, fasting, supplements, training, recovery, sleep, and how different environments affect the body. Trying things, observing what happens, keeping what works, discarding what doesn't. Over time, you build a system for handling all of this. Something that holds together. Something that supports how you want to function.

However, that system depends on stability.

And that's where the problem started.

When I began traveling more seriously, everything that used to feel structured became unstable. The conditions kept changing, and a lot of what I thought I had figured out stopped working the same way. Things that made sense in a controlled environment became difficult to apply on the move.

What I kept running into was a mismatch that didn't resolve no matter how I approached it.

There are endless “biohacking” books about optimizing your body, your routines, and your environment. Most of them assume you have control over those things. And there are also plenty of ordinary, “non-biohacking” books about travel that treat the body as something secondary, something that will somehow adapt on its own. Isn't that ironic?

So I started testing what actually holds when you're moving. Not in perfect conditions, but in real ones. Different places, different time zones, different constraints. Some things fell apart quickly. Others adapted. A few turned out to be more useful than I expected.

After all, travel is not something you can eliminate. People move. For work, for relationships, for curiosity, for the need to see what's beyond what they already know. There's something deeply human about being in different places, about experiencing the world directly instead of staying in one fixed point.

At the same time, modern travel is not aligned with how the body works.

Sitting for hours, artificial environments, disrupted circadian rhythms, constant transitions. It pulls you away from the conditions your biology expects, and it does it repeatedly.

So if you care about your body, ignoring this doesn't make sense. Intervention is necessary if you want to turn this “junk” into something healthier.

However, travel is not like junk habits that you can simply remove from your life. That's the reason why I'm not interested in writing a book called *Fast Food Biohacker. Or How to Shoot Heroin While Maintaining Mitochondrial Function*.

There's no point in optimizing things that are fundamentally destructive when you can simply not do them. Abusing your body and ignoring obvious signals – those are choices with clear alternatives. You don't need a protocol for that. You just need better decisions.

Travel, at least in my opinion, doesn't fall into that category.

People move. They explore. They leave what they know and go somewhere else, not because it's perfectly aligned with their biology, but because something in them wants to. And that matters.

The problem is not that travel exists. The problem is how we do it now.

This book exists to make that conflict more manageable. To bridge the gap between how the body actually works and how modern travel is structured.

If you are a traveler, a biohacker, or somewhere in between, this is for you. I hope you will enjoy it.

Attention: What Biohacking Is and What It Isn't

Before we move into the topics of this book, there is something worth clarifying – something that keeps coming up whenever people talk about biohacking, and something that never quite sits right with me.

If you look up the term biohacking online – or ask an AI – you will usually encounter a textbook-style definition. Biohacking is described as an interdisciplinary approach to optimizing the human organism, and the environment surrounding it, in order to achieve specific and measurable outcomes.

The description tends to follow a predictable pattern. Adjust lifestyle variables deliberately. Modify nutrition, sleep, movement, stress management, light exposure, temperature, or technology. Measure the effects. Iterate. The idea is systematic self-experimentation – using insights from biology, neuroscience, and behavioral science to improve physical and cognitive performance.

Am I wrong?

Personally, I have never been particularly fond of this definition. Something about it reflects the cultural moment we live in. It carries the mindset most of us already have too much of – constant achievement, constant measurement, constant performance improvement. Control the environment. Scale the system. As if the human body were an underperforming startup waiting for optimization. Another project to upgrade.

For me, biohacking means something different.

It is not primarily about outcomes. It is not about extracting the maximum possible output from yourself. The starting point is much quieter. Biohacking begins with redirecting attention – away from results, away from goal chasing, away from endless productivity fantasies and to-do lists – and turning that attention inward.

It begins with noticing.

The breath.

Tension in the shoulders.

Fatigue that we usually dismiss.

Energy levels that rise and fall during the day.

How the body reacts to food, climate, time zones, or unfamiliar environments.

The subtle anxiety that appears when the mind starts overthinking.

Only after we can perceive what is happening inside us does change become meaningful. Only when we understand the signals of our own physiology does it make sense to apply methods, protocols, or techniques.

At some point, however, you start trying many new things. You introduce a change, observe the response, adjust. That's where most people associate biohacking with experimentation – and not without reason.

This becomes especially visible with supplements and plant compounds. There are thousands of them – herbs, extracts, traditional remedies, adaptogens. I've tried a lot over time. Some of them are genuinely interesting. Some are useful. Many are forgettable.

It doesn't take much for that to get out of hand.

One addition turns into five. Five turns into ten. Each one has a rationale, a mechanism, a reason to exist. But taken together, they stop being informative. The signal gets buried under accumulation.

At that point, it's no longer clear what is doing what – or whether anything is doing much at all.

That's why the number of compounds in this book is deliberately limited. Not because the rest don't exist, but because most of them don't change the underlying picture in a meaningful way. You don't need dozens of inputs to understand how your system behaves. A few are enough to see patterns clearly.

Methods are the second step.

Attention is (and should always be) the first.

Without that foundation, biohacking easily becomes just another extension of performance culture – result-oriented, extractive, disconnected from individual needs. With attention, it becomes something else entirely – a process of learning how your own biology behaves.

And that distinction matters, especially in the context of travel.

In addition to supplements, this book contains many hacks. Real ones. Protocols, strategies, and experiments – some simple, some unconventional. But none of them are more important than your own signals. What works for me, or for someone speaking confidently on a podcast, may not work for you. What is easy to implement in a controlled home environment may be unrealistic on an overnight train in India or during a chaotic airport transfer.

Travel removes control and all subsequent texts are written accordingly.

Light exposure changes. Food quality changes. Water composition changes. Sleep timing shifts. Social context becomes unpredictable. Decision load increases while recovery opportunities shrink. In such conditions, rigid protocols often collapse very quickly. What remains is the ability to recognize what is happening inside your own system and respond intelligently.

That is the real skill this book tries to cultivate.

Not perfect sleep in every country.

Not optimizing every variable.

Not turning travel into a laboratory experiment.

The goal is simpler and far more practical – maintaining biological coherence when the environment around you becomes unstable.

You may also notice another pattern as you move through the chapters. You will not find recommendations for exotic machines, invasive procedures, or gadgets costing thousands of dollars that promise instant transformation. Most of the tools discussed here are simple, inexpensive, and widely accessible. They are interventions that tend to produce disproportionately large effects relative to their cost and complexity.

Some of the methods you encounter will resonate with you and become part of your routine. Others you will try once and abandon. That is perfectly normal. Adaptation

is not about following every recommendation. It is about choosing the right intervention at the right time – and occasionally choosing not to intervene at all.

On the road, it is easy to become absorbed by logistics – itineraries, meetings, destinations, experiences. The body quietly moves into the background. Fatigue is negotiated with. Hunger is postponed. Overload is justified.

Travel does not create these patterns; it amplifies them.

If you ignore your physiology at home, travel will intensify that tendency. If you already pay attention, travel becomes something else: a testing ground where those skills are sharpened.

As you read the following chapters, treat every method as a possibility rather than an obligation. Techniques are tools. Their value depends entirely on context.

And in this book, the most important context is you.

Why Travel Is Biologically Disruptive

For most of human history, the sun structured human physiology. Light entering the retina does not merely enable sight; it calibrates hormonal timing, sleep–wake cycles, metabolic activity, and temperature regulation. Darkness allows a different biological mode to emerge...

Seasonal variation certainly existed, but it unfolded gradually. The organism could anticipate change because change had duration.

Of course, preindustrial humans moved. Some moved constantly. Yet movement occurred at a biological scale. Hunter-gatherer groups shifted camps within ecological territories. Environmental transitions were continuous rather than abrupt. Light timing changed with latitude slowly enough for the circadian system to recalibrate. The climate shifted progressively. Microbial exposure evolved across landscapes, not across continents in a single afternoon.

What distinguishes modern travel is not movement itself.

It is compression.

A single day can now contain multiple climate zones, artificial light at biologically inappropriate hours, altered atmospheric pressure, different water composition, new microbial ecosystems, unfamiliar languages, and unfamiliar faces. The organism experiences discontinuity rather than transition.

Commercial aviation is barely more than a century old. Intercontinental long-haul travel is even younger. On the evolutionary timeline, the ability to wake up in one hemisphere and fall asleep in another does not meaningfully register.

Human regulatory systems evolved to recalibrate across days, seasons, and generations. Airplanes, however, move faster than our endocrine systems.

The same compression applies to food. Human diets were historically diverse, yet geographically bounded. People consumed what their environment repeatedly provided. The gut microbiome co-adapted with local soil, local water, recurring plant species, and familiar animal proteins. Even seasonal dietary shifts occurred within ecological limits. Exposure built familiarity and familiarity built stability.

Today, within a single week, the digestive system may encounter radically different bacterial environments, unfamiliar fermentation practices, abrupt macronutrient shifts, and irregular meal timing. The organism is not merely tasting new cuisine; it is confronting new ecosystems.

The digestive system is adaptable, but adaptation depends on continuity. Microbial communities stabilize through repetition. When inputs change faster than regulatory systems can reorganize, digestion becomes reactive rather than anticipatory.

Social stability follows a similar pattern. Humans evolved in relatively small, repeated groups. Faces were familiar. Roles were predictable. Hierarchies were stable enough to reduce uncertainty. The brain economizes through repetition; it automates what it encounters frequently.

Travel interrupts that automation. Entering a new cultural and perceptual environment increases interpretive demand. A phenomenon known as the **cross-race effect** illustrates how exposure shapes neural precision: we distinguish more accurately among faces we repeatedly encounter and less accurately among those we do not.

Why mention this in a book about biohacking? Because the comparison matters. Human neurobiology developed under repeated exposure, not perpetual novelty. When familiarity is replaced by a dense stream of new input – new faces, new cues, new rules – the brain spends more energy categorizing and predicting.

When it comes to anything related to travel, we really just need to remember one thing – novelty itself is not pathological. Humans tolerate change remarkably well. Yet most ancestral novelty appeared episodically and within a stable background. Modern travel layers novelty upon novelty without pause.

Across light exposure, geography, food ecosystems, and social structure, the same pattern emerges. The organism evolved in environments where instability had limits and buffers. Even hardship unfolded within biological tolerances. There was friction, but there was also time.

And travel removes that time.

Anyone who travels regularly recognizes this, even without naming it. When biological rhythm begins to thin out, subtle shifts appear. Sleep drifts. Digestion becomes less predictable. Recovery slows. Mood fluctuates. Not dramatically at first. Often not enough to be called illness. But enough to be felt.

Adapting intelligently requires understanding what becomes destabilized when movement begins to outpace biological expectations.

That is the layer we will examine next.

What Travel Does to the Body?

In the previous chapter, we established a core principle – travel compresses environmental change into timeframes that biology did not evolve to handle smoothly. Light shifts, climate shifts, microbial shifts, social shifts – layered on top of each other. Easy.

Nevertheless, the next step is to move from principle to mechanism.

What exactly happens inside the body when that compression occurs?

Circadian Rhythm

Circadian disruption is the natural starting point – not because it is important in the context of travel, but because it is fundamental in the context of the human body in general.

Light is not merely something that allows you to see. It is biological information. When light enters the eye, specialized cells in the retina transmit signals to a small structure deep in the brain called the **suprachiasmatic nucleus (SCN)**. The SCN

sits in the hypothalamus and functions as the body's master clock, coordinating daily rhythms across the entire organism.

When the SCN receives light input – particularly blue-enriched light – it adjusts timing signals that regulate sleep, alertness, body temperature, metabolism, and hormone release.

One of the most studied consequences is the suppression of **melatonin**, the hormone that signals biological night. Controlled laboratory studies repeatedly demonstrate that light exposure at the wrong time can delay melatonin secretion and shift circadian phase (Czeisler et al., 1986; Gooley et al., 2011).

If this doesn't sound fundamental to you, keep listening. Because melatonin is only one layer of the story.

The circadian system is hierarchical. The SCN functions as the conductor, yet nearly every major organ contains its own local clock — in the liver, pancreas, adipose tissue, and even immune cells. These peripheral clocks regulate when tissues expect food, when insulin sensitivity peaks, when repair processes occur, and when immune responses are activated.

It's kind of magical, isn't it? Our bodies are literally made to live on this planet.

Under stable conditions, the master clock and the peripheral clocks remain broadly aligned. Light informs the brain about the time of day. Meal timing reinforces metabolic rhythms. Sleep consolidates the cycle. Perfect regularity is not required, but some degree of consistency is.

Even within a single city, modern habits can destabilize this system. Late-night screen exposure, artificial light at biologically inappropriate hours, irregular meal timing, and chronic sleep restriction all shift melatonin rhythms and weaken circadian coordination without crossing a time zone. Travel therefore does not introduce circadian disruption – it just intensifies the same mechanisms under more extreme and compressed conditions.

When time zones change, the master clock begins adjusting to the new light–dark cycle. And as we might expect, the rest of the body does not instantly follow. The liver may still operate on yesterday's schedule. The pancreas may remain primed for glucose handling at what used to be breakfast time. Adipose tissue may still expect fasting while dinner is eaten in the new location.

This internal lag is known as **circadian misalignment**.

Experimental research shows that when sleep–wake cycles and feeding schedules fall out of alignment with endogenous circadian timing, metabolic responses change. Controlled human studies demonstrate that circadian misalignment impairs glucose tolerance and reduces insulin sensitivity (Scheer et al., 2009). Even short-term disruption increases post-meal blood glucose levels and alters appetite-related hormones such as **leptin** (hunger increasing hormone).

Put simply, the body becomes temporarily less efficient at handling energy when its internal clocks disagree.

This helps explain why jet lag often feels different from ordinary sleep deprivation. Fatigue is only part of the experience. What’s actually happening, though, is that multiple physiological systems inside the body disagree about what time it is.

And disagreement inside a biological system always carries metabolic cost.

Stress

Cortisol, one of the body’s primary stress hormones, provides a clear example of how strongly physiology depends on timing.

Under stable conditions, cortisol follows a predictable daily rhythm. Levels rise sharply within the first **30–45 minutes after waking** – the **cortisol awakening response** – and then decline gradually throughout the day. This pattern supports morning alertness, regulates blood glucose availability, and modulates immune activity.

Crossing time zones clearly disrupts this rhythm. Behavior shifts immediately – waking, eating, and moving according to the local clock – but internal hormonal timing adjusts more slowly. For several days, cortisol secretion may still reflect the original time zone.

This misalignment can manifest as early-morning waking, afternoon crashes, restlessness at night, or a vague sense of unease that many travelers struggle to explain. As we’ve already mentioned, jet lag isn’t just about sleep deprivation, but about a comprehensive disruption of our body’s systems. And the areas affected go far beyond just melatonin (and, as we’ll see later, cortisol as well).

Most of us are also familiar with an interesting phenomenon – eastward travel tends to be more disruptive than westward travel.

What is the reason for this?

The answer is most likely the average intrinsic human circadian period, which is slightly longer than 24 hours – commonly estimated around **24.2 hours** (Wright et al., 2001). Because of this, delaying the clock (staying awake later) is generally easier than advancing it (falling asleep earlier). Eastward travel requires a phase advance, which is physiologically more demanding for most people.

In a nutshell – cortisol's influence extends far beyond what is commonly labeled "stress." It regulates glucose release from the liver, influences insulin sensitivity, and shapes immune signaling. When cortisol timing becomes misaligned with food intake, sleep, and activity, these systems operate with reduced coordination. Glucose handling becomes less efficient, immune responses fluctuate, and subjective energy levels may feel inconsistent.

Aircraft

Now add the aircraft environment itself.

Commercial aircraft cabins are typically pressurized to an altitude equivalent of **6,000 to 8,000 feet (≈1,800–2,400 meters) above sea level**. The oxygen percentage in the air does not change, but the lower air pressure reduces the amount of oxygen that diffuses into the bloodstream with each breath. Controlled measurements show that even healthy individuals experience a measurable drop in blood oxygen saturation at these cabin-equivalent altitudes (Cottrell et al., 1995). The reduction is usually modest and well tolerated, yet it still alters physiology.

Mild **hypobaric hypoxia** increases sympathetic nervous system activity and can influence sleep architecture. Studies conducted at moderate altitude report more fragmented sleep and reduced **slow-wave (deep) sleep**, the stage associated with physical restoration. On an overnight flight, that difference can determine whether someone arrives slightly tired or significantly depleted.

Humidity inside aircraft cabins adds another variable. Levels commonly range between **10 and 20 percent**, far below typical indoor environments. In fact, these are the kinds of conditions our bodies normally encounter only in very dry, desert-like climates. In such extremely dry air, water evaporates more readily from the skin and respiratory tract. The airway lining becomes drier, and **mucociliary clearance** – the mechanism that removes particles and pathogens – becomes less efficient.

Environmental physiology research links low-humidity exposure to increased subjective fatigue and airway discomfort (Nagda & Koontz, 2003). Each factor is

relatively small, but they accumulate across hours. For example, after a 10-hour flight, I personally suffered quite a bit from an inflamed salivary gland, which was certainly exacerbated by the air in the plane (this has never happened to me outside of a flight). A dry nose and chapped lips, however, are almost inevitable.

Reduced oxygen availability, low humidity, dehydration, circadian disruption, and immobility together increase metabolic demand. Aviation physiology studies have therefore documented transient elevations in **oxidative stress markers** during the flights (Netzer et al., 2024).

Oxidative stress refers to a temporary imbalance between reactive oxygen species and antioxidant defenses. The body compensates through robust antioxidant systems, yet that compensation requires metabolic resources and energy.

Over a long flight, these environmental changes rarely produce dramatic symptoms. More often they appear as subtle shifts – in addition to the aforementioned dried mucous membranes you can also experience lighter sleep, dry throat, mild headache, heavy legs, increased fatigue upon arrival. Each factor may seem minor in isolation. But together they create a physiological state meaningfully different from baseline.

I don't want to bash airplanes. I'm grateful for them, and personally, I've been an airplane nerd since I was a kid – I'm always excited about the type of plane I'm flying. However, airplanes are engineered for safety, efficiency, and structural constraints. Human physiology adapts to that environment – but it does so through active regulation, not because the environment is neutral.

Circulation

Blood does not return from the legs to the heart by magic. Movement plays a central role, particularly through contraction of the **calf muscles**.

Each time you walk, flex your ankles, or shift your weight, these muscles compress deep veins and help push blood upward against gravity. This mechanism is commonly known as the **calf muscle pump**, one of the simplest yet most important circulatory support systems in the body.

When sitting for extended periods – especially in the confined space of an airplane seat – that pump becomes inactive. Blood flow slows in the lower extremities. This slowing, known as **venous stasis**, is one of the three main factors contributing to clot formation.

The relationship between long-haul air travel and **venous thromboembolism (VTE)** has been studied extensively. Although the absolute risk for a healthy individual remains low, relative risk increases with prolonged immobility, particularly on flights longer than four hours (Kuipers et al., 2007). For individuals with additional risk factors – previous clotting events, hormonal therapy, or certain genetic predispositions – the risk becomes more relevant.

Simple mechanical interventions can reduce this risk. Graduated compression stockings, which apply controlled pressure to the lower legs and support venous return, have been shown in systematic reviews to reduce the incidence of asymptomatic deep vein thrombosis during long flights (Clarke et al., Cochrane Review 2016). I remember one long flight where I didn't bring them, and because of that, the whole thing turned into a nightmare (trust me, they're worth having).

In fact, even without clot formation, immobility affects vascular function more subtly. **Endothelial cells**, the thin layer lining blood vessels, respond to movement and blood flow. When flow decreases for extended periods, endothelial responsiveness declines. Researchers often measure this using **flow-mediated dilation (FMD)**, a marker of how effectively blood vessels expand in response to increased blood flow. Prolonged sitting frequently reduces FMD, indicating temporarily decreased vascular flexibility.

Why does this matter outside of cardiovascular disease?

Because vascular tone influences blood pressure regulation, tissue perfusion, and overall circulatory efficiency. And yes, combined with dehydration and mild hypoxia, immobility contributes to the heavy, foggy, slightly “off” feeling that many of us notice after long travel.

For this reason – movement is not only exercise. It is circulation. And circulation is foundational physiology.

Immunity, Gut and Electromagnetic Exposure

The immune system adds another layer to how the body responds to travel.

Immunity is not static. Many immune processes follow daily rhythms. The number and activity of certain immune cells change across the 24-hour cycle. **Cytokine production** – the signaling molecules coordinating inflammation – also fluctuates

according to circadian timing. These rhythms are regulated partly by the central clock in the brain and partly by local clocks inside immune cells.

When circadian timing shifts, immune regulation follows.

Experimental studies in humans and animals show that sleep disruption and circadian misalignment can alter inflammatory signaling and **reduce natural killer (NK) cell activity** (Irwin et al., 1996). NK cells contribute to early defense against viral infections and help identify abnormal cells. A temporary reduction in activity does not automatically produce illness, but it represents a measurable shift in immune readiness.

Sleep introduces another variable. Sleep restriction alone has been shown to blunt **antibody responses** to vaccination (Spiegel et al., 2002). In controlled studies, individuals who slept less produced fewer antibodies after vaccination than those with adequate sleep. One short night does not eliminate immunity, yet the finding demonstrates how sensitive immune performance is to sleep duration and quality.

Travel often combines several of these stressors at once – circadian shift, fragmented sleep, mild dehydration, hypoxic cabin air, and exposure to unfamiliar microbial environments. And all of this affects our immune system in some way.

The result is not necessarily immediate illness. More often it appears as a temporary reduction in resilience – a narrower margin between exposure and response. Many people report becoming ill after returning from a trip rather than during it. The immune system is recalibrating while simultaneously encountering new variables.

This is not fragility.

It is regulation under load.

And the gut adds another layer.

The **intestinal microbiome** is not static across the day. Microbial populations and their metabolic activity follow daily oscillations influenced by feeding rhythms and the host circadian clock (Thaiss et al., 2014). When feeding timing shifts or circadian alignment weakens, these microbial rhythms shift as well.

In animal models, circadian disruption has been associated with altered microbial composition and increased intestinal permeability – sometimes described as reduced **barrier integrity**. This matters because the intestinal lining is not a passive tube. It is a regulated interface composed of tightly connected epithelial cells joined

by protein structures known as **tight junctions**, which control what passes from the gut into circulation.

When barrier function weakens, small components of gut bacteria may enter the bloodstream. One of the most studied is **lipopolysaccharide (LPS)**, a structural molecule in the outer membrane of Gram-negative bacteria. Elevated circulating LPS – sometimes described in metabolic research as **metabolic endotoxemia** – has been associated with low-grade systemic inflammation in experimental settings (Cani et al., 2007).

Precision matters here. Bacteria do not suddenly “produce more toxins” because someone crossed a time zone. The LPS example I used is a normal structural component of certain bacteria. What may shift under stress, sleep disruption, and circadian misalignment, however, is the composition of the microbiome and the permeability of the intestinal barrier. When these regulatory systems shift, symptoms such as bloating, irregular bowel movements, altered appetite, or cognitive dullness can appear.

For many travelers, that pattern is familiar.

The symptoms are not mysterious. They reflect interactions between altered rhythm, new microbial exposure, and temporary changes in barrier regulation.

Electromagnetic exposure adds a smaller and more uncertain layer to the discussion.

Measurements inside commercial aircraft cabins show that **radiofrequency (RF) exposure** generally remains below international safety limits established by organizations such as ICNIRP. These limits focus primarily on thermal effects – the degree to which RF energy can heat tissue – and typical cabin exposure remains well below those thresholds.

At the same time, a separate line of research explores possible non-thermal biological interactions of electromagnetic fields. Some experimental studies suggest that certain electromagnetic frequencies may influence **voltage-gated calcium channels (VGCCs)**. These membrane proteins regulate calcium entry into cells. Calcium is not merely a mineral; it functions as a signaling molecule involved in neuronal firing, muscle contraction, hormone release, and activation of oxidative pathways within the cell.

When intracellular calcium levels rise, excitatory signaling increases. In neurons, this may translate into heightened arousal. In other tissues, increased calcium signaling

can stimulate production of reactive oxygen species as part of normal cellular metabolism.

The theoretical concern is that persistent low-level stimulation of VGCCs could contribute to a slightly more excitable physiological state – increased sympathetic tone, greater difficulty transitioning into deep sleep, or irritability and restlessness in susceptible individuals.

Unfortunately, most mechanistic evidence for VGCC modulation comes from laboratory settings, often **in vitro**. Whether typical cabin-level RF exposure produces meaningful effects in humans during real-world travel remains uncertain. Circadian disruption, sleep fragmentation, dehydration, and immobility currently have much stronger human evidence behind them.

That alone does not make the VGCC hypothesis irrelevant. It places it among biologically plausible mechanisms that remain under investigation rather than established drivers of travel physiology.

For now – at least in my opinion – it belongs in the category of interesting questions that warrant further research rather than immediate optimization.

From Disruption to Strategy

Travel never affects us through a single dramatic stressor. It targets multiple regulatory systems at once, compressing changes in timing, movement, environment, and exposure into a window too short for smooth recalibration.

Human physiology depends on coordination. Hormones fluctuate in relation to one another. Organs anticipate metabolic demand through rhythm. Immune cells traffic according to time of day. Vascular tone adjusts to movement. Digestion follows expected feeding windows. When these processes remain aligned, energy expenditure stays efficient. When alignment weakens, regulatory effort increases.

Most travelers will not develop overt pathology from a few trips. The shifts are more often quiet than catastrophic. But subtle does not mean irrelevant. Frequent travelers often notice drift – lighter sleep, slower recovery, more irregular digestion, reduced stress tolerance, variability in focus or mood.

This is the point where biohacking becomes useful.

Not as a performance theater.

Not as gadget worship.

Not as an excuse to carry half a laboratory in your luggage.

But as a strategy.

Once you understand what travel destabilizes, the question becomes practical – where are the leverage points? What actually helps? Which small interventions prevent the system from drifting further away from coherence?

Travel rewards improvisation. Biology does not.

At some point, however, I stopped trying to optimize travel at any cost. I started trying to stabilize myself inside it. And that shift made the difference.

I do not travel with a pharmacy. I do not try to engineer perfection in imperfect spaces. I look for leverage points – places where a small adjustment prevents disproportionate drift.

Sometimes that means reducing light at night in whatever crude way the room allows.

Sometimes it means moving when I do not feel like moving.

Sometimes it means not eating, even when food is available.

Sometimes it means taking something simple that helps the nervous system settle when the environment will not.

Most of what I use is unimpressive.

A piece of tape over a blinking LED.

A shirt closing a gap in a curtain.

Standing up on a long flight when it would be easier to stay seated.

Walking into daylight before opening a laptop.

Keeping inputs simple when everything around me is novel.

Occasionally I use biochemical tools. Carefully. Contextually. Not stacked for the sake of stacking, and not because more feels safer.

What matters is not the individual tool.

It is the decision logic behind it.

This is where the book turns from mechanism to application.

In the following chapters I introduce what I call the **Traveling Biohacker's Toolkit** – a set of simple interventions designed to stabilize the systems that travel tends to destabilize.

The toolkit is not organized by destinations, airports, or itineraries. It follows biology instead. Each section focuses on one physiological system that tends to drift under travel conditions – circadian rhythm, sleep, metabolism, circulation, stress regulation, and digestion – and on the leverage points that help bring those systems back toward coherence.

You will not need every single tool discussed there. In fact, trying to use everything would create the very instability you are trying to reduce, as I tried to mention at the beginning.

But some of these patterns will be recognizable immediately.

They will match the signals your own body already gives you – light sensitivity, sleep drift, digestive unpredictability, heavy legs, mental fog, nervous overactivation.

So let's get started!

Intervention: The Traveling Biohacker's Toolkit

Temporal Control: stabilizing time when time is unstable

When people talk about sleep “biohacking,” the conversation usually collapses into one question – how quickly can I fall asleep, and how long will I sleep? Even in ordinary life, that framing misses most of the physiology. On the road it becomes almost useless. The central problem is rarely the mechanics of falling asleep. It is timing – not only of sleep, but of almost every regulatory process in the body.

Sleep is only the visible surface of a deeper system. As you might guess, beneath it operates a network of circadian clocks distributed throughout the organism – in the brain, the liver, muscle tissue, adipose tissue, and even immune cells. These clocks regulate when hormones are released, when glucose is handled efficiently, when body temperature rises and falls, and when alertness naturally peaks. Under stable conditions these rhythms remain synchronized because the organism receives consistent environmental signals. Light appears at roughly predictable hours. Food tends to arrive in repeated patterns. Movement clusters in similar parts of the day, while darkness allows another phase of physiology to unfold.

That is why the real challenge on the road is not simply sleeping in a new place. It is preserving coherence among the signals that tell the body what time it is.

Among those signals, light is the most powerful.

Before discussing supplements, routines, and travel-specific tactics, it is necessary to understand how light interacts with the circadian system and why its timing matters far more than most people realize.

Light is not illumination. It's instruction.

In the previous chapter, when we discussed circadian rhythm in the section *What Travel Does to the Body*, we already introduced the suprachiasmatic nucleus (SCN) – the small structure in the hypothalamus that acts as the master circadian pacemaker of the human body. Here we return to it briefly, because understanding how light reaches this structure explains why the timing of light exposure matters so much.

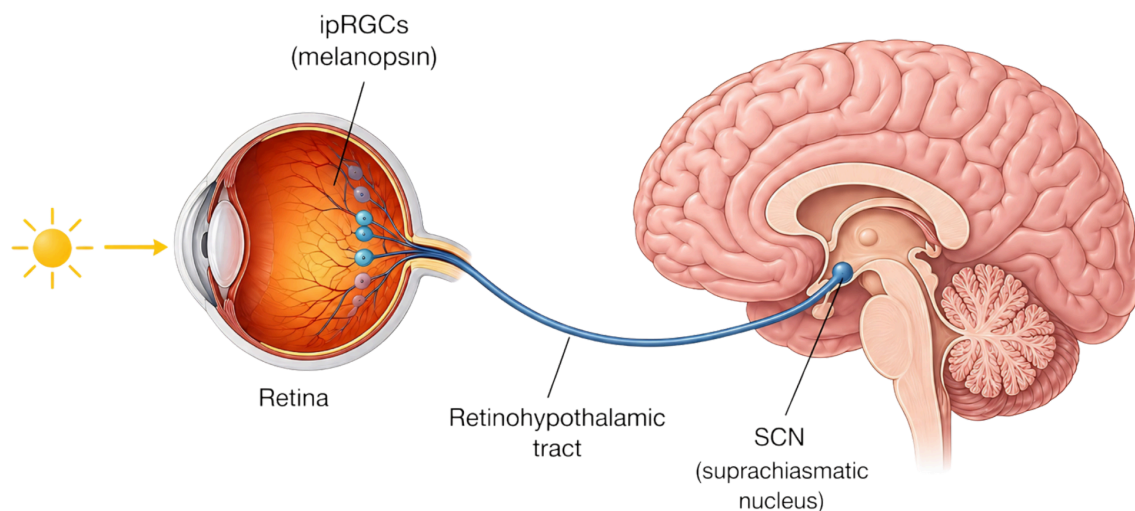
When light enters the eye, it does not simply generate visual perception. Part of that signal is routed into a separate neural pathway dedicated to biological timekeeping. Light information from the retina travels through a direct connection known as the **retinohypothalamic tract**, which carries timing signals from the eye to the SCN. This pathway bypasses the visual cortex entirely. In other words, the circadian system does not need you to consciously see light in order to react to it. As long as light reaches the retina, the timing system receives the message.

The cells responsible for this signal are not the rods and cones that produce visual images. In the early 2000s researchers identified a separate class of retinal neurons called **intrinsically photosensitive retinal ganglion cells**, or **ipRGCs**. These cells contain a light-sensitive photopigment known as **melanopsin**, which allows them to

detect environmental light directly rather than relying only on input from rods and cones.

The discovery of these cells fundamentally changed how scientists understand the relationship between light and circadian physiology.

In 2002, David Berson and colleagues demonstrated that certain retinal ganglion cells could respond to light even when traditional photoreceptors were inactive, showing that these neurons possess their own photoreceptive capability (Berson et al., 2002). So, yes – even in individuals who are visually blind because rods and cones are damaged, **light can still influence circadian physiology** if melanopsin-containing retinal ganglion cells remain intact.



Light does not need to be seen to be interpreted – retinal signaling alone is sufficient to influence circadian timing.

In the same year, Samer Hattar and colleagues mapped the neural projections of melanopsin-containing cells and showed that they **connect directly to the suprachiasmatic nucleus** and other brain regions involved in circadian regulation (Hattar et al., 2002). Together, these studies established the anatomical and physiological pathway by which environmental light directly influences biological timekeeping.

Now back to SCN, the brain's circadian master clock and the primary target of the retinohypothalamic light pathway. This part of the brain is remarkably small – roughly twenty thousand neurons – but its influence is enormous. It sits just above the optic chiasm in the anterior hypothalamus, positioned in exactly the place where retinal information can reach it efficiently. From there it coordinates daily rhythms across the organism. It communicates with other hypothalamic nuclei, regulates signals to the pineal gland that influence melatonin secretion, and synchronizes peripheral clocks distributed across various tissues that we've already discussed.

Calling the SCN a conductor is not a bad metaphor. It does not perform every biological process itself, but it maintains temporal coherence across systems that would otherwise drift apart. When the SCN receives a strong and consistent light signal, it anchors the organism to a predictable daily phase structure – biological morning, biological afternoon, biological night.

And this is why mistimed light exposure has measurable physiological consequences. The circadian system interprets light not merely as brightness, but as information about the time of day. A strong light signal in the early morning reinforces the transition from biological night into biological day. A strong light signal late in the evening tells the SCN that the day has not ended yet, delaying the internal night.

This is not theoretical. Controlled laboratory experiments have shown for decades that light exposure can shift circadian phase and alter the timing of melatonin secretion. The magnitude of that shift depends on intensity, duration, spectrum, and above all the timing of exposure relative to the internal clock.

Travel does not destroy these mechanisms, but it disorganizes them.

The phase response curve: why morning light works and night light backfires

Once those signals fall out of alignment, it's not just disruption anymore. The system starts moving the wrong way.

Airports make this problem obvious. They are engineered for visibility, security, and commerce rather than biological rhythm. Lighting remains intense late into the night, often with blue-enriched LEDs that strongly stimulate melanopsin-sensitive circadian photoreceptors. The same terminal may be brightly illuminated at 11 p.m., 2 a.m., and again at 5 a.m. From the perspective of the circadian system, those signals resemble daylight.

When you walk through that environment during the night portion of your internal clock, your physiology receives contradictory instructions. Sleep pressure is rising, but the retinal signal says that daytime conditions still exist. The SCN adjusts accordingly and delays the onset of biological night.

Airports aren't just tiring because you're experiencing "travel stress" or have just been on a long flight. The main problem is that **the light there continues to stimulate you** – even when you're tired.

There is a well-described phenomenon in chronobiology called **the phase response curve**. It explains how light exposure shifts the timing of the internal circadian clock depending on when that light is received relative to the biological night.

This relationship has been studied for decades. Controlled human experiments have consistently shown that light exposure can shift circadian phase and alter the timing of melatonin secretion.

What matters is not just that light shifts the clock, but that the same light can shift it in opposite directions depending on when it occurs.

Jet lag is simply the result of this mismatch. After crossing time zones, your internal clock is no longer aligned with the local light-dark cycle. It has to shift until biological night and environmental night coincide again.

Eastward travel requires the clock to move earlier.

Westward travel requires it to move later.

And this is where the broader environment starts to matter. Airports are just one example. The same pattern appears across modern travel as a whole – aircraft cabins, hotels, and constant screen exposure all extend light into hours where the body expects darkness. These signals tend to push the clock later, often at times when the body would otherwise expect darkness.

Once you understand that, the situation becomes easier to navigate. You are not trying to control every variable. You are trying to avoid signals that move the system further away from alignment.

Outdoor light is not “a bit stronger” than indoor light

One of the quiet illusions of modern life is that indoor lighting can substitute for daylight. It cannot. A hotel room lamp may look bright at six in the morning when

you are half awake and staring into a mirror, but biologically it is closer to a candle than to the sun. Sitting under ceiling lights in a breakfast café and assuming your circadian system has received a proper morning signal is a bit like trying to warm yourself by standing next to a laptop charger and calling it a fireplace.

The difference is not subtle. Humans evolved under a sky that routinely delivers orders of magnitude more light than almost any indoor environment. Even on an overcast day, outdoor light can easily reach several thousand lux. Under direct sunlight, levels may exceed fifty thousand lux. By comparison, **most indoor spaces operate in the range of a few hundred lux**. Office lighting often falls somewhere between three hundred and seven hundred lux, which is comfortable for reading but biologically modest.

This gap matters because the circadian system responds to dose. Both intensity and duration of light exposure influence how strongly the internal clock shifts and how powerfully melatonin production is suppressed. Bright outdoor light is interpreted by the brain as a decisive daytime signal. Dim indoor lighting sends a much weaker instruction, sometimes too weak to stabilize the rhythm at all.

To appreciate the scale difference, it helps to look at typical illuminance levels measured in lux. These are approximate ranges rather than fixed numbers, but they illustrate how dramatically the environment changes between indoor and outdoor light:

Environment	Typical lux range (approx.)
Dim bedroom / night lighting	1–30
Typical home lighting	50–300
Bright office lighting	300–1,000
Overcast daylight outdoors	1,000–10,000
Shade on a sunny day	10,000–25,000
Direct sunlight	50,000–100,000+

The point is not to memorize lux values. It is to understand why “sit near a window” usually does not compete with “walk outside for ten minutes.”

This distinction has been demonstrated directly in human experiments. One of the clearest illustrations comes from a field study by Kenneth Wright and colleagues (Wright et al., 2013), in which participants spent a week living under natural outdoor light-dark cycles without artificial lighting. During that period daytime light exposure increased dramatically while nighttime illumination dropped close to natural darkness. By the end of the week, the timing of melatonin secretion – a reliable marker of circadian phase – had shifted earlier by roughly two hours and became closely aligned with sunset.

The lesson is not that everyone should start camping in the wilderness. It is that the circadian system responds strongly when daylight is truly bright and nighttime is genuinely dark.

Morning light exposure is therefore not a motivational “habit.” It is a dose-response relationship. The strength of the signal matters. A short walk outside under real daylight often provides a much clearer timing cue than hours spent under indoor lighting.

Avoiding artificial light at night: the highest leverage you don’t want to do

If morning light is the accelerator, darkness is the steering wheel.

In the previous section we discussed something that initially sounds counterintuitive – indoor lighting is usually far too weak to function as a convincing daytime signal for the circadian system. Even bright-looking rooms rarely approach the intensity of outdoor daylight.

Yet the reverse is also true in an important and slightly unfair way – relatively modest artificial light at night can still disrupt the biological signals that define nighttime.

This asymmetry has been demonstrated repeatedly in laboratory studies. Strong daylight exposure often **requires thousands of lux to produce large circadian phase shifts**. Melatonin suppression and circadian delay, by contrast, can occur at much lower intensities.

In controlled human experiments, Gooley and colleagues (2011) showed that typical indoor evening lighting – around 100–200 lux – significantly suppressed melatonin compared with dim-light conditions. Earlier work by Zeitzer et al. (2000) demonstrated a clear dose-response relationship between light intensity and

melatonin suppression, showing that even relatively low levels of light can measurably influence the circadian system when exposure occurs near biological night.

This is why artificial light behaves differently depending on when it is seen. During the day, most indoor environments are too dim to strongly reinforce circadian timing. At night, that same level of light can act as a meaningful biological signal telling the brain that the day has not yet ended.

Part of the explanation lies in the specialized retinal cells described earlier. The ipRGCs do not help you see the world; they function more like environmental light meters. Their role is to measure the intensity, duration, and spectral quality of ambient light and convert that information into timing signals for the circadian system.

The photopigment inside these cells – **melanopsin** – is most sensitive to short-wavelength blue light, roughly in the range of **460–480 nanometers**. That detail matters in the real world. Many modern artificial light sources – LED lighting, phone screens, airport lighting (I don't have to say it twice), laptop displays – are enriched in exactly this portion of the spectrum.

The visual system and the circadian system are therefore using partially different sensors. A light source that appears modestly bright to your eyes may still deliver a strong signal to melanopsin-containing cells. This is one reason artificial lighting interferes with biological night more easily than people expect.

Mechanistically, the pathway is straightforward – light reaching the retina activates these photoreceptors, which transmit timing information through the retinohypothalamic tract to the SCN. When this signal arrives during the biological evening or night, the circadian system interprets it as evidence that daytime conditions are still present. Melatonin secretion is delayed, and the internal night shifts later.

Melatonin is not the central character of the story. It is simply a convenient marker that researchers can measure. The deeper point is that light at night tells the brain that the environmental day has not ended.

If you want the body to treat a certain window as nighttime, the most effective intervention is removing the strongest “it is still day” signal available to the circadian system.

That is difficult in environments you do not control. Hotel rooms come with standby LEDs, glowing control panels, hallway light leaking under doors, and screens that remain active long after sunset. Airports and airplanes extend the same problem with bright, blue-enriched illumination at the hours when the body expects darkness.

Perfection is not available here.

Reduction is.

Once the problem is framed that way, some of the small and slightly ridiculous interventions begin to make sense.

Strategic darkness: why duct tape is not a joke

I know how it sounds – you travel with duct tape to cover LEDs. It sounds like a meme. Personally, I prefer being a walking meme to being a sleep-deprived mess with a broken circadian rhythm.

What matters biologically is that those small light sources are often close to the eyes, and the circadian system responds to retinal exposure rather than to whether the room “feels bright.” A dark room with a single bright point source can still deliver a measurable light signal to the retina.

So yes: I use tape. I cover the AC display. I cover the router. I cover the TV standby light. I cover anything that creates a “night is not allowed” mood.

Is one LED going to destroy your health? Of course not. But the real problem is rarely one LED. It is the accumulation – indicator lights, hallway leakage, screens, airport lighting, late dinners under bright LEDs, and the added stress of crossing time zones. Tape is not drama. It is simply one more way to remove unnecessary nighttime input so the system can downshift.

There is a less noobish way to say the same thing – sleep physiology responds strongly to contrast. Bright days and dark nights create a robust timing signal, while travel environments flatten that contrast. Covering LEDs is a crude but effective way of restoring contrast with whatever tools you have available.

And if you want to add something else to that, there is also a more speculative reason some people find this topic fascinating. A line of research has investigated **ultra-weak photon emission from living tissues**, often called biophotons. These emissions are extremely faint – usually in the visible and near-ultraviolet range – and are thought to arise from metabolic reactions, particularly oxidative processes inside

cells. Early work by Fritz-Albert Popp and colleagues in the 1970s and 1980s suggested that living organisms emit measurable photon signals related to cellular metabolism. Later studies using highly sensitive photomultiplier detectors confirmed that human tissues do emit these ultra-weak photons and that emission intensity varies with metabolic activity.

More recent work has explored whether these emissions follow circadian patterns. Kobayashi et al. (2009), for example, measured spontaneous photon emission from human skin and found that it fluctuates across the day, peaking in the late afternoon and decreasing at night. Other experimental work has linked biophoton production to mitochondrial activity and oxidative metabolism, since reactive oxygen species generated during cellular respiration can create excited molecular states that release photons as they relax.

This research is fascinating and should also be interpreted carefully. It does not demonstrate that mitochondria are communicating with each other through visible light signals in a way that room LEDs directly disrupt (as many “biohacking influencers”, for example, try to suggest). The emission levels involved are extremely small, and the functional role of these photons in biological signaling remains an open question. Still, the fact that living tissues emit measurable ultra-weak photons linked to metabolic activity suggests that biological systems may interact with light in ways that extend beyond the classical circadian pathway. Whether these emissions are metabolic byproducts, regulatory signals, or something more complex remains unsettled.

Fortunately, none of this speculative territory is required to justify protecting darkness. The circadian literature alone is already strong enough. Controlled studies have repeatedly shown that light exposure at night suppresses melatonin, shifts circadian timing, and alters sleep physiology (Zeitzer et al., 2000; Gooley et al., 2011; Chang et al., 2015). Even without invoking deeper mechanisms, nighttime light exposure clearly interferes with biological night.

The biophoton research simply reinforces a broader point – biological systems are sensitive to light in ways we are still learning to understand.

Which brings us back to tape.

There are now small commercial products marketed to travelers and biohackers specifically for covering LED indicators in hotel rooms. Some are adhesive light-blocking stickers designed for routers, televisions, or electronics. They are convenient, but hardly necessary.

A strip of ordinary duct tape works perfectly well.

The goal is not elegance. The goal is darkness.

And in environments you do not control, a small roll of tape often accomplishes that faster than any “optimized” product.

Blue-light blockers: what they do, what they don't

Blue-blocking glasses occupy a strange place in the sleep world. Some people talk about them as if they are essential medical equipment. Others dismiss them as an overpriced placebo. The reality is actually very simple – **they reduce the amount of short-wavelength light reaching the retina**, and the circadian system is particularly sensitive to that portion of the spectrum.

The relevant biology is already on the table. If you don't remember, here's a quick recap:

The ipRGCs that send environmental light information directly to the SCN contain melanopsin and are particularly sensitive to short-wavelength blue light in the 460–480 nm range. Because of that spectral sensitivity, blue-enriched light can produce strong circadian responses even when the environment does not appear visually very bright. Filtering that portion of the spectrum therefore reduces one of the signals the brain interprets as daytime.

Easy, right?

Laboratory studies have demonstrated for years that evening light exposure can suppress melatonin and shift circadian timing. In controlled experiments, even typical indoor lighting in the evening – **around 100–200 lux** – significantly suppressed melatonin compared with dim-light conditions (Gooley et al., 2011). Earlier work also demonstrated a clear dose-response relationship between light intensity and melatonin suppression, showing that even relatively modest light levels can measurably influence the circadian system when exposure occurs during biological night (Zeitzer et al., 2000).

Spectral composition matters as well. Light is not just brightness; it is a mixture of wavelengths. Research comparing different wavelengths has shown that short-wavelength light – **the blue portion of the spectrum** – produces stronger circadian and melatonin-suppressing effects than longer wavelengths such as red light at comparable intensities (Brainard et al., 2001; Thapan et al., 2001). Two light

sources can therefore appear similarly bright to the eye while producing very different signals for the circadian system.

One widely cited demonstration of evening light effects comes from a study comparing reading on a light-emitting electronic device with reading a printed book before bedtime. Participants using the e-reader showed **suppressed melatonin**, delayed circadian timing, and reduced next-morning alertness relative to the printed-book condition (Chang et al., 2015). Such experiments cannot completely isolate spectral effects from behavioral stimulation – interacting with a device may itself increase cognitive arousal. Still, the physiological results align with a large body of controlled circadian research showing that evening exposure to light, particularly blue-enriched light, delays biological night.

That is the context in which blue-blocking glasses become interesting. If evening light influences circadian timing partly through melanopsin-driven responses, then filtering that portion of the spectrum should **reduce the strength of the signal**.

Several studies have tested exactly that. Experiments using amber lenses that filter short wavelengths have shown reduced melatonin suppression and increased subjective sleepiness in the evening compared with clear lenses under otherwise similar lighting conditions (Burkhart & Phelps, 2009; Sasseville et al., 2006). Other work has reported improvements in subjective sleep quality and insomnia symptoms when participants wore blue-blocking lenses before bedtime (Shechter et al., 2018).

Systematic reviews and broader analyses generally conclude that **the results are mixed**, despite a clear physiological rationale, largely because interventions differ substantially in lens properties, timing, and environmental lighting conditions (e.g., systematic reviews on light exposure and circadian outcomes).

So the mechanism is biologically sensible, but the effectiveness depends heavily on how the tool is used.

I personally use blue-blocking glasses and I use them in situations where controlling the lighting environment is difficult. I wear them in the evening when I cannot adjust the spectrum of the room lighting, and I often use them in brightly lit transit environments where artificial light is unavoidable. I also tend to wear them during the final couple of hours before sleep when screens or indoor lighting would otherwise deliver a strong short-wavelength signal to the retina.

One practical detail is often overlooked: **coverage**. I generally prefer glasses that wrap around the eyes or block light from the sides rather than thin frames that leave

large gaps. If bright light enters from the side or above, it still reaches the retina and can stimulate the same melanopsin pathways you are trying to reduce. Many commercial blue-blocking glasses look like ordinary eyewear, but designs that cover more of the eye – sometimes resembling lightweight ski or safety glasses – often do a better job of actually reducing retinal light exposure.

I do not think these glasses are a magic solution. Circadian timing depends on multiple variables – daylight exposure, light intensity, sleep pressure, meal timing, and behavioral stimulation all interact. But if melanopsin-mediated pathways are particularly responsive to short-wavelength light, reducing that component during biological night is at least logically consistent with the physiology.

They should also not become a license to stay on bright screens for three more hours, eat right before bed, or have coffee at nine in the evening. Intensity and duration still matter. Filtering part of the spectrum does not cancel the rest of the stimulus.

The most honest summary is probably this: the circadian literature clearly shows that light timing and spectral composition influence biological night, and blue-blocking lenses are one practical way to modify that signal when you cannot control the environment itself.

For me, that combination of physiological plausibility and partial empirical support is enough.

Curtain engineering

Sometimes, however, we can't wear blue light blocking glasses everywhere. Especially not when we're sleeping. Yes, we can wear a sleep mask, but personally, I don't prefer them.

So what's the problem? We've already covered some LED lights with tape, but we're often left with other issues we need to solve. Hotels love aesthetic curtains. Biology prefers darkness. Those priorities do not always overlap.

If light is shining through the window or under the door, you can just deal with it and sacrifice the quality of your sleep, or you can do what travelers have always done: improvise. Clothes hangers can pull curtains together more tightly. A shirt can block the bottom edge. A towel can cover a gap.

It's the same idea as using tape to cover the screens. It's funny, but the reason I'm writing this is so you won't be afraid to improvise and will try out optimizations like this at the lowest possible cost.

Meal timing: the second clock you keep ignoring

Light is the strongest circadian input for the brain clock, but food timing is a major timing cue for peripheral clocks, particularly in metabolic organs like the liver. If your light schedule says "it's morning" but you're eating heavy meals at what is biologically midnight, you are creating internal disagreement – central timing versus metabolic timing.

This has been demonstrated experimentally. In animal experiments, restricted feeding schedules can shift the phase of circadian gene expression in peripheral tissues even when the central clock in the suprachiasmatic nucleus remains unchanged. Feeding time alone can re-phase metabolic clocks without moving the brain clock, demonstrating that the circadian system is hierarchical but partially separable (Damiola et al., 2000; Stokkan et al., 2001).

Human studies show that this type of misalignment has measurable metabolic consequences. In controlled laboratory experiments simulating circadian misalignment similar to shift work, participants showed reduced insulin sensitivity, impaired glucose tolerance, and changes in appetite-regulating hormones such as leptin, even over short time periods (Scheer et al., 2009).

The point is not to condemn occasional late meals, but to recognize that the metabolic system has timing preferences, and forcing it to process meals at the wrong internal time is part of why jet lag can feel inflammatory rather than merely sleepy.

So how do you apply this without turning travel into moralism?

You aim for **metabolic agreement**:

- If you are trying to shift earlier after eastbound travel, heavy meals during what is still your biological night are usually a bad bargain.
- If you're landing late and your body thinks it's 3 a.m., eating a full dinner because "it's dinner time here" can feel like you swallowed a brick. It's usually not worth it.

There is also growing human evidence that shifting caloric intake earlier in the waking day can influence metabolic outcomes. In a controlled clinical trial, Sutton

and colleagues tested an early time-restricted feeding protocol in men with prediabetes, in which all daily food intake occurred within an earlier daytime window without intentional calorie reduction. Participants showed improvements in insulin sensitivity, blood pressure, and oxidative stress markers compared with a control schedule (Sutton et al., 2018).

Other controlled studies suggest that, under certain conditions, consuming a larger proportion of calories earlier in the day may improve glucose control compared with identical caloric intake distributed later in the evening (Jakubowicz et al., 2013). However, these effects are not uniform and appear to depend on factors such as metabolic status, meal composition, and overall study design.

Rather than pointing to a single “best” eating schedule, these findings are better understood in the context of circadian biology.

What the chronobiology literature suggests more specifically is that food timing relative to internal circadian phase matters. Late-night eating during biological night appears to produce less favorable glucose and metabolic responses in several experimental studies (Scheer et al., 2009; Morris et al., 2015). The issue is not simply when the clock says dinner, but whether the metabolic system is in its active phase or its resting phase.

Fasting during flights: why it sometimes works, and when it backfires

Fasting during flights is one of the cleaner temporal tools because it reduces signals. Travel is already novel. Your system is already processing unusual stimuli. Removing food as an additional timing and inflammatory variable can make the transition easier.

There are two reasons fasting can help on travel days:

- It prevents you from reinforcing the wrong time zone with food signals. If you keep eating at “old time,” you may slow the shift of metabolic clocks.
- It reduces digestive load in an environment that already dehydrates and stresses the gut.

But fasting is not automatically smart. It can backfire if you are already underfed, stressed, or sleep-deprived, or if it tends to push cortisol higher for you. In those situations, the body may interpret fasting as an additional stressor rather than a useful signal.

Experimental work on metabolic and neuroendocrine responses shows that **fasting can increase cortisol and sympathetic activity** under certain conditions, particularly when combined with sleep disruption or psychological stress (Heilbronn et al., 2005; Tomiyama et al., 2010).

The result can be a state that feels both depleted and overstimulated – poor sleep, unstable energy, and a higher likelihood of compensatory eating later.

So the travel-safe way to frame fasting is not “discipline.” It’s signal management:

- If fasting makes you calmer and clearer, it may be useful on long-haul days.
- If fasting makes you shaky, obsessive, irritable, or insomniac, it’s the wrong lever for you in that context.

A middle path usually works better than strict rules: keep intake simple – protein, fat, vegetables, low novelty – rather than stacking airport carbs, alcohol, random junk food, and sleep deprivation on top of each other.

Eastbound travel is where this tool often becomes especially useful. When you fly east, the local clock often tells you to eat dinner while your internal physiology still thinks it is the middle of the night. In that situation, fasting or very light intake can help avoid building a metabolic rhythm around the wrong circadian phase.

When sleep simply isn’t possible

Sometimes the schedule wins. Overnight flights, immigration lines at 3 a.m., or early hotel check-ins can force a temporary all-nighter. The goal in those situations is not perfect sleep. It is preventing additional circadian confusion.

One common mistake is assuming that if sleep is impossible, circadian timing no longer matters. It still matters. Being awake all night does not switch off the biological clock. Light, food, and activity continue sending timing signals to the system whether you are asleep or not.

The difference can be noticeable. I have had plenty of travel days where sleep simply was not possible – maybe one or two hours on a plane at best. When I ignored timing completely, the next day felt chaotic and heavy. When I still managed light exposure, meal timing, and blue-light reduction, adaptation was noticeably easier.

In forced all-nighter situations, focus on the strongest signals:

- Keep light exposure **bright** during the period you want the body to treat as day
- Keep light **dim and warm** once you decide the night has started
- Avoid heavy meals during what is still **biological night**

This is where blue blockers and strategic darkness stop feeling optional and start making a real difference.

Morning movement

Movement is not just about being fit; it is also a biological timing signal.

In chronobiology, light is the dominant cue for the central circadian clock, but physical activity can act as a secondary zeitgeber – a stimulus capable of influencing circadian phase. Experimental work in humans shows that the timing of exercise can affect internal rhythms, including melatonin secretion and other markers of circadian timing.

Under controlled conditions, exercise performed earlier in the biological day has been associated with phase-advancing effects, while exercise later in the evening can produce delaying effects (Youngstedt et al., 2019; Barger et al., 2004). However, these responses are not consistent. The magnitude and direction of the effect depend on factors such as baseline circadian phase, chronotype, intensity of exercise, and overall physiological state.

In practice, this means that exercise can reinforce or shift your internal timing, but it does not behave like a precise switch.

I've seen this directly in my own data. When I trained early in the morning, my recovery metrics consistently dropped – higher strain, worse readiness, slower recovery. The same type of training performed later in the day didn't produce that effect. In fact, late sessions were often neutral or even slightly better from a recovery perspective.

My experience doesn't perfectly match the research, but both have their limits – including the device that measures my data and the research itself. Some people lean more toward an evening chronotype (myself included), so a late workout might not bother them much, while others may be extremely sensitive to it.

However, we should be aware that exercise clearly affects circadian timing, and the mechanism is likely multifactorial.. Physical activity alters body temperature rhythms, influences metabolic signaling, and affects autonomic activity, all of which interact with circadian regulation. Exercise also increases circulating hormones such as cortisol and catecholamines – epinephrine, norepinephrine, and dopamine – which are naturally higher in the morning and contribute to the transition from biological night to biological day.

This doesn't require an intense workout. A short walk outside, ten or fifteen minutes of movement, or a brief body-weight routine is often enough. I usually combine several cues at once: step outside shortly after waking for natural light exposure, walk for a few minutes, and add some light movement – a short yoga sequence, a few push-ups, or a brief gym session. That combination is usually enough to shift the body into daytime mode.

The reason this works so reliably is that it stacks multiple circadian inputs at the same time. Outdoor light provides the strongest signal for the central clock. Physical activity increases body temperature and metabolic activity. Upright posture and locomotion reinforce wakefulness. When these signals arrive together in the morning, they create a coherent physiological pattern that helps stabilize circadian timing.

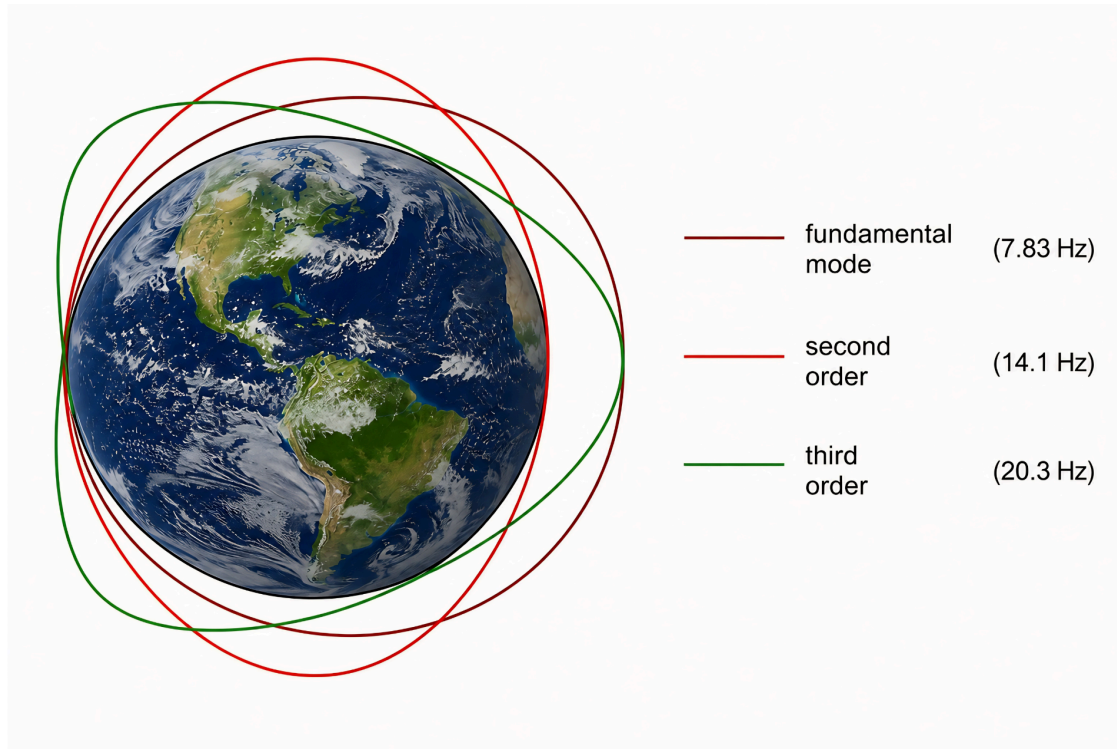
Grounding

Another practice that often accompanies morning movement outdoors is grounding – direct skin contact with the surface of the Earth, such as walking barefoot on soil, grass, sand, or natural stone.

Grounding attracts interest, but the evidence remains limited and controversial. Some of the claims made around it in popular health culture go far beyond what current evidence can support. The practice does, however, sit alongside a broader set of environmental factors that shape human physiology, especially light, movement, and exposure to natural surroundings.

The Earth is part of a global atmospheric electrical circuit. The planet's surface carries a net negative electrical charge, while the **ionosphere** – a layer of ionized particles beginning roughly 50–60 km above the surface – carries a net positive charge. Lightning activity continuously maintains the voltage difference between these layers, producing extremely low-frequency electromagnetic oscillations known as Schumann resonances.

The fundamental **Schumann resonance** occurs at approximately 7.83 Hz, with additional harmonics around 14, 20, and 27 Hz. These resonant frequencies arise because the Earth-ionosphere cavity acts as a natural waveguide for electromagnetic waves generated by lightning discharges. The planet is therefore continuously immersed in extremely low-frequency electromagnetic fields produced by this global electrical circuit.



Simplified representation of Schumann resonance modes around Earth, showing the fundamental (~7.83 Hz) and higher-frequency modes as standing electromagnetic wave patterns around Earth.

Because these frequencies overlap with ranges observed in human brain activity – for example, the alpha band around 8–12 Hz – some authors have suggested that biological systems might be sensitive to aspects of this background. That, however, remains a hypothesis rather than an established mechanism.

In long-term bunker experiments conducted at the Max Planck Institute, participants lived for extended periods isolated from normal environmental cues. Under those conditions, circadian rhythms could drift or desynchronize, showing how strongly human timing depends on external signals (Wever, 1974). Those studies support the importance of time cues in general, but they do not establish a specific role for grounding or geophysical electromagnetic fields.

Separate research has examined grounding or earthing as direct electrical contact with the Earth's surface. Small experimental studies have explored whether grounding influences markers such as **cortisol rhythms**, **heart rate variability**,

inflammation, and autonomic balance. Some reports suggest changes in these measures, including during sleep, but the evidence remains limited, heterogeneous, and not yet strong enough for firm conclusions (Ghaly & Teplitz, 2004; Chevalier et al., 2012; Oschman et al., 2015).

One proposed mechanism is that contact with the Earth's surface may influence **electron transfer and redox balance.** That idea is biologically interesting, but again – it's still just one of several possible hypotheses, not a specific pathway.

Still, grounding is simple enough that I include it whenever possible. You obviously cannot do it in an airport terminal, but once you step outside, walking barefoot on grass, sand, or soil for a few minutes is an easy addition to a morning routine. I do it often – usually while getting sunlight, walking, or doing a short yoga sequence – and it feels like a simple way to reconnect with the external environment after travel.

There is also a useful contextual point. The multi-day camping study mentioned earlier – the one showing that living outdoors for several days can realign human circadian rhythms with the solar cycle – was driven primarily by natural light-dark exposure (Wright et al., 2013). Participants showed a clear shift in melatonin timing, with biological night beginning earlier and aligning more closely with sunset. In other words, their internal timing moved toward the external environment without any deliberate intervention beyond changing the conditions they were living in.

That study does not isolate grounding as a variable, and light exposure was almost certainly the dominant factor. But the environment in that experiment was not reduced to light alone. Participants were continuously exposed to a full set of natural inputs – daylight intensity, darkness at night, temperature variation, movement across natural terrain, and direct physical contact with the ground. It is therefore difficult to completely separate these signals. While grounding has not been shown to independently drive circadian shifts in the way light does, some experimental and theoretical work suggests that biological systems may respond to electrical and electromagnetic aspects of the environment as well. That does not establish a mechanism, but it places grounding within a broader context of environmental inputs that may contribute, even if modestly, to overall physiological alignment.

Whether direct contact with the Earth contributes meaningfully to circadian regulation on its own is still an open question. But as part of that larger context, it becomes more interesting.

For me, that is enough. It costs nothing, takes no effort, and aligns with conditions the body already recognizes. I do it regularly – not because I expect a measurable effect every time, but because it just naturally fits into a pattern that tends to work.

Putting it together: what you're really doing

At some point, all of this stops being about individual tricks. What you're actually managing is timing – how different signals line up across the day.

Under stable conditions, those signals reinforce each other. Light, food, movement, and rest all point in the same direction.

Travel breaks that alignment.

You can be exhausted at midnight under airport lighting that looks like noon. You can eat dinner when your body still treats it as the middle of the night. The signals are there – they're just out of sync.

Temporal control is simply reducing that mismatch.

In practice, that usually means focusing on a few strong anchors:

- **make days bright** – real outdoor light early in the day is the strongest signal you can give the central clock
- **make nights dark** – reduce artificial light exposure when the day should end
- **keep food aligned with the active phase** – avoid heavy meals during what is still biological night

Everything else – movement, morning walks, grounding, routines – helps reinforce those same signals.

Circadian Supplements

Supplements belong at the end of this chapter for a reason – they do not replace light, darkness, or timing. At best, they support a system you have already made coherent. When environmental cues are aligned, certain compounds can occasionally reinforce or nudge the system in the desired direction. They remain secondary.

It is also important to distinguish between circadian tools and sleep tools. Many compounds commonly included in “sleep stacks” do not influence circadian phase at all. They change arousal state – they make the nervous system calmer, more sedated, or less reactive. Those substances can be useful – especially when

sympathetic activation remains high on the road – but they belong in the next chapter on autonomic regulation rather than here.

The list of compounds that meaningfully interact with circadian timing itself is surprisingly short.

Melatonin

Melatonin has already appeared several times in this book because it sits directly at the intersection of light exposure and circadian timing. Endogenous melatonin secretion is tightly linked to **retinal light input through the melanopsin pathway** projecting to the SCN, and artificial light exposure during the biological night – particularly blue-enriched light – can suppress melatonin production and delay circadian timing (Gooley et al., 2011; Chang et al., 2015).

That same physiology is the reason melatonin supplementation is sometimes used as a circadian phase-shifting tool, particularly during jet lag.

Several controlled studies have shown that melatonin taken at the appropriate circadian time can **advance or delay circadian phase** and reduce subjective jet lag symptoms after long-haul travel (Arendt et al., 1987; Herxheimer & Petrie, 2002; Sack et al., 2007). Meta-analyses of clinical trials similarly conclude that melatonin can improve sleep onset latency and reduce jet lag severity when taken close to the target bedtime at the destination, especially after eastbound travel where circadian phase advancement is required (Herxheimer & Petrie, 2002).

Its usefulness still depends on context. If environmental signals remain contradictory – for example, if a traveler takes melatonin and then continues exposing themselves to bright artificial light late at night – the intervention becomes physiologically less coherent. One signal comes from the supplement, the opposite signal comes from the environment, so the circadian system receives mixed instructions.

That is why melatonin works best when paired with light control rather than used against a strongly lit evening environment. In practice, environmental signals usually matter first.

Another consideration is long-term routine use. Melatonin is generally considered safe for short-term circadian adjustment, and meta-analyses show modest benefits for sleep onset and duration (Ferracioli-Oda et al., 2013). However, evidence for long-term nightly supplementation is more limited, and responses appear to vary between individuals. Higher doses may be more likely to cause unwanted effects

such as morning grogginess, vivid dreams, or residual sleepiness rather than meaningfully improving sleep further.

Many clinicians and circadian researchers therefore treat melatonin primarily as a **situational circadian tool** rather than a nightly sleep aid.

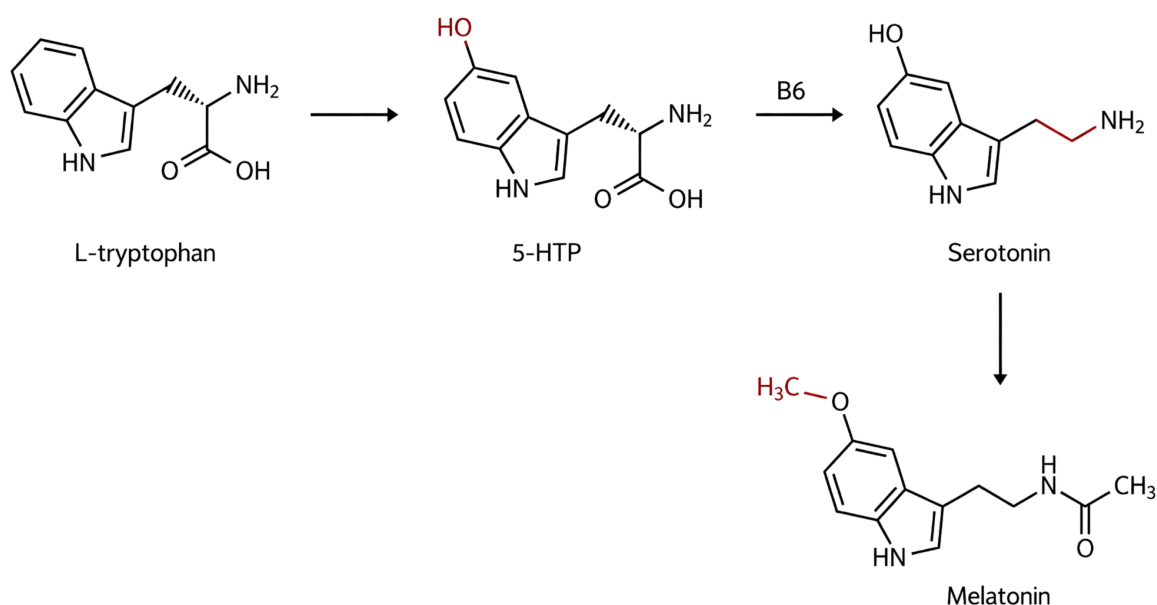
Personally, I rarely use melatonin. In most situations I prefer to rely first on the stronger regulators discussed earlier in this chapter – light exposure, darkness, movement timing, and meal timing. When those signals are aligned, the body usually restores its own melatonin rhythm naturally.

However, it may work differently for you. Some eastbound journeys are more demanding, and your body may benefit from help. Or, thanks to melatonin, you might even be able to sleep better, for example, in a tiny, uncomfortable airplane seat where you otherwise wouldn't be able to sleep at all. That experiment is yours to run.

Serotonin precursors: L-tryptophan and 5-HTP

Another group of compounds sometimes used for sleep or circadian support involves the biochemical pathway that leads to melatonin production.

Melatonin is synthesized from serotonin, which itself is derived from the essential amino acid L-tryptophan. In simplified form, the pathway looks like this:



Serotonin and melatonin synthesized from L-tryptophan through a sequence of enzymatic steps linking metabolism to neurotransmission and circadian regulation.

Because of this pathway, some people experiment with precursors such as L-tryptophan or 5-HTP instead of taking melatonin directly. The logic is straightforward: by supplying earlier steps in the pathway, the organism retains greater regulatory flexibility. Instead of externally imposing melatonin levels, the body can synthesize serotonin and melatonin according to its own enzymatic and circadian signals.

In theory, this may allow endogenous regulatory systems – **including circadian regulatory pathways** – to determine how much of each compound is required at a given time.

Several experimental studies have investigated whether these precursors influence sleep and circadian physiology. Early human experiments suggest that L-tryptophan supplementation can shorten sleep latency and increase subjective sleepiness, particularly when taken in the evening (Hartmann, 1982; Silber & Schmitt, 2010). However, the magnitude of these effects appears modest and varies depending on dose, timing, and baseline physiology.

Similar pathways have been explored with 5-HTP, which bypasses the rate-limiting enzymatic step converting tryptophan into serotonin and therefore increases central serotonin availability more directly.

Small clinical trials using 5-HTP in the range of roughly 100–300 mg in the evening **have reported possible effects on sleep onset and REM sleep**, although the findings are heterogeneous and not consistently replicated (Wyatt et al., 1971; Rondanelli et al., 2011). Lower doses have also been explored in some settings to influence serotonergic tone without producing strong sedative effects, but these effects are less well characterized.

These compounds should not be thought of as simple “sleep triggers.” The physiological response is dose-dependent, context-dependent, and significantly shaped by the state of the underlying system.

At lower doses, tryptophan or 5-HTP may simply increase serotonin availability, which can affect mood, cognition, or appetite regulation without necessarily producing sleepiness. At higher doses – particularly when taken in the evening – the increased serotonergic substrate may lead to greater melatonin synthesis later in the pathway and support sleep onset.

The outcome of ingesting a serotonin precursor depends on enzymatic conversion rates, circadian phase, light exposure, and the individual's baseline neurochemistry.

Responses vary widely. Some people report mild improvements in sleep onset or mood when using these precursors, while others notice little effect. In certain cases higher serotonergic activity may even feel stimulating rather than calming, especially when doses are taken earlier in the day.

I tend to treat these compounds as experimental tools rather than core circadian interventions. They can be interesting for individuals who want to explore the serotonin-melatonin pathway, but they are not required for stabilizing circadian timing.

A note on caffeine

One molecule deserves mention here not because it supports circadian timing, but because it often disrupts it – **caffeine**.

Caffeine works primarily by blocking adenosine receptors in the brain. Adenosine is a molecule that accumulates during wakefulness and contributes to the sensation of sleep pressure. By blocking this signal, caffeine temporarily reduces fatigue and increases alertness. It is no surprise that caffeine is such a popular stimulant and that many people use it every day.

The circadian problem appears when caffeine is consumed late in the day. By delaying the buildup and perception of sleep pressure, caffeine can postpone sleep onset and indirectly shift circadian timing. Research has also shown that evening caffeine intake can **delay melatonin secretion and push circadian phase later**, especially when taken close to bedtime (Drake et al., 2013).

Even little kids understand the obvious version of this: caffeine before bed makes sleep harder. What becomes more relevant for travelers is that caffeine does not merely influence sleep onset. It can interfere with the adjustment you are trying to achieve. When the goal is to stabilize biological night in a new time zone, late caffeine intake can push the internal clock in the opposite direction.

That makes caffeine more consequential on the road than it might feel in everyday life.

Personally, I no longer use caffeine itself. I still enjoy coffee – but only decaffeinated coffee – because I like the flavor and the complex polyphenolic compounds present

in the beverage. But I avoid caffeine as a stimulant because even small amounts can make me stressed and make my circadian timing less predictable.

Autonomic Regulation and Sleep: Sympathetic–Parasympathetic Balance During Travel

Timing your regimen and protecting circadian rhythm matter enormously.

But circadian timing is only part of the story.

Even when light exposure is managed well, meals are aligned with local time, and the most obvious circadian disruptors are avoided, another physiological pattern often appears: the body remains activated when it should be settling down. We all know this very well – sleep opportunities exist, the lights are off, the clock says it is night – and yet the nervous system continues to behave as if the day has not ended.

This reflects a second layer of regulation: the balance between **sympathetic and parasympathetic activity** within the autonomic nervous system.

The **autonomic nervous system** regulates processes that operate largely outside conscious control – heart rate, vascular tone, digestion, respiratory patterns, and many aspects of hormonal signaling. Within this system, two broad modes dominate. The sympathetic branch mobilizes the organism for action, increasing alertness, cardiovascular output, and metabolic readiness. The parasympathetic branch, mediated largely through the vagus nerve, promotes recovery, digestion, and the transition toward rest.

However, these states are not moral categories. Neither is “good” or “bad.” They are just regulatory modes that evolved to match different environmental demands. Sympathetic activation prepares the organism to engage with the environment. Parasympathetic activation allows repair, energy conservation, and restoration.

Under stable conditions, these states cycle naturally across the day.

Morning light exposure, physical movement, and rising cortisol levels promote sympathetic activation during the active phase of the circadian cycle. As daylight declines and behavioral stimulation decreases, parasympathetic tone gradually increases, facilitating the transition toward sleep. Circadian timing and autonomic

regulation normally move together: the biological clock signals that night is approaching, and the nervous system gradually downshifts into a state compatible with rest.

Modern life can disrupt that synchrony in many ways, and traveling is one of the most effective ways to do so.

Think back to the last time you traveled. From an evolutionary perspective, the pattern makes perfect sense. The nervous system interprets novelty as uncertainty, and uncertainty historically carried survival relevance. When the brain encounters unfamiliar environments, unfamiliar faces, unfamiliar rules, and unfamiliar sounds, the autonomic system shifts toward vigilance.

Studies of flight crews and shift workers show **disrupted sleep, fatigue, and physiological stress responses** during irregular schedules, often accompanied by autonomic dysregulation (Gander et al., 2001). Even without obvious psychological stress, environmental novelty alone increases cognitive and physiological load.

This contributes to the distinct quality of travel fatigue compared to ordinary tiredness. The body may be physically exhausted while the nervous system remains partially activated. Heart rate stays slightly elevated. Sleep becomes lighter. Small environmental stimuli – hallway noise, light leaks, unfamiliar bedding – trigger stronger arousal responses than they would at home.

Neurophysiological research has also documented something interesting known as the **“first-night effect.”** During sleep in unfamiliar environments, brain activity can become asymmetrical, with one hemisphere remaining more responsive to external stimuli. In laboratory studies, this looks like a low-level monitoring mode – as if part of the brain stays slightly alert during the first night in a new place (Tamaki et al., 2016).

Personally, I am not especially sensitive to this problem. I usually do not struggle much to fall asleep in new places. But I have experienced something close to it a few times. A few nights in hotels felt almost like having caffeine before bed – as if part of my brain remained awake throughout the sleep period and never really rested.

From an evolutionary perspective, that mechanism likely served a protective function. Sleeping in an unfamiliar location carried risk, so maintaining partial vigilance increased survival probability. In modern hotel rooms the risk is minimal, but the physiology persists.

And this is where autonomic regulation shows up in a more practical way. Even when you are tired, the system does not fully switch off. The body lies down, but part of the brain keeps scanning the environment. Sleep becomes lighter, more fragmented, and less restorative – not because something is “wrong,” but because the system is still slightly on guard.

Furthermore, **deep sleep**, slow-wave activity, and parasympathetic dominance are closely linked. Experimental research shows that parasympathetic activation increases during slow-wave sleep and is associated with reduced heart rate and increased vagal tone (Trinder et al., 2001). When sympathetic activation remains elevated, sleep architecture becomes fragmented and restorative stages may be reduced.

Travelers therefore often face two distinct sleep problems:

1. **Circadian misalignment** – the body does not know what time it is.
2. **Autonomic activation** – the body knows it is night but cannot downshift.

The distinction matters because the solutions differ. Circadian problems are addressed primarily through light exposure, darkness, and timing cues. Autonomic problems require tools that help the nervous system change state.

Those tools can be behavioral, physiological, or occasionally biochemical. But before we get to them, it is worth looking at one concept that has become increasingly important in sleep and stress research – **heart rate variability**.

Heart Rate Variability

Heart rate variability (HRV) is one of the most useful indirect markers of autonomic balance. HRV refers to the natural variation in time intervals between consecutive heartbeats. Contrary to what intuition might suggest, a perfectly regular heartbeat is not a sign of optimal regulation. In healthy individuals, the intervals between beats fluctuate slightly from moment to moment as the autonomic nervous system continuously adjusts cardiovascular output.

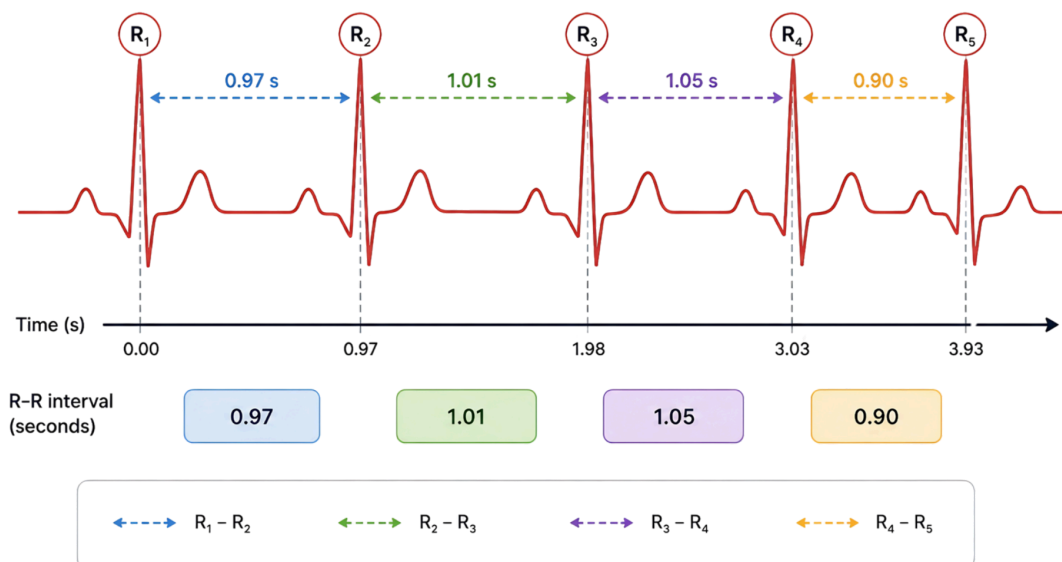
These fluctuations reflect the dynamic interplay between sympathetic and parasympathetic signals acting on the sinoatrial node of the heart.

A resilient cardiovascular system therefore produces slightly irregular beat-to-beat intervals – for example **0.97 seconds, 1.01 seconds, 1.05 seconds, 0.90 seconds** – rather than a perfectly mechanical pattern such as 1.00, 1.00, 1.00, 1.00. It is precisely this variability that can distinguish, for example, a relaxed athlete from a

stressed, sleep-deprived person. One heart beats like an anxious metronome; the other adapts from moment to moment. I hope this example has helped to make the concept more intuitive.

Heart Rate Variability (HRV)

HRV reflects the variation in time intervals between consecutive heartbeats (R-R intervals).



Beat-to-beat intervals (R-R) are not uniform; their subtle fluctuations reflect continuous autonomic modulation rather than mechanical regularity.

Higher HRV values generally correspond to **stronger parasympathetic influence and greater physiological flexibility**, whereas reduced HRV is often associated with sympathetic dominance, fatigue, or physiological stress. Numerous studies have linked HRV metrics to recovery status, stress resilience, and sleep quality (Shaffer & Ginsberg, 2017).

For travel, HRV provides a useful window into how the body is responding to environmental change.

What matters is not the number itself, but how it moves relative to your baseline. If HRV drops after travel, poor sleep, or a stressful day, it usually means the system is still under load. In practice, that can influence simple decisions – whether to train hard or take it easier, whether to prioritize recovery instead of stimulation, or whether the environment is still asking more from the system than it can comfortably handle. HRV does not tell you what to do. It gives you a second opinion when your subjective sense is unclear or biased.

Modern wearables increasingly include HRV tracking as part of sleep and recovery monitoring.

Nevertheless, no wearable metric should be treated as absolute truth. Sensor limitations, measurement noise, and individual variability all influence readings. Used consistently over time, however, HRV tracking can reveal patterns that align surprisingly well with subjective experience – poor sleep, intense travel stress, or illness often correspond with noticeable HRV changes.

That kind of feedback can be valuable in unstable environments. It does not replace self-awareness, but it often reveals patterns that subjective perception alone might miss. If you want a device that tracks vital signs, it is worth doing some research and choosing one that measures HRV reliably. In my opinion, that metric matters far more than step count or calories burned.

Breathing as an Autonomic Lever

If you want to influence your autonomic state, the simplest place to start is breathing. And really, I am not exaggerating.

Respiration occupies a unique position in physiology. Most autonomic processes operate entirely outside conscious control, but breathing sits at the boundary between voluntary and involuntary regulation. Because respiratory patterns interact directly with vagal nerve activity, altering breathing rhythm can influence autonomic balance within minutes.

There are countless breathing exercises online, often presented with a wide range of claimed benefits. Many of these effects are often exaggerated and far from reality. Since this book is about travel, and this chapter is about autonomic regulation, it makes sense to keep it simple and science-based.

Slow, controlled breathing **increases vagal tone and enhances parasympathetic activity**. Experimental studies have shown that breathing frequencies around five to six breaths per minute can significantly increase heart rate variability and baroreflex sensitivity, both markers associated with parasympathetic dominance (Lehrer & Gevirtz, 2014).

That is one reason slow breathing appears across so many traditions – from yogic pranayama to modern clinical relaxation protocols.

One particularly simple technique is the **physiological sigh**, a breathing pattern involving two short inhalations followed by a longer exhalation. Neurophysiological

research suggests that this pattern helps reinflate collapsed alveoli in the lungs while also activating parasympathetic pathways associated with relaxation (Huberman et al., 2023).

Another widely used method is **nadi shodhana**, or alternate-nostril breathing, a pranayama practice that has been studied for its effects on autonomic balance. Several small clinical studies have reported increases in parasympathetic activity and reductions in stress markers following regular practice (Telles et al., 2013). It is also one of my personal favorite breathing practices. I use it frequently and often include it in my daily routine because it reliably produces a noticeable calming effect.

The technique itself is simple. Sitting comfortably with the spine upright, you gently close the right nostril with your thumb and inhale slowly through the left nostril. Then you close the left nostril with a finger, release the right nostril, and exhale through the right side. The next inhalation is taken through the right nostril, after which the nostrils switch again and the breath is exhaled through the left. This alternating pattern continues for several minutes at a slow, relaxed pace. In many traditional versions of the practice, brief pauses are also introduced between breaths – holding the breath gently after inhalation or exhalation for a few seconds before continuing the cycle. These pauses, sometimes referred to as **kumbhaka** in yogic breathing practices, can slow the respiratory rhythm even further and deepen the calming effect when performed comfortably and without strain.

Whether for yogis or travelers, the appeal of breathing interventions is obvious – they require no equipment, no supplements, and no environmental control. They are available anywhere – in a crowded airport, on a long flight, in a hot unfamiliar room, or at night when sleep should come but doesn't.

A few minutes of slow breathing, or a simple pattern like nadi shodhana or a physiological sigh, can be enough to reduce the activation level of the system. When the environment cannot be adjusted and the body is still reacting to it, breathing remains one of the few levers you can use immediately.

Supplements for autonomic down-regulation

Behavioral regulation should always come first. Light exposure, timing, movement, and breathing influence the nervous system more powerfully than most molecules. But there are situations in which environmental control alone is not enough.

Travel environments are unpredictable. You may arrive in a noisy city after a long flight, be mildly dehydrated, sleep in an unfamiliar bed, or try to rest while the

nervous system is still partially activated from the day's stimuli. In those situations some travelers experiment with compounds that influence inhibitory neurotransmission, stress hormone regulation, or metabolic resilience.

These substances do not substitute for sleep itself. Nor do they solve circadian misalignment. Their role is narrower – they may help the system shift toward a state compatible with rest.

Several compounds appear repeatedly in discussions of sleep and autonomic regulation. Some have substantial historical use, others have modest experimental evidence, and a few remain controversial. Understanding their mechanisms and limitations is more useful than memorizing a supplement stack.

Kava (*Piper methysticum*)

Few plants illustrate the intersection of traditional medicine, pharmacology, and controversy as clearly as kava.

Kava, derived from the roots of the plant *Piper methysticum*, has been consumed for centuries in the South Pacific as a ceremonial and social beverage. In its traditional preparation, the lateral roots of the plant are ground and mixed with water to produce a mildly sedative drink with anxiolytic properties.

The pharmacological effects of kava are attributed to a group of compounds known as **kavalactones**, including kavain, dihydrokavain, methysticin, and yangonin. These molecules interact with several neural targets relevant to autonomic regulation.

Experimental studies suggest that kavalactones:

- positively modulate **GABA-A receptors**, increasing inhibitory neurotransmission (Davies et al., 1992)
- inhibit **voltage-gated sodium and calcium channels**, reducing neuronal excitability (Magura et al., 1997)
- influence neural circuits involved in **stress and emotional regulation**

The combined result is reduced neural hyperexcitability without the profound sedation seen with many pharmaceutical anxiolytics. Clinical trials have reported modest reductions in anxiety symptoms compared with placebo in several populations (Sarris et al., 2013).

For travelers experiencing persistent autonomic activation – the familiar “tired but wired” state after long journeys – this mechanism can theoretically support the shift toward parasympathetic dominance.

Kava is also one of my personal favorite herbal remedies in the context of travel. I often use it before long journeys or early flights. The reason is not about sleep support, but rather a complete shift in mindset. Travel mornings are often chaotic – you wake early, you are slightly sleep-deprived, and you are rushing through logistics, airports, security lines, and unfamiliar environments. In that state I may still feel physically tired, but kava changes the mental experience in a very noticeable way. The fatigue remains, but the usual irritability and nervous tension are much lower. I am tired, but not stressed. The mind feels calmer, and the whole situation becomes easier to navigate without the sharp, overactivated edge that sleep deprivation sometimes creates.

This subjective effect is consistent with the general pharmacology of kavalactones, which tend to reduce neural excitability while preserving mental clarity. Many people therefore experience kava less as a sedative and more as a shift toward calm alertness.

However, kava also carries one of the most debated safety profiles in herbal medicine.

During the early 2000s, several reports linked kava consumption to **cases of liver toxicity**. These concerns led to regulatory restrictions or temporary bans in several countries, particularly within the European Union. Subsequent investigations suggested that many adverse cases involved poor-quality extracts, incorrect plant parts, or co-ingestion with alcohol and hepatotoxic medications (Teschke et al., 2008).

Traditional preparations rely specifically on noble cultivars and the lateral roots of the plant. Some commercial extracts historically included stem peelings or non-noble varieties containing higher concentrations of potentially hepatotoxic compounds.

Because of this history, quality control is essential. Reliable kava products should specify cultivar type, extraction method, and kavalactone profile. If you are careful and thorough in your selection, I suspect you will understand very quickly why so many people become fond of kava.

Ashwagandha (*Withania somnifera*)

Another widely discussed adaptogenic plant is ashwagandha, a traditional Ayurvedic herb used for centuries to support stress resilience and recovery.

The primary active compounds in ashwagandha are **withanolides**, steroidal lactones that appear to influence several physiological systems associated with stress regulation. Controlled studies have reported reductions in perceived stress and serum cortisol levels following supplementation in chronically stressed individuals (Chandrasekhar et al., 2012).

Cortisol plays an important role in circadian physiology. Under normal conditions, cortisol peaks shortly after waking and declines gradually throughout the day. Chronic stress, irregular schedules, or travel fatigue can flatten or distort this rhythm.

By modestly lowering elevated cortisol levels, ashwagandha may indirectly support the transition toward sleep in individuals whose sympathetic activation remains elevated in the evening.

Responses vary significantly between individuals.

Some people report improved sleep onset and reduced mental agitation when taking ashwagandha in the evening. Others experience gastrointestinal effects or altered energy patterns when using it regularly. Personally, I find that frequent use can produce noticeable digestive stimulation – increased urination or bowel activity – which makes it less appealing to me as a daily supplement.

For that reason, I view ashwagandha as a contextual tool rather than a universal solution.

Reishi (*Ganoderma lucidum*)

Another interesting adaptogenic substance that can be used to support autonomic regulation during periods of stress or travel fatigue is reishi, a medicinal mushroom traditionally used in Chinese and Japanese herbal medicine. In classical pharmacological texts it is often described as a **shen-tonifying** herb. In traditional terminology, *Shen* roughly refers to the state of mental calmness, emotional stability, and clarity of consciousness – what we might describe in modern terms as balanced nervous system regulation. Herbs described as supporting *Shen* are traditionally used for states of restlessness, poor sleep, irritability, and mental agitation.

Reishi (*Ganoderma lucidum*) has attracted increasing interest in modern research and is one of the more extensively studied medicinal or adaptogenic mushrooms in

both human and experimental literature. The primary bioactive compounds in reishi include triterpenoids (ganoderic acids) and β -glucan polysaccharides. These compounds appear to influence several physiological systems at once, including immune signaling, inflammatory pathways, and central nervous system activity.

Animal studies and preliminary human trials suggest that reishi extracts may interact with **GABAergic signaling pathways**, potentially contributing to mild sedative or anxiolytic effects (Zhu et al., 2019). Several experiments also indicate that reishi extracts can increase non-REM sleep duration and shorten sleep latency in rodent models (Gao et al., 2004). These effects are thought to arise from modulation of cytokine signaling and neurotransmitter balance rather than direct circadian phase shifting.

Unlike classical sedatives, reishi does not appear to function as a strong hypnotic. Its effects are generally subtle and cumulative. In traditional practice it is often consumed regularly over longer periods rather than used as an acute sleep aid.

Because of this slower profile, reishi is closer to adaptogenic plants like ashwagandha than to compounds that directly induce sleep. Some individuals report improvements in sleep quality and perceived recovery when using reishi extracts, particularly during periods of elevated stress.

As with many medicinal mushrooms, however, extract quality matters significantly. The active triterpenoids are concentrated primarily in the fruiting body and spores of the mushroom, and extraction methods strongly influence the final composition of the product. Standardized dual extracts (water and alcohol extraction) are generally considered more pharmacologically complete than simple powdered mushroom preparations.

Personally, I really like reishi and use it regularly. It's one of the adaptogens I trust the most, especially during periods of a hectic lifestyle, when everything is slightly out of sync and the goal is to bring the system back into a more stable state.

Glycine

Among the compounds most consistently associated with sleep onset is the simple amino acid **glycine**.

Glycine functions as both a neurotransmitter and a metabolic intermediate. Within the central nervous system it acts primarily as an inhibitory neurotransmitter, particularly in the spinal cord and brainstem, while also serving as a **co-agonist at NMDA receptors**. Through these mechanisms, glycine participates in neural

signaling that regulates excitability, sensory processing, and aspects of autonomic balance.

Several human studies have reported that glycine ingestion before bedtime may improve subjective sleep quality and reduce sleep latency (Yamadera et al., 2007). One of the most plausible mechanisms involves thermoregulation. Sleep onset is associated with a gradual decline in core body temperature, a process coordinated by the hypothalamus and closely tied to circadian and autonomic regulation. Glycine appears to **enhance peripheral vasodilation**, particularly in distal skin regions, facilitating heat dissipation and thereby accelerating the temperature decline that normally accompanies sleep initiation. This thermoregulatory shift is thought to support the transition from sympathetic activation toward parasympathetic dominance.

From an autonomic perspective, this matters because the body often struggles to enter sleep not simply because the brain is “awake,” but because the physiological signals that define nighttime physiology have not yet fully engaged. Core temperature remains too high, peripheral circulation remains relatively constricted, and sympathetic tone is still elevated. By promoting heat dissipation and supporting inhibitory neural signaling, glycine may help the system shift toward the physiological state that sleep requires.

In practice, glycine often produces a mild calming effect without heavy sedation. Unlike many sleep aids, it rarely causes morning grogginess and appears metabolically well tolerated in moderate doses. For people who are traveling, mildly stressed, or dealing with disrupted sleep schedules, this gentle shift toward parasympathetic dominance is often more useful than stronger sedative interventions.

Glycine is also interesting because it sits at the intersection of neurotransmission and structural metabolism. Although the human body can synthesize glycine endogenously and obtain small amounts from dietary protein, a large proportion of the glycine pool is continuously used for structural maintenance – particularly collagen turnover. Collagen, the most abundant protein in the human body, contains roughly one-third glycine residues. Because connective tissues, skin, bone, cartilage, and the extracellular matrix undergo constant remodeling, a substantial fraction of available glycine is effectively allocated to maintaining these structures.

This becomes more interesting when considered alongside **methionine metabolism**. Diets high in muscle meat tend to provide relatively large amounts of methionine but comparatively little glycine. Experimental work in animals has shown

that methionine restriction is associated with improved metabolic health and extended lifespan, although the mechanism is still being explored. One interpretation is that it is not methionine alone, but the balance between methionine and glycine that matters. Glycine participates in pathways that **buffer excess methyl groups and support metabolic equilibrium**. Increasing glycine intake may therefore partially offset a methionine-heavy dietary pattern, at least at the level of biochemical balance.

Some researchers have argued that modern diets may provide less glycine than the body historically obtained from whole-animal foods such as skin, cartilage, and connective tissue. This hypothesis has led to the idea that glycine could become conditionally limiting in certain physiological contexts, particularly when collagen turnover and glutathione synthesis both draw from the same metabolic pool (Meléndez-Hevia et al., 2009).

Beyond its role in collagen, glycine also contributes to **glutathione** synthesis, one of the body's major intracellular antioxidant systems. Glutathione participates in redox regulation, detoxification, and mitochondrial protection (we will discuss glutathione in more detail in the chapter on *Oxidative Load and Recovery*).

So, to sum it all up – glycine shows up in structural maintenance, antioxidant defense, neurotransmission, and even temperature regulation. If that still doesn't interest you, then I'm not sure what will. For something so simple, it is doing a surprising amount of work.

For the purposes of this chapter, however, the most relevant effect remains its influence on the transition into sleep. Glycine behaves like a metabolic nudge – a small adjustment that can help the autonomic system move toward the nighttime state when the rest of the environment is already encouraging it.

It is also relatively inexpensive. If you have room in your luggage, packing a small amount of glycine is a very reasonable idea.

Magnesium

Magnesium occupies a strange position in modern health culture. It is one of the most fundamental minerals in human physiology, involved in hundreds of enzymatic reactions across the body, yet it is often reduced to a single line in sleep advice – ***take magnesium, it helps you relax***.

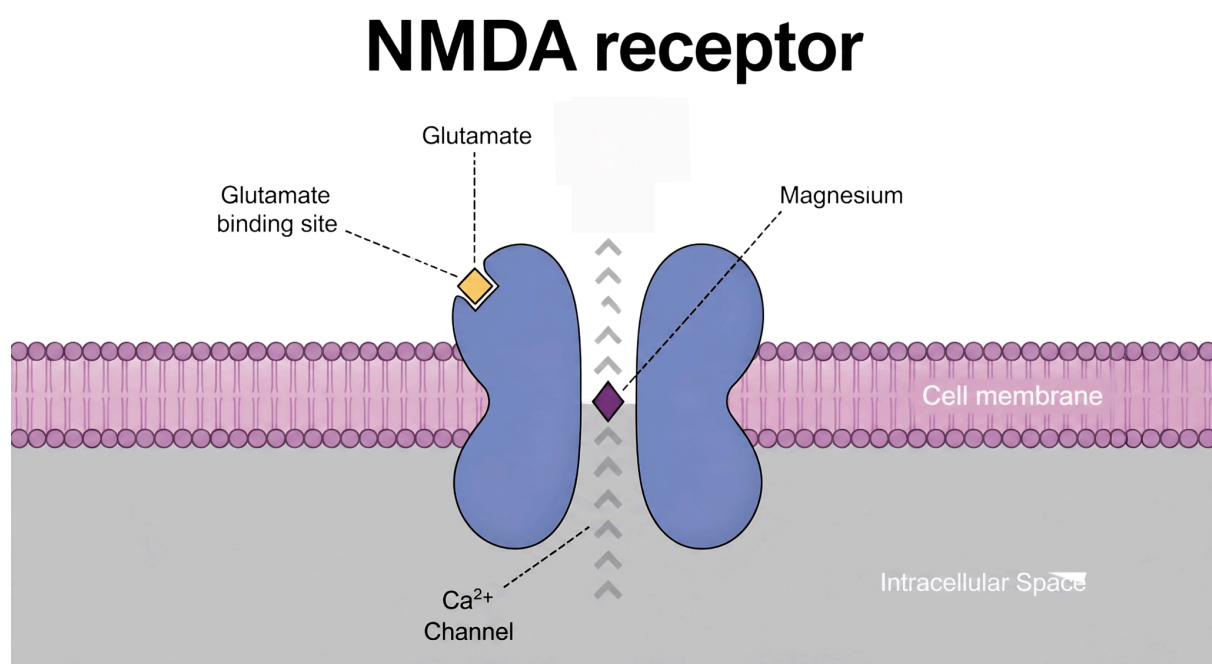
That explanation is not exactly wrong, but it misses the scale of what magnesium is actually doing.

Magnesium is involved in energy metabolism, mitochondrial function, neuronal signaling, muscle contraction and relaxation, DNA synthesis, insulin signaling, vascular tone, and bone metabolism. Estimates suggest that it participates as a cofactor in more than 300 enzymatic reactions in the human body (de Baaij et al., 2015). Roughly half of total body magnesium is stored in bone, while the rest is distributed across muscles and soft tissues.

Reducing all of that to “something that helps you sleep” is a bit like describing oxygen as a breathing aid.

The reason it appears in this chapter is not that magnesium matters only for autonomic regulation. It appears here because one of its most noticeable day-to-day effects is its influence on nervous system excitability and the balance between sympathetic and parasympathetic activity.

Magnesium acts as a natural modulator of neuronal excitability. One of its best-known roles involves regulating **NMDA receptors** (already mentioned in the section on glycine), which participate in excitatory glutamatergic signaling in the brain. Under normal physiological conditions, magnesium sits inside the NMDA receptor channel and helps regulate its activity. When magnesium levels are low, excitatory signaling becomes easier to trigger, which may contribute to neural hyperexcitability and stress responses (Volpe, 2013).



Magnesium regulates NMDA receptor activity by occupying the channel and limiting calcium influx, helping prevent excessive excitatory signaling.

Magnesium also interacts with GABAergic signaling, calcium channels, and stress-related neuroendocrine pathways. From the perspective of autonomic regulation, that translates into something very practical: magnesium tends to reduce excessive neural activation and support the shift toward parasympathetic dominance.

That's why magnesium keeps coming up whenever people talk about stress, sleep, and recovery.

And even that is just one layer.

One of the most interesting features of magnesium is its role in **ATP metabolism**. The biologically active form of ATP in the cell is not free ATP, but a complex known as **Mg-ATP**. Magnesium availability therefore directly influences cellular energy processes. It is also essential for muscle function, insulin signaling, and vascular health. Low magnesium status has been associated with increased risk of hypertension, insulin resistance, and metabolic syndrome in several epidemiological studies (Barbagallo & Dominguez, 2010).

Bone health is another major but often overlooked dimension. Around 50–60% of total body magnesium resides in bone tissue, where it contributes to structural integrity and interacts with calcium metabolism and vitamin D regulation. Magnesium deficiency has been associated with reduced bone mineral density and increased fracture risk (Castiglioni et al., 2013).

So yes, magnesium can help you relax.

But if that is the only reason you are taking it, you are missing most of the picture.

Magnesium forms

One confusing aspect of magnesium supplementation is that magnesium rarely appears as a pure mineral in supplements. Instead, it is bound to another molecule – forming what is known as a **magnesium salt or chelate**.

The molecule attached to magnesium influences several things:

- absorption efficiency
- gastrointestinal tolerance
- tissue distribution

- and sometimes functional effects

Some of the most common forms include magnesium oxide, citrate, malate, taurate, glycinate (bisglycinate), and L-threonate.

Magnesium oxide is inexpensive but poorly absorbed.

Magnesium citrate is well absorbed but often acts as a laxative because of its osmotic effects in the gut.

Magnesium malate and magnesium taurate are sometimes used for metabolic and cardiovascular contexts.

For the purposes of autonomic regulation and nervous system support, two forms are particularly interesting: **magnesium glycinate (bisglycinate)** and **magnesium L-threonate**.

Magnesium glycinate: magnesium plus glycine

Magnesium glycinate – often written as magnesium bisglycinate – is a chelated form in which magnesium is bound to the amino acid glycine.

This is interesting for two reasons.

First, chelation with glycine tends to improve gastrointestinal tolerance and absorption compared with some inorganic magnesium salts. Chelated forms are generally more stable during digestion and may be less likely to produce the laxative effects associated with citrate or oxide.

Second, glycine itself has physiological effects. As discussed above, glycine functions as an inhibitory neurotransmitter and plays roles in thermoregulation, sleep initiation, collagen synthesis, and glutathione production.

The interesting part is not that both are present, but how their effects overlap.

In practice, this creates a subtle stacking effect. Magnesium contributes to reduced neuronal excitability and muscle relaxation, while the glycine component may support inhibitory signaling and thermoregulatory processes that facilitate sleep onset.

That combination is one reason magnesium glycinate has become one of the most widely recommended forms for evening use.

Personally, this is the form I use most consistently. Magnesium glycinate is part of my nightly routine, and I take it almost every evening. It produces a calm, physiologically grounded effect rather than heavy sedation.

Magnesium L-threonate: the brain-focused magnesium

Magnesium L-threonate is a newer and more specialized form of magnesium that has attracted attention for its potential effects on brain magnesium levels.

L-threonate is a metabolite of vitamin C. When bound to magnesium, it forms a compound that appears capable of increasing magnesium concentrations in the brain more effectively than some other magnesium salts.

Animal experiments and early studies suggested that magnesium L-threonate supplementation increased brain magnesium levels and improved synaptic plasticity and learning in rodents (Slutsky et al., 2010). These findings generated substantial interest in the compound as a potential cognitive-support supplement.

The literature around magnesium L-threonate, however, deserves careful interpretation. Some of the most widely cited studies were funded or supported by groups involved in the development and commercialization of the compound.

That does not automatically invalidate the findings – industry funding is common in supplement research – but it does mean the results should be interpreted cautiously and replicated independently.

Magnesium L-threonate is usually presented as a brain-focused form of magnesium, used more for cognition than for physical relaxation.

By contrast, magnesium glycinate tends to feel more physically grounding – useful for muscle tension, nervous system downregulation, and evening relaxation.

That distinction is not absolute, but it roughly matches how many people describe the two forms.

Personally, I treat magnesium L-threonate as an occasional tool rather than the foundation of my magnesium routine. Most of the time I prefer forms like glycinate, which have a more noticeable whole-body effect. L-threonate is something I reach for selectively – when the cognitive angle is interesting – not something I rely on every day.

Does magnesium cross the blood–brain barrier?

Part of the appeal of magnesium L-threonate comes from how magnesium is handled in the body.

Magnesium **does cross the blood–brain barrier**, but the process is tightly regulated. Increasing magnesium levels in the blood does not automatically translate into large increases in brain magnesium.

This is the context in which L-threonate was developed. The idea was not to create something that “finally crosses into the brain,” but rather a form that might increase brain magnesium levels more efficiently under certain conditions.

That distinction is often lost in marketing. The conversation gets simplified into whether a form “crosses the blood–brain barrier,” which makes it sound as if other forms do not affect the brain at all.

That is not the case.

Forms like magnesium glycinate or citrate still influence the nervous system – just less directly. They improve overall magnesium status, which in turn affects neuronal excitability, NMDA receptor function, and autonomic balance. Glycinate also adds glycine, which has its own calming and inhibitory effects.

So the real difference is not whether magnesium reaches the brain, but how direct and targeted that effect might be.

Magnesium deficiency

Another reason magnesium supplementation is discussed so widely is that magnesium intake in modern populations often appears to fall below physiologically optimal levels.

Magnesium is present in many whole foods – particularly leafy greens, legumes, nuts, seeds, and mineral-rich water – yet several structural changes in modern food systems may reduce average intake. Agricultural soils in many regions contain lower magnesium concentrations than they did historically, partly due to long-term intensive farming practices that deplete mineral content without equivalent replenishment. At the same time, modern food processing tends to remove magnesium-rich components of plants. Grain refining, for example, strips away the bran and germ layers where a substantial proportion of minerals are stored.

What you eat is only one side of the balance. Magnesium balance is also influenced by physiological stress. Magnesium participates in hundreds of enzymatic reactions, including processes involved in ATP metabolism and stress signaling. When

sympathetic activation increases – during psychological stress, sleep deprivation, illness, or heavy physical exertion – magnesium turnover can rise as well. Alcohol consumption, certain medications, and high circulating stress hormone levels may further increase urinary magnesium excretion.

What often emerges is not a dramatic, clinically obvious deficiency, but a quieter and harder-to-detect shortfall sometimes described as **subclinical magnesium insufficiency**. Several researchers have argued that this condition may be relatively common in industrialized populations (Rosanoff et al., 2012).

One reason this matters is that the symptoms associated with low magnesium status overlap strongly with the physiological states discussed throughout this book. Magnesium insufficiency has been associated with increased neuromuscular excitability, heightened stress responsiveness, impaired sleep quality, fatigue, headaches, and irritability. Many of the signals people interpret as “being stressed,” “sleep-deprived,” or “out of balance” may therefore be influenced at least in part by magnesium status.

This does not make magnesium a universal solution. Human physiology is rarely that simple. But given how deeply magnesium participates in neural signaling, energy metabolism, vascular regulation, and bone health, even modest shifts in magnesium availability can have system-wide effects.

That is why magnesium supplementation often becomes less about correcting a dramatic deficiency and more about restoring a mineral environment in which multiple physiological systems can operate more smoothly.

For me, magnesium glycinate has become part of a simple nightly routine. I take it every evening, and it’s probably the most important supplement in my stack. I take it because it quietly shows up in dozens of processes we rely on every day – from muscle relaxation to neural signaling to cellular energy metabolism. In a physiological environment already stressed by travel, sleep disruption, and irregular routines, maintaining adequate magnesium availability is one of the more straightforward ways to support system-wide stability.

Creatine and resilience to sleep loss

Creatine is usually discussed in the context of athletic performance, but its physiological relevance extends well beyond muscle metabolism.

Calling creatine a “gym supplement” misses most of what it actually does. It is part of a broader cellular energy-buffering system.

Within cells, creatine participates in the **phosphocreatine energy system**, which functions as a rapid buffer for ATP – the primary energy currency of the cell. During periods of sudden metabolic demand, phosphocreatine can donate a phosphate group to regenerate ATP almost instantly. This system operates in tissues that require rapid energy turnover, particularly skeletal muscle and the brain.

This becomes especially relevant under conditions of sleep restriction, circadian disruption, and psychological stress, all of which increase the energetic burden on the brain. Sleep loss in particular places significant strain on cognitive energy metabolism. Experimental studies examining sleep deprivation have reported that creatine supplementation may improve aspects of cognitive performance and reduce mental fatigue during periods of sleep loss (McMorris et al., 2006). The mechanism is likely related to improved energy buffering in neural tissue rather than any direct sedative or sleep-promoting effect.

Creatine does not directly make you sleep better. What it may do is support metabolic resilience when sleep is temporarily insufficient, which is a more realistic goal in many stressful or unstable conditions.

Several additional physiological roles also make creatine interesting beyond athletic performance. It appears to support **muscle recovery** and strength output during repeated effort, which is why it has been studied so extensively in sports science. It may also influence brain energetics, neuromuscular performance, and aspects of cellular hydration and osmotic balance. Some research has explored its potential role in neuroprotection and cognitive function, although that area is still evolving.

At the same time, creatine is not perfect for everyone.

Personally, I notice that creatine can disrupt my digestion if I take it regularly. For me it can feel slightly irritating to the stomach, particularly at higher doses. Interestingly, most people I know do not report this problem. In the research literature creatine is generally considered very well tolerated, and gastrointestinal discomfort seems to affect only a minority of users.

So while creatine can be useful, responses still vary.

What makes creatine useful in this context is that it does not try to force the body into sleep or sedation. It supports the energetic side of the system when sleep is temporarily compromised.

Carrying a large container of creatine in your hand luggage may not be entirely practical, but if you happen to be traveling with checked luggage, bringing a small amount can be a practical addition.

Taurine

Taurine is a sulfur-containing amino acid that plays a role in calcium signaling, membrane stability, and cellular fluid balance.

Unlike most amino acids, taurine is not used to build proteins. Instead, it functions as a free intracellular molecule involved in multiple physiological systems. High concentrations are found in excitable tissues such as the brain, retina, skeletal muscle, and heart, suggesting an important role in regulating cellular excitability and ion balance.

In the context of autonomic regulation, taurine becomes particularly interesting because it appears to interact with inhibitory neurotransmission. Experimental work indicates that taurine can activate glycine receptors and modulate **GABAergic signaling pathways**, both of which contribute to inhibitory tone in the central nervous system (El Idrissi & Trenkner, 2004). Through these mechanisms, taurine may reduce neuronal excitability and promote a shift toward parasympathetic dominance.

These effects have been explored in several experimental studies. In animal models, taurine administration has been shown to produce **anxiolytic-like effects** and to modulate GABA receptor activity in brain regions associated with stress regulation (Kong et al., 2006). Human research is more limited but still suggestive. Some small clinical and physiological studies report that taurine supplementation may influence cardiovascular autonomic regulation, including modest reductions in blood pressure and improvements in markers of autonomic balance (Zhang et al., 2004; Militante & Lombardini, 2002).

Taurine has also been investigated for its broader cardiovascular role. Because of its effects on calcium handling and membrane stability, it has been studied in contexts ranging from arrhythmia protection to vascular function and exercise performance. These properties probably explain why taurine is commonly included in energy drinks – although the physiological effects there are heavily confounded by large doses of caffeine.

Despite these interesting mechanisms, taurine occupies a somewhat ambiguous position in the context of sleep and stress regulation. Compared with compounds

such as magnesium, glycine, or melatonin, the human evidence for direct sleep-related benefits remains relatively limited.

For that reason, taurine falls more into the category of optional experimental compounds than core interventions. Some individuals report noticeable calming effects or improvements in stress tolerance, while others notice very little.

Behavioral regulators of autonomic balance

Biochemistry can support autonomic regulation, but behavioral signals remain the primary drivers of autonomic state.

The nervous system constantly integrates information from movement, temperature, light exposure, respiration, posture, and environmental stimulation. These signals influence the balance between sympathetic activation and parasympathetic recovery in ways no supplement can fully replicate. Pills can assist regulation, but the body still responds most strongly to what you do.

Morning movement and autonomic reset

Morning movement has already appeared elsewhere in the book as a circadian signal – a way of anchoring the biological clock through light exposure, physical activity, and environmental engagement. The same behavior also plays a direct role in autonomic regulation.

Physical activity is one of the most reliable modulators of autonomic tone. During exercise, sympathetic activation naturally increases: heart rate rises, breathing accelerates, and metabolic demand increases. That response is expected.

The key part comes afterward.

Following exercise, the nervous system typically shifts toward **parasympathetic recovery**. This rebound increase in parasympathetic tone can be measured in HRV studies and is one reason regular physical activity is associated with improved autonomic flexibility and stress resilience.

Even modest morning movement can function as an autonomic reset. A short walk outside, light stretching, yoga, or a brief body-weight routine can activate the sympathetic system just enough to trigger a later recovery phase. The body moves through a complete activation–recovery cycle rather than remaining stuck in the stagnant, half-activated state that often follows poor sleep, prolonged sitting, or environmental overload.

This is not about pushing intensity. What matters more than intensity is doing it regularly. Even ten or fifteen minutes of movement can shift the physiological trajectory of the day.

Cold exposure

Cold exposure is another behavioral stimulus that interacts strongly with autonomic regulation.

When the body encounters cold water, an immediate sympathetic activation response occurs. Heart rate increases, breathing becomes rapid, and alertness rises sharply. This acute response is sometimes called the **cold shock response** and represents a powerful activation of the sympathetic nervous system.

With repeated exposure, however, the nervous system often becomes more adaptable. Studies examining regular cold exposure suggest improvements in stress tolerance, mood, and autonomic regulation (Buijze et al., 2016).

In the biohacking world, this often translates into ice baths, winter swimming, or other extreme practices. In reality, most people do not have access to a frozen lake or an ice bath while traveling.

Fortunately, the stimulus does not need to be extreme.

Even cold showers can trigger many of the same physiological responses. Exposure to cold water during a shower activates thermoregulatory pathways, increases sympathetic arousal, and may improve subjective alertness and mood. Some observational and experimental studies suggest that regular cold showering can influence energy levels and perceived stress resilience, although the literature remains limited and heterogeneous (Buijze et al., 2016).

A brief cold shower in the morning can function as a strong environmental signal that shifts the nervous system toward alertness. Later in the day, however, the same stimulus may be counterproductive if the goal is relaxation and sleep initiation.

As with many autonomic interventions, timing matters.

Personally, I enjoy cold showers a lot. They're not subtle – the effect is immediate. Even a short exposure is enough to shift the system toward alertness and mental clarity. I also tend to notice a fairly immediate reduction in allergy symptoms after cold exposure. While this is only a subjective observation, it could plausibly reflect transient changes in inflammatory signaling, vascular tone, or autonomic regulation. Because of that, I use cold showers regularly, especially in the morning.

Environmental quieting

One of the most overlooked behavioral regulators of autonomic balance is also one of the simplest: reducing environmental stimulation.

The nervous system continuously scans the environment for novelty, movement, noise, and potential threats. This process – sometimes referred to as **neuroception** in polyvagal theory – operates largely outside conscious awareness. When the environment contains constant stimulation – bright lights, noise, screens, social interaction, and information streams – the nervous system tends to remain oriented toward vigilance.

Reduce that stimulation, and the opposite often begins to happen.

Lower lighting levels, less noise, slower pacing of activity, and reduced cognitive input all signal to the nervous system that the environment is safe enough to shift toward parasympathetic recovery. That is one reason dimming lights in the evening, turning off screens, or simply sitting quietly in a darker room can produce disproportionate improvements in sleep readiness.

These interventions do not force sleep, and they do not override circadian physiology. What they do is remove competing signals that keep the nervous system oriented toward alertness.

Intentionally creating periods of sensory quiet can therefore become one of the simplest ways to restore autonomic balance.

How to put it together

Sleep is not governed only by timing. It is also governed by state.

You can align light, food, and schedules perfectly and still lie in bed with a system that refuses to settle. That is where autonomic regulation becomes relevant. The issue is not the absence of sleep opportunities, but the presence of too much activation.

The aim is not to force sleep or override the system. It is to remove enough of the signals that keep it alert.

Sometimes that means breathing. Sometimes it means less light, less noise, a slower evening, or simply doing less. Sometimes this may include adaptogens, or even a supplement that has immediate effects.

But the pattern is always the same – reduce the inputs that keep the system engaged, and allow it to shift on its own.

Glucose & Feeding Control

Under stable conditions, glucose regulation is shaped not only by what you eat, but by when you eat it, in what order, after what kind of light exposure, after how much sleep, after how much sitting, and within which circadian phase your tissues believe they are operating. Travel disrupts nearly all of these variables simultaneously. Meal timing shifts, movement decreases, food novelty increases, sleep fragments, and artificial light extends the day into hours that the body would normally treat as biological night. Feeding therefore becomes metabolically less predictable even before the food itself changes. (Scheer et al., 2009; Morris et al., 2015)

The first thing to understand is that feeding is not only fuel intake. It is also a timing signal. In the chapter on temporal control, meal timing appeared as a cue for peripheral clocks. Here the focus is narrower and more practical: what travel does to postprandial glucose handling, insulin sensitivity, appetite regulation, and the subjective state many travelers know well but rarely describe in metabolic language – the sluggish, inflamed, heavy feeling that often follows irregular travel meals, poor sleep, and long hours of sitting. Circadian misalignment in humans has been shown to **impair glucose tolerance**, **reduce insulin sensitivity**, and **alter leptin** and related metabolic signals even over short periods, which means that “travel metabolism” is not imaginary. It is measurable. (Scheer et al., 2009)

Three recurring problems tend to appear together. The first is meal timing, already discussed from a circadian perspective. The second is meal structure – too much fast carbohydrate, too little fiber, too little protein, too much novelty, too many mixed signals. The third is inactivity immediately before and after meals, particularly during long travel days when skeletal muscle – the primary site of postprandial glucose disposal – is barely used. Skeletal muscle functions as the main sink for glucose after meals and during activity, and its glucose uptake is influenced both by insulin and by muscle contraction itself through **GLUT4 translocation** – a process that essentially moves glucose transporters to the cell surface, allowing muscle cells to absorb glucose more efficiently. That is why movement becomes such an important variable here (we will discuss the GLUT4 process in more detail below in the section titled “Why movement after carbohydrates works”) (Holloszy, 2005; Richter & Hargreaves, 2013).

Light remains the dominant cue for the central clock, but feeding and muscle contraction are two of the most practical levers for keeping the metabolic side of the system from drifting too far out of sync. The advantage of these levers is that they do not require perfect control. You do not need to calculate glycemic load in an airport queue or track every blue-light exposure and step count. What you need are a few robust rules that still function when the environment is messy.

Why macronutrient order matters

One of the simplest tools in this chapter is also one of the easiest to overlook because it sounds almost too basic – eat vegetables first, then protein, then fat, and carbohydrates last. The mechanism is straightforward, and it does not require a perfectly structured meal. It simply changes the postprandial trajectory of glucose and insulin.

Human trials examining meal order have shown that consuming vegetables and protein before carbohydrates can substantially blunt post-meal glucose excursions compared with eating carbohydrates first. In a randomized crossover study in people with prediabetes, Shukla and colleagues found that eating protein and vegetables before carbohydrates, or vegetables first followed by protein and carbohydrate, lowered postprandial glucose and insulin responses compared with carbohydrate-first eating. A recent review of ordered eating concluded that carbohydrate-last meal sequences tend to **reduce acute glucose and insulin excursions** with at least moderate certainty across studies. (Shukla et al., 2015; Shukla et al., 2017; Campbell et al., 2023)

This does not replace overall diet quality, calorie balance, or underlying insulin sensitivity. It simply shows that the same meal can be handled differently depending on the sequence in which it enters the system. Fiber and non-starchy vegetables slow gastric emptying, modify the rate at which carbohydrates reach the intestine, and reduce the sharpness of the glucose spike that follows. Protein early in the meal can further influence the subsequent glycemic response. During travel this kind of intervention is particularly useful because it is not all-or-nothing. Even if the meal itself is imperfect, the sequence can still be adjusted. (Shukla et al., 2015)

In practice this means that in restaurants, airport lounges, hotel buffets, or someone's kitchen in a country where you have little control over ingredients, you can often still control the order. Start with vegetables if they are available. Then eat the protein. Carbohydrates come later. Fat is less central to the sequence than the vegetable/protein-versus-carbohydrate distinction, but in real meals it often

accompanies the protein anyway. In this context, it's about reducing the size and speed of the glucose spike in an already unstable environment.

Travel weakens insulin sensitivity in boring, predictable ways

When people talk about insulin sensitivity, they often imagine chronic disease, obesity, or long-term diet. But one of the things that traveling can show us is that insulin sensitivity is also highly situational.

Sleep restriction impairs glucose handling. Circadian misalignment impairs glucose handling. Prolonged inactivity impairs glucose handling. Late meals impair glucose handling. Add novelty, stress hormones, dehydration, and poorly timed feeding, and you have a perfectly ordinary travel day that behaves metabolically like a poorly designed experiment. (Spiegel et al., 1999; Buxton et al., 2012; Scheer et al., 2009)

Sleep deprivation also alters food choice itself. Experimental studies have shown that when people are sleep deprived, they tend to select **more energy-dense, highly processed foods** and consume more simple carbohydrates and fat than when well rested. In other words, insufficient sleep does not only worsen glucose handling after a poor meal; it can make that meal more appealing in the first place. Laboratory sleep-restriction studies and neuroimaging work both show increased reward reactivity to palatable foods after sleep deprivation (St-Onge et al., 2011; Greer et al., 2013).

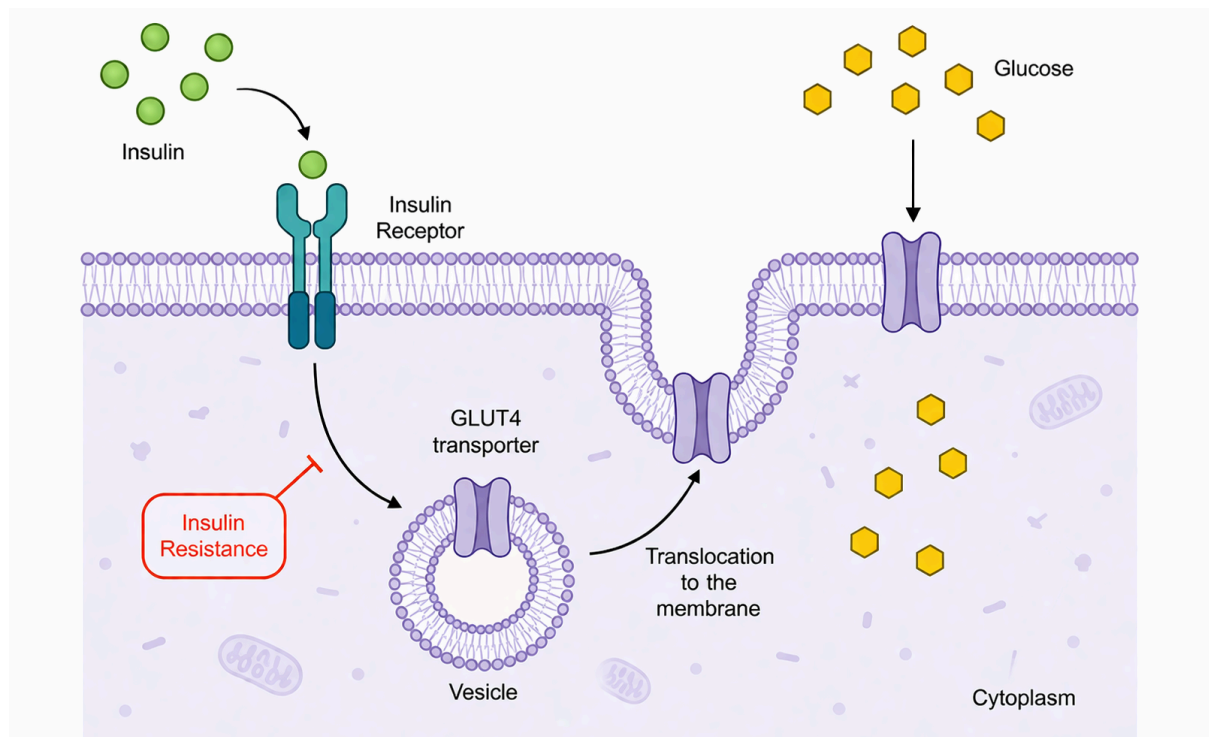
Many travel decisions therefore unintentionally push metabolism toward poorer glucose control. You sit for hours, eat quickly, and then sit again. You arrive somewhere late and exhausted and eat the heaviest meal of the day simply because it is available. Or you spend the day under-eating and later compensate with sugar, alcohol, and starch at the hour when your body is least prepared to handle it.

None of these patterns say much about discipline. They are predictable responses to disorganized inputs. Sometimes it is not a lack of control at all – it is simply the physiology of a sleep-deprived brain selecting the wrong fuel.

Why movement after carbohydrates works

Skeletal muscle is metabolically expensive tissue, and that turns out to be useful.

Contracting muscle increases glucose uptake through mechanisms that are partly independent of insulin, primarily through **GLUT4 translocation** to the muscle membrane. GLUT4 is a specialized glucose transporter protein that acts as a gateway allowing glucose to move from the bloodstream into muscle cells. Under resting conditions many of these transporters remain stored inside the cell. When muscle contracts, **GLUT4 proteins rapidly move to the cell surface, opening additional entry points for glucose to enter the muscle** and be used for energy or stored as glycogen. This allows working muscles to clear glucose from the bloodstream even when insulin signaling is not particularly strong.



Insulin signaling and muscle contraction promote translocation of GLUT4 transporters to the cell membrane, increasing glucose uptake from the bloodstream.

Because of this mechanism, exercise – even modest postprandial movement – can blunt glucose excursions after meals. The effect appears consistently across mechanistic reviews, acute trials, and meta-analyses. (Holloszy, 2005; Richter & Hargreaves, 2013; Bird & Hawley, 2017)

The practical translation is straightforward – after a carbohydrate-heavy meal, move.

Walking after meals has particularly solid evidence behind it. In adults at risk of metabolic disease, distributing short walks after meals proved more effective for lowering postprandial glucose than performing a single daily walk, with especially strong effects after the evening meal. Reviews and meta-analyses also support

post-meal exercise as beneficial for postprandial glycemia, and timing appears relevant – exercising soon after eating tends to work better for glucose control than waiting too long. Even slow walking after a carbohydrate-rich meal has been shown to reduce the incremental glucose response. (DiPietro et al., 2013; Colberg et al., 2016)

From a metabolic perspective the key principle is not that “walking wins.” It is that **muscle contraction matters**. (Richter & Hargreaves, 2013)

Resistance exercise, aerobic exercise, and mixed movement can all help.

Walking after meals sounds almost too ordinary to count as biohacking, which is exactly why it belongs here. It works in real life. It costs almost nothing. It does not require supplements, devices, or ideal conditions. It can happen after airport food, after late dinners, after hotel breakfasts, after noodles in a train station, or after one of those “I had no choice” meals that travel generates regularly.

Personally, I tend to walk after almost every meal containing carbohydrates, which is why I generally avoid eating such foods on public transport where movement is difficult. I still remember the looks from fellow passengers when I ate sweet potatoes on the train from Bratislava to Prague and then spent the next half hour walking through the compartment and doing yoga in the aisle. I may have looked like a madman, but my blood sugar certainly appreciated the effort.

Meal simplicity is underrated

Travel encourages dietary novelty. That is part of the appeal of travel itself. But novelty adds up. New cuisine, new meal timing, unfamiliar microbial exposures, irregular sleep, airport alcohol, dessert because “why not,” and a seat for nine hours – combined, these create a much larger physiological disturbance than any one variable would alone.

Modern food environments are designed primarily to maximize palatability rather than metabolic stability. Salt, sugar, fat, and strong flavor combinations activate reward circuits in the brain and increase the likelihood of overeating. This phenomenon – often described as **hyper-palatable food** – has been studied extensively. Experimental work shows that foods combining refined carbohydrates, fats, and sodium can significantly increase caloric intake and alter appetite regulation compared with simpler foods. (Fazzino et al., 2019; Hall et al., 2019)

Anthropological and epidemiological research offers an interesting contrast. Several traditional populations have historically consumed diets that would appear extreme

by modern standards – high carbohydrate intake, substantial animal food consumption, and large total caloric intake – yet have shown extremely low rates of cardiovascular disease and metabolic disorders. Studies of the Kitava population in Papua New Guinea reported very low prevalence of obesity, hypertension, and cardiovascular disease despite a diet rich in tubers, fruit, coconut fat, and fish. (Lindeberg et al., 1994; Lindeberg et al., 2003)

Similarly, large epidemiological investigations of the Tsimané population in the Bolivian Amazon documented remarkably low levels of coronary artery disease and vascular calcification despite high carbohydrate consumption and substantial caloric intake. (Kaplan et al., 2017)

These populations differ from modern industrial societies in many ways – including physical activity, pathogen exposure, sleep patterns, and food processing. However, one notable difference is the relative simplicity of their food environments. Meals typically consist of a small number of whole foods prepared with minimal processing and limited use of refined flavor enhancers such as concentrated sugars, excess salt, and industrial fats. Food provides energy and nutrients without continuously stimulating reward circuits in the way highly engineered foods often do.

This does not mean that seasoning or culinary enjoyment causes metabolic disease. Human nutrition is far too complex for such a simplistic conclusion. But comparisons like these point to a useful pattern: the sensory intensity and complexity of food environments can influence how much we eat and how stable metabolic regulation becomes.

Another overlooked dimension is **individual tolerance**. Nutrition science tends to focus on population averages, yet individuals frequently respond differently to specific foods. Paying attention to personal responses and experimenting carefully with tolerance can therefore be extremely useful. Travel actually provides a surprisingly good setting for such observation because changes in routine often make subtle physiological reactions easier to notice.

In my own case, garlic provides a good example. Under normal circumstances I usually avoid it because it tends to keep my heart rate elevated, especially if I eat it in the evening, which interferes with sleep. Interestingly, when I am traveling or when my immune system is slightly stressed, that reaction often changes and garlic becomes much more tolerable. I cannot explain the mechanism, but I have noticed the pattern repeatedly.

Observations like this highlight something important: the body's response to food is not fixed. It can shift depending on stress, immune status, sleep, circadian disruption, and many other physiological variables. Paying attention to these patterns – and occasionally using simple measurements such as heart rate, HRV, or glucose – can make personal responses much clearer than generalized advice.

Within this context, fasting windows can also play a stabilizing role. As discussed in the chapter on temporal control, meal timing functions as a biological signal that helps anchor circadian rhythms. Short periods without food – particularly during flights or long travel segments – can sometimes reduce metabolic noise by limiting the number of postprandial glucose spikes during an already chaotic day. This does not mean fasting should become a rigid rule. Travel already imposes multiple stressors, and excessive restriction can easily backfire. Used thoughtfully, occasional fasting windows can complement the broader strategy in this chapter: reducing unnecessary metabolic signals so the body can regain rhythm more easily.

Other Glucose-Stabilizing Adjuncts

Not every useful intervention in glucose regulation has to be a primary lever. Meal order, post-meal movement, light timing, sleep, and meal simplicity matter more than most supplements ever will. But secondary tools can still be useful when placed in the correct hierarchy – adjuncts rather than foundations.

Some of these compounds are relatively common and safe, while others require more caution.

Cinnamon

Cinnamon sits in a middle ground – enough evidence to be worth discussing, but enough inconsistency to prevent it from becoming a primary intervention.

Acute studies have reported **reductions in postprandial glucose** after cinnamon or cinnamon-containing preparations in some contexts, and meta-analytic data suggest possible benefits for fasting glucose and other metabolic parameters in certain populations. At the same time, the cinnamon literature is heterogeneous: species differ, doses differ, extracts differ, study populations differ, and results are not uniformly strong. (Allen et al., 2013; Akilen et al., 2012)

One reason cinnamon is difficult to interpret scientifically is that “cinnamon” is not a single intervention. Different studies use different species – most commonly *Cinnamomum cassia* or *Cinnamomum verum* – in different doses and preparations. Some trials examine whole powder, others aqueous extracts, and others

encapsulated preparations. If one study shows benefit and another does not, the discrepancy may reflect differences in the intervention itself rather than proving the entire concept wrong.

Mechanistically, cinnamon has been proposed to influence glucose metabolism through several pathways. Experimental work suggests possible effects on insulin receptor signaling, gastric emptying, carbohydrate digestion, and glucose transporter activity. Some cinnamon compounds appear to slow carbohydrate absorption or improve cellular response to insulin in vitro and in some animal models. Human data remain more modest, but the overall picture suggests cinnamon may slightly improve postprandial control in certain contexts rather than functioning as a dramatic glucose-lowering agent.

Cinnamon fits into this framework the same way it does in practice: not as a core lever, but as a low-cost optional adjunct available almost everywhere. If it becomes a superstition that excuses poor meal structure, inactivity, and bad timing, it has moved far outside its lane.

One additional practical detail: *cassia cinnamon* can contain significant amounts of **coumarin**, a compound that may become problematic in high habitual intake, particularly for the liver. Culinary use in normal amounts is not dangerous, but very large supplemental doses taken daily are not automatically a good idea.

Berberine

Berberine belongs to a different category than cinnamon. It is pharmacologically stronger and its use is correspondingly different.

Berberine is a plant alkaloid found in several herbs, including *Berberis* species, and it has been studied extensively for effects on glucose regulation, insulin sensitivity, and lipid metabolism. Clinical trials have reported reductions in fasting glucose, HbA1c – a marker reflecting average blood glucose levels over the previous two to three months – and triglycerides in people with impaired glucose regulation or type 2 diabetes. Some studies even report effects in the same general range as first-line metabolic drugs, although the quality of evidence varies across trials and formulations. (Yin et al., 2008; Lan et al., 2015; Zhang et al., 2021)

One of the most discussed mechanisms involves activation of **AMPK**, an intracellular energy-sensing pathway influencing glucose uptake, fatty acid metabolism, and hepatic glucose production. In simple terms, berberine pushes metabolism toward a more energy-efficient and insulin-sensitive state. It may also

reduce hepatic glucose output and alter gut microbiota composition, further complicating the picture.

Impressive mechanisms aside, berberine is not a casual “travel hack.” It is pharmacologically active enough to produce gastrointestinal side effects, interact with medications, and in some cases push glucose lower than expected. For healthy travelers simply trying to maintain stability, berberine may be more intervention than necessary. It makes more sense for people already dealing with clear glucose dysregulation rather than as a supplement casually thrown into a backpack because someone online called it natural metformin.

If you enjoy measuring glucose, experimenting with metabolic interventions, and generally behaving like a slightly obsessive biohacker, berberine might be an interesting compound to explore. But it is not something I would place in a standard travel toolkit.

Curcumin

Curcumin, the best-known polyphenol from turmeric (*Curcuma longa*), is usually discussed in relation to inflammation, joint health, or oxidative stress, yet it also has relevance to glucose metabolism. Experimental and clinical studies suggest that curcumin may improve insulin sensitivity, reduce inflammatory signaling associated with metabolic dysfunction, and modestly improve markers of glycemic control in certain populations. (Na et al., 2019; Panahi et al., 2017)

Mechanistically, curcumin appears to influence several regulatory pathways involved in metabolic homeostasis. Experimental work suggests it can suppress **NF-κB signaling**, a central inflammatory pathway linked to insulin resistance, while also modulating metabolic regulators such as **AMPK** and **PPAR** pathways involved in cellular energy balance and insulin signaling. In practical terms, curcumin may influence glucose regulation indirectly by improving the metabolic environment in which nutrients are processed.

This matters in travel because glucose instability is rarely just about carbohydrate intake. Inflammation, oxidative stress, sleep disruption, and circadian misalignment all push metabolism in the wrong direction, and travel tends to combine all of them. Curcumin does not directly lower glucose in the way berberine might, but it may help stabilize the broader metabolic environment in which glucose is handled.

Two practical complications are worth keeping in mind. First, curcumin has very low bioavailability in its basic form, which is why many supplements use phospholipid complexes, nanoparticles, or piperine to increase absorption. Second, the research

is highly formulation-dependent. Results obtained with specialized extracts do not translate directly to simply adding turmeric to food.

Vinegar and acetic acid

Among practical glucose adjuncts, vinegar deserves mention before many trendier compounds.

Acetic acid has been shown in several human studies to reduce postprandial glucose and insulin responses when consumed with carbohydrate-containing meals, likely through effects on gastric emptying and glucose absorption, and possibly through acute improvements in insulin sensitivity. (Johnston et al., 2004; Liljeberg & Björck, 1998)

It is simple, but it works. A simple acidic pre-load – for example vinegar in a salad or diluted before a meal – may blunt the glucose excursion from a starch-heavy meal. In many ways this belongs to the same category as meal order – modest, practical, and not dependent on expensive supplement branding.

Vinegar also has limitations. It can irritate the stomach, worsen reflux in some individuals, and is not something everyone enjoys before meals. Still, from a scientific plausibility and cost-to-benefit perspective, vinegar remains one of the more underrated glucose tools. If gastric acidity is not already high and vinegar does not irritate your stomach, adding it to a salad at an airport meal may be a surprisingly reasonable choice.

What this means in practice

Several other compounds often appear in discussions of glucose regulation – alpha-lipoic acid, chromium, bitter melon, gymnema, and others. Some of them have interesting data, particularly in metabolically unhealthy populations. But they all share the same limitation: inconsistent evidence, variable formulations, and unclear real-world impact in already functional systems.

They are not useless. They are simply not where most of the leverage is.

Glucose regulation during travel does not fall apart because someone forgot to take chromium. It falls apart because meal timing shifts, food becomes more chaotic, movement disappears, and sleep degrades.

That is where the system breaks.

Once you see that clearly, the hierarchy becomes obvious:

- 1.) *First, fix the meal itself. If the day is chaotic, simplify the meal rather than making it more “special.” If the meal is unstable, control the order: vegetables first if possible, protein next, carbohydrates later.*
- 2.) *Then look at what happens after the meal. If carbohydrates are involved, move. Walking is ideal, but any form of muscle contraction is useful. The mechanism does not care whether it looks like “exercise.”*
- 3.) *From there, timing can be adjusted. Fasting windows can sometimes reduce metabolic noise, especially during long travel segments, but they are optional. If they make the system feel worse, they are not helping.*
- 4.) *Only after these layers are in place do small adjuncts make sense. Vinegar, cinnamon, or similar interventions may slightly improve postprandial control, but they operate at the margins.*
- 5.) *More aggressive tools – such as berberine or targeted supplementation – belong to a different category entirely. They are not part of a default travel strategy.*

The more useful shift is not adding tools, but understanding why the system feels unstable in the first place.

Because it often does not feel like “glucose regulation.”

In this book, we have repeatedly talked about fatigue, brain fog, irritability without a clear cause, and hunger that does not quite fit the situation. What is often missed is that glucose dysregulation can be a major contributor to these states, and yet many people underestimate it.

Circulation & Structural Health: Blood Flow, Mobility, Vascular Function

If we want to biohack travel well, we have to consider multiple systems at once. Circadian rhythm, blood sugar, sleep, stress regulation – all of these matter. But the issue addressed in this chapter is probably the most obvious even to the laziest, most physically disconnected person imaginable: physical stagnation.

You do not need three PhDs to appreciate the consequences of not moving while traveling. Your stiff body usually explains the problem well enough. Still, it helps to look beyond soreness and vague discomfort.

Blood flow depends heavily on movement. Venous circulation in the legs does not function efficiently without muscular activity. The large veins of the lower extremities rely heavily on what is known as the **skeletal muscle pump**. When the muscles in the calves and thighs contract, they compress nearby veins and push blood upward toward the heart. Venous valves prevent backward flow, allowing this mechanical pumping system to work efficiently during walking and ordinary daily movement.

During long flights, however, this system becomes partially disabled. Sitting for extended periods reduces calf muscle activity, slows venous return, and increases blood pooling in the lower limbs. The result is **venous stasis** – one of the components of **Virchow's triad**, the three major physiological conditions associated with thrombosis formation: venous stasis, endothelial injury, and hypercoagulability.

Because of this, prolonged immobility during travel has been associated with an increased risk of **venous thromboembolism (VTE)**, including deep vein thrombosis and pulmonary embolism. Epidemiological studies examining long-distance travel have reported that flights longer than four hours are associated with roughly a two- to fourfold increase in relative risk of VTE, although the absolute risk for healthy individuals remains low (Kuipers et al., 2007; Aryal et al., 2018).

That distinction between **relative risk** and **absolute risk** matters. For most healthy travelers, the probability of developing a serious thrombotic event after a single flight remains small. The risk rises much more substantially in people with underlying conditions such as previous thrombosis, inherited thrombophilia, active cancer, major surgery in the preceding months, pregnancy, use of estrogen-containing medications, or severe obesity. Clinical guidelines therefore emphasize that long-distance travel is not equally stressful for everyone's circulatory system (Schünemann et al., 2012).

So this chapter is not about fear.

It is about mechanical reality.

The circulatory system works best when the body moves. When movement disappears for six, eight, or twelve hours, the system behaves differently. Blood flows more slowly through the veins of the legs, tissues remain under prolonged compression from seated posture, and fluid distribution shifts in ways that can produce swelling, stiffness, and fatigue.

Fortunately, many of these effects can be countered with simple interventions.

Compression Socks

We briefly mentioned compression socks earlier – in *What Travel Does to Your Body*, under the circulation section. This is one of those cases where the boring, obvious tool ends up being one of the most useful and the most practical.

Compression stockings apply external pressure to the lower leg, with the highest pressure typically at the ankle and gradually decreasing pressure toward the knee. This pressure gradient helps **reduce venous pooling and supports venous return** toward the heart. In simple terms, compression stockings assist the venous system in doing part of the work normally provided by the calf muscle pump during walking.

Clinical trials examining long-distance air travelers have consistently shown that compression stockings reduce the incidence of asymptomatic deep vein thrombosis and significantly decrease leg swelling and discomfort during prolonged flights (Clarke et al., 2006; Scurr et al., 2001).

In one randomized trial involving long-haul travelers, passengers wearing compression stockings during flights longer than eight hours showed dramatically lower rates of symptomless **deep vein thrombosis** detected by ultrasound compared with control groups who did not wear stockings (Scurr et al., 2001).

Even when thrombosis risk is not the main concern, many travelers report subjective improvements in comfort, leg fatigue, and swelling when using compression socks during long flights. The physiological explanation is straightforward – improved venous return reduces fluid accumulation in the lower extremities and supports more stable circulation during prolonged sitting.

Personally, compression socks are one of the few travel tools I almost never skip. They are not glamorous. They are also pretty weird and not exactly comfortable to wear. But the important part is simple – they work and they are reliable. And it is not just my legs that feel better after a flight with them than after a flight without them.

For individuals with known circulatory disorders, previous thrombosis, or clotting disorders, properly fitted compression stockings may be particularly valuable. In those cases, however, travel strategies should always be discussed with a physician.

Movement During Flights

Yes, compression socks are great and they really do the job well. They help to reduce venous pooling and support blood return toward the heart. But they are only part of the system. They assist circulation – they do not replace movement.

Sure, we all know that the movement is beneficial. Earlier in the book we discussed movement primarily in relation to circadian rhythm and glucose regulation and now we know that physical activity in the morning helps anchor circadian timing, and movement after meals helps skeletal muscle absorb glucose through insulin-independent mechanisms such as GLUT4 translocation.

In this chapter, however, movement serves a different function. And as is often the case, this is another great reason to get moving.

When passengers remain seated for hours, venous blood flow in the legs slows significantly. Doppler ultrasound studies show that calf muscle contractions can **increase venous blood flow severalfold** compared with passive sitting (Delis et al., 2001). That is why immobility is one of the major contributors to travel-related venous stasis.

The physiology is simple. The calf muscles – especially the **gastrocnemius and soleus** – act as a kind of peripheral heart. When they contract, they compress deep veins in the lower leg and push blood upward toward the heart. Venous valves prevent backward flow, allowing each contraction to move blood progressively upward.

Without those contractions, venous circulation slows and fluid begins to pool in the lower limbs.

Fortunately, it does not take much movement to interrupt this process. Even small contractions help maintain circulation.

Simple movements that can be performed directly in the seat include:

- ankle pumps (alternating flexion and extension of the foot)
- calf contractions
- knee lifts
- seated leg extensions
- alternating heel raises

These movements activate the calf muscles and restore the venous pumping mechanism that sitting temporarily suppresses. Airlines often recommend similar exercises in safety materials for exactly this reason.

Whenever possible, however, standing and walking through the cabin is even better.

Personally, when the seatbelt sign is off, I often walk to the back of the plane near the lavatories and perform a few simple stretches or light yoga movements. This is

rarely elegant. Flight attendants sometimes need to pass through the aisle, and I occasionally have to move aside to avoid blocking traffic. But the difference in how the body feels afterward is very noticeable.

Mobility and Postural Stress During Flights

Maintaining circulation is one part of the problem.

The other is mechanical.

Prolonged sitting places the body into a very specific mechanical configuration. The hips remain flexed, the lumbar spine loses its natural curvature, the thoracic spine gradually rounds forward, and the shoulders drift into internal rotation. Several muscle groups remain shortened for hours while others remain largely inactive.

Over time this creates stiffness, mild tissue compression, and the familiar folded-human feeling that many travelers recognize after long flights.

Certain muscle groups tend to suffer more than others.

Hip flexors – especially the iliopsoas and rectus femoris – remain in a shortened position during sitting. Over time they stiffen and can contribute to lower-back discomfort once you stand up.

Gluteal muscles remain relatively inactive while the hip flexors stay shortened. This prolonged inactivity reduces muscular support for the pelvis and contributes to stiffness in the hips.

Hamstrings are not fully shortened, but they remain under continuous passive tension when sitting for long periods, especially when the knees stay bent.

Thoracic spine and chest muscles are also affected. Airplane seating encourages a slightly forward-flexed posture. Over time this can tighten the chest muscles – particularly pectoralis minor and major – while reducing mobility in the thoracic spine.

Lumbar stabilizers are compressed and fatigued by prolonged sitting, which helps explain why the lower back can feel dull, tight, and mechanically unhappy after a flight.

Standing periodically and performing a few simple stretches can counteract many of these effects.

Useful movements include:

- **Hip flexor stretches** – stepping one foot behind the other and gently extending the hip helps reverse prolonged hip flexion.
- **Spinal extension movements** – gently arching the back restores mobility to the lumbar and thoracic spine.
- **Torso rotations** – slow rotations help mobilize the thoracic spine after prolonged sitting.
- **Hamstring stretches** – placing one heel slightly forward and hinging gently at the hips relieves posterior leg tension.

These movements do not need to be elaborate. This book is not a physiotherapy manual or a yoga guide, and there are countless mobility routines elsewhere for people who want to go deeper into corrective exercises after travel. Many of them are excellent for restoring muscular balance – strengthening muscles that become underactive during long flights and loosening those that remain shortened for hours.

The goal here is much simpler.

These are small, practical interventions you can perform in the narrow aisle of an airplane without turning the whole thing into a performance. A few minutes of stretching, gentle spinal extension, or hip-opening movements can already interrupt the mechanical stiffness created by prolonged sitting.

Changing posture redistributes pressure across tissues, improves circulation in compressed areas, and restores some degree of joint mobility after immobility.

The body is simply not designed for prolonged stillness. Even brief movement helps in many ways.

Massage Tools and Postural Support

At first glance this topic may seem almost too simple to deserve its own section. It is not a complex protocol, and the solution can be as basic as a towel. But in practice it can make a surprisingly large difference. For me, as someone who is nearly two meters tall, maintaining a tolerable posture during long flights is a constant challenge – and small mechanical adjustments to spinal support can easily become one of the most useful “hacks” during travel.

One tool I often use during long flights is a **small physiotherapy massage ball**. Some designs consist of two connected spheres that fit comfortably along the spine. When placed between the lower back and the seat, this device provides gentle pressure and support to the lumbar region and functions almost like a portable myofascial release tool.

There are also small devices designed specifically for this purpose – compact lumbar supports that travelers sometimes carry for long flights or train rides. But even simpler solutions can work surprisingly well. A rolled towel, rolled sweatshirt, the wrinkled blanket that airlines hand out on long flights or small pillow placed behind the lumbar spine can improve posture and reduce mechanical strain on the back.

These small adjustments help maintain a more neutral spinal position during prolonged sitting. That alone can noticeably improve comfort, but it can also influence circulation, muscle tension, and the amount of fatigue that accumulates in the lower back during long periods of immobility. Why not give it a try?

Resistance Bands and In-Seat Muscle Activation

Resistance bands are a compact way to add muscle activation during long periods of sitting.

For example:

- pressing the knees outward against the band
- pulling the band with the hands for upper-body activation
- performing small rowing movements

As discussed elsewhere in the book, skeletal muscle contraction has systemic effects – improving circulation, enhancing glucose uptake, and maintaining neuromuscular activation.

You can even take a resistance band into the airplane aisle and do a little more with it when space allows. Yes, you'll look weird.

Your circulation will not care.

Hydration and Electrolytes

Hydration is another frequently overlooked component of circulatory health during flights. Airplane cabins typically maintain humidity levels around **10–20 percent**, far lower than the humidity of most terrestrial environments. This low humidity increases insensible water loss through respiration and skin evaporation (Ergan et al., 2018; Zubac et al., 2020).

Mild dehydration can increase blood viscosity and alter circulatory dynamics, although the extent to which this directly contributes to travel-related thrombosis

risk remains debated (Schobersberger et al., 2007). Even when clotting risk is not the main concern, reduced plasma volume can still worsen fatigue, swelling, dry airways, and the general sense that the body is functioning in a slightly more compressed and less efficient way during flight (Zubac et al., 2020).

Maintaining hydration during flights is therefore a simple but useful precaution. But water alone is not the whole story. Fluid balance depends not only on how much you drink, but also on the electrolyte environment in which that fluid is retained and distributed.

Sodium is the **main extracellular electrolyte** and plays a central role in maintaining plasma volume and fluid distribution. Too little sodium relative to fluid intake can impair effective hydration, while excessive sodium in heavily processed travel food may increase thirst and contribute to fluid retention in ways that do not feel particularly good during long sitting.

Potassium, by contrast, is the **main intracellular electrolyte** and is essential for membrane excitability, neuromuscular function, and normal cardiac conduction.

Magnesium – already discussed elsewhere in the book – influences neuromuscular stability, vascular tone, and cellular energy metabolism. It also functions as a natural calcium antagonist in smooth muscle, which is one reason it matters for **vascular regulation** as well as stress physiology (Ahmed et al., 2019; Pickering et al., 2020).

Electrolytes are usually associated with heat, sweating, and physical exertion. That is where most people think about them.

But the airplane environment may be just as relevant, because it undoubtedly creates conditions in which hydration becomes less efficient even without visible sweating.

If you already use electrolytes during physically demanding days, it is worth remembering that long flights place the body under a different kind of stress – one that can benefit from the same support.

In practice, this does not require anything complicated. Drinking water regularly, occasionally including mineral-containing fluids, and avoiding a day built entirely on coffee, alcohol, and low-mineral food is often enough to keep the system more stable.

Blood-Thinning Agents and Natural Compounds

Some travelers with known clotting risk factors use pharmacological agents such as low-dose aspirin or prescription anticoagulants under medical supervision. Aspirin works by inhibiting platelet aggregation through irreversible acetylation of the cyclooxygenase-1 (COX-1) enzyme, which reduces the production of thromboxane A₂ and limits platelet activation.

However, aspirin mainly affects **arterial clot formation**, while travel-related problems are more often associated with **venous thrombosis**. For this reason its role in preventing travel-related venous thromboembolism remains debated, and current clinical guidelines generally do not recommend aspirin as routine prophylaxis for otherwise healthy travelers (Schünemann et al., 2012).

For most young and healthy people, the strategies discussed earlier in this chapter – movement, calf muscle activation, compression socks, and hydration – are usually far more relevant than pharmacological blood-thinning strategies.

That said, some travelers like to experiment with substances that may have **mild antithrombotic effects**. One interesting example is the mushroom *Auricularia auricula-judae*, sometimes called “wood ear mushroom.” Experimental studies suggest that extracts from this mushroom can inhibit platelet aggregation and may influence blood viscosity to a small degree (Wasser, 2014). Personally, I occasionally use *Auricularia* preparations because they appear relatively gentle compared with pharmaceutical anticoagulants.

Still, it is important to keep perspective. These effects are subtle, and most people do not need to actively manipulate blood coagulation during travel. Anyone with a personal history of thrombosis, clotting disorders, cardiovascular disease, or those already taking anticoagulant medication should only consider additional blood-thinning strategies after discussing them with a physician. Even seemingly mild compounds can interact with medications or influence platelet function in unpredictable ways.

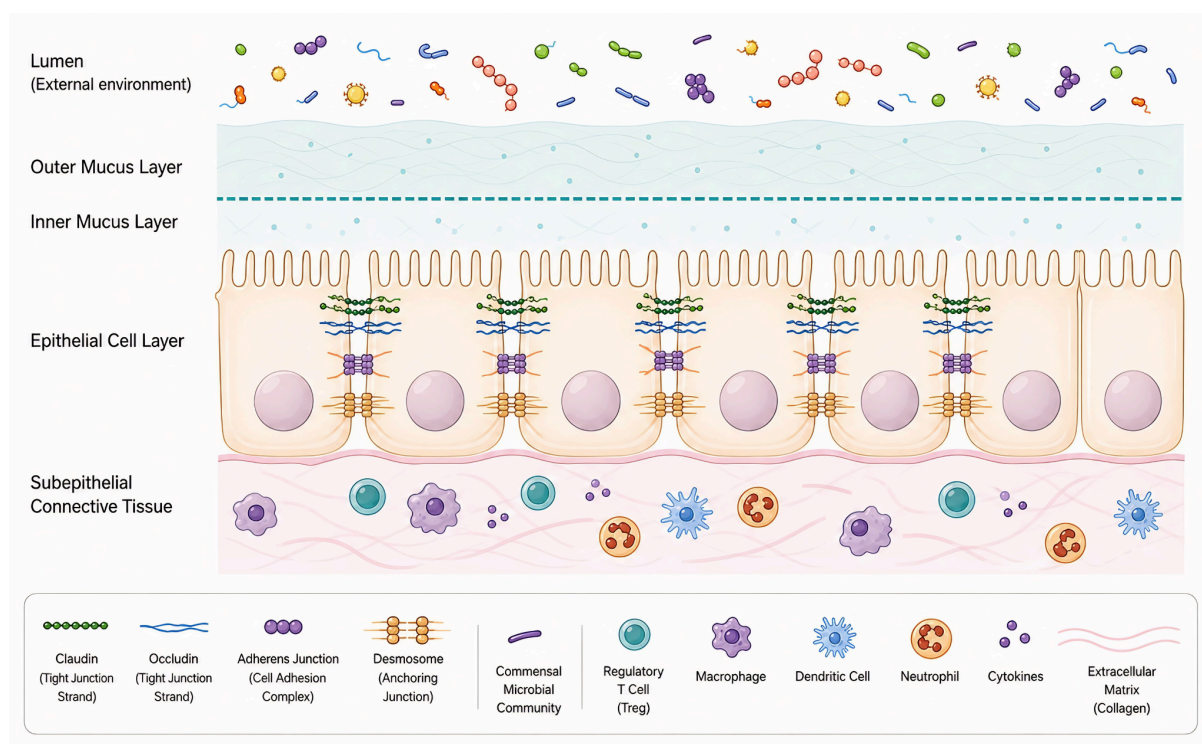
For the majority of travelers, **movement and circulation remain the real intervention.**

Gut & Barrier Integrity: Microbial and Immune Interface

Travel medicine often focuses on vaccines, sanitation, and obvious pathogens. But the first system that experiences travel stress is not the immune system in isolation. It is the intestinal ecosystem. The gut is where unfamiliar food, unfamiliar microbes, unfamiliar water chemistry, and unfamiliar behavioral rhythms collide. For a traveler, this interface becomes a testing ground for physiological stability.

The intestine is not simply a digestive tube. It is a highly regulated barrier composed of epithelial cells joined together by specialized protein structures known as **tight junctions**. These junctions act as molecular gatekeepers. Nutrients, electrolytes, and water are absorbed selectively, while bacteria, toxins, and partially digested macromolecules are largely prevented from crossing into circulation.

From an evolutionary perspective, the gut barrier maintains a delicate balance. The intestinal lumen contains trillions of microorganisms belonging to hundreds of species. Yet the immune system does not normally treat this microbial population as an invasion. Instead, immune tolerance and microbial competition maintain a dynamic equilibrium. The gut lining communicates continuously with the immune system through cytokines, antimicrobial peptides, and secretory IgA antibodies that regulate microbial behavior at the mucosal surface (Belkaid & Hand, 2014).



Simplified structure of the intestinal barrier and its key regulatory layers. Epithelial cells are connected by tight junction proteins such as claudin and occludin, which regulate what can pass between cells. Above them, mucus layers create separation from the microbiome, while immune cells below the epithelium monitor and respond to signals crossing the barrier.

As we have already discussed throughout this book, travel disrupts several variables that normally stabilize physiology. Sleep timing shifts, circadian signals change, hydration fluctuates, and dietary patterns often become irregular. At the same time, the gut is exposed to unfamiliar microbes through food, water, and the surrounding environment.

These changes do not immediately produce disease. In many cases the first effect is **a temporary destabilization of the gut microbiome itself.**

Microbial communities respond rapidly to environmental change. Even dietary shifts alone can alter microbial composition within 24–48 hours. In controlled feeding experiments, switching from plant-based diets to animal-based diets changed microbial populations in less than two days (David et al., 2014). When travel adds new microbial exposure, circadian disruption, and behavioral changes on top of this, the disturbance can become much larger.

During this period the microbiome may temporarily lose some of its **colonization resistance** – the ability of resident microbes to prevent invading organisms from establishing themselves.

This is the point where pathogens begin to matter.

One visible manifestation of this ecological disturbance is traveler's diarrhea, one of the most common medical problems associated with international travel.

Why Traveler's Diarrhea Happens

Depending on the destination, between **30% and 70% of international travelers** experience some form of gastrointestinal illness during their trip (Steffen et al., 2015). The risk is particularly high in regions where sanitation systems, water treatment, and food handling practices differ significantly from those in the traveler's home country.

However, traveler's diarrhea is not caused by a single organism. A variety of microbes can be responsible, including enterotoxigenic *Escherichia coli* (ETEC), *Campylobacter jejuni*, *Shigella*, and *Salmonella*. Viral pathogens such as norovirus also contribute in certain environments (Connor, 2015).

These organisms produce symptoms through several mechanisms. Some release toxins that stimulate intestinal secretion of water and electrolytes, producing watery diarrhea. Others invade intestinal epithelial cells directly, triggering inflammation and disrupting barrier function. In both cases the intestine responds by accelerating motility and increasing fluid secretion – the body’s way of flushing out pathogens.

Yet exposure alone does not explain why some travelers get sick while others do not, even when they eat the same food.

The missing piece is the **state of the gut ecosystem at the moment of exposure**.

A stable microbiome acts as a powerful defensive barrier. Resident microbes compete with pathogens for nutrients, produce antimicrobial compounds, and occupy ecological niches that invading organisms might otherwise colonize. When microbial diversity is high and the ecosystem is stable, pathogens often fail to gain a foothold.

When the microbiome is already undergoing ecological reorganization – as frequently happens during travel – this defensive barrier can temporarily weaken. In that window of instability, invading organisms have a greater chance of establishing themselves.

One additional factor that can amplify this instability is what we earlier described when we recommended choosing simple foods over exotic ones. Travel often introduces several dietary changes simultaneously – and along with them come various microbial exposures through food preparation.

For this reason, a simple practical strategy is to introduce new foods gradually rather than stacking multiple unfamiliar meals in a single day while the body is already adapting to jet lag, stress, and environmental change.

Sometimes, however, control is limited. When the inputs are already chaotic, other strategies become useful.

One of the most studied tools in this context is the probiotic yeast ***Saccharomyces boulardii***.

Saccharomyces boulardii

Many probiotics marketed to travelers contain bacterial strains such as *Lactobacillus* or *Bifidobacterium*. These organisms can be useful in certain contexts, but they have limitations. Many bacterial probiotics are sensitive to stomach acid, heat, and antibiotic exposure.

Saccharomyces boulardii is different.

It is not a bacterium but a probiotic yeast. This distinction has important consequences. Because it is a yeast, *S. boulardii* is resistant to antibiotics that target bacteria. It also survives gastric acidity relatively well, allowing it to reach the intestine intact.

Research into *S. boulardii* began in the early twentieth century when French microbiologist Henri Boulard isolated the yeast from lychee fruit while studying treatments for cholera-associated diarrhea.

Modern research has identified several mechanisms through which this organism may protect the intestinal environment.

First, *S. boulardii* can interfere with pathogen adhesion. Certain pathogens attach to intestinal epithelial cells using specific receptor interactions. Experimental studies show that *S. boulardii* can inhibit these interactions and **reduce pathogen colonization** (McFarland, 2010).

Second, the yeast appears to influence toxin activity. In laboratory models, it can **neutralize toxins produced by pathogenic *E. coli* strains**, reducing the secretory response that drives diarrhea.

Third, *S. boulardii* influences immune activity at the mucosal surface. Studies show increased secretion of **IgA antibodies** and modulation of inflammatory cytokines in its presence (Kelesidis & Pothoulakis, 2012).

These mechanisms translate into measurable outcomes in clinical studies.

One randomized study examined European travelers visiting high-risk destinations. Participants who took *S. boulardii* before and during travel experienced significantly lower rates of traveler's diarrhea compared with placebo groups (Kollaritsch et al., 1993).

Subsequent meta-analyses report similar protective effects, particularly when supplementation begins before travel rather than after symptoms appear (McFarland, 2010).

The logic is straightforward. Establishing the organism before exposure to unfamiliar pathogens may provide both competitive and immunological advantages.

For this reason, I personally take *S. boulardii* several days before departure and continue during the trip.

From a practical standpoint, it also has several advantages. It is resistant to many antibiotics, relatively stable at room temperature, and appears to have a strong safety profile in healthy individuals.

For these reasons, it has become one of the most widely recommended microbiome-supporting supplements in travel medicine. I definitely recommend it.

Fiber and Microbiome Stability

If probiotic organisms such as *Saccharomyces boulardii* can help defend the intestinal ecosystem during travel, the next question naturally follows – **what determines whether that ecosystem remains stable once the trip begins?**

The answer is not a single supplement. It is the **metabolic environment of the microbiome**. Gut microbes do not survive on enthusiasm, probiotics, or good intentions. They survive on substrates. When those substrates change suddenly, microbial populations reorganize.

One of the most important substrates for a healthy gut ecosystem is **dietary fiber**.

Human digestive enzymes cannot break down many plant fibers. Instead, these compounds reach the colon largely intact, where they are fermented by gut microbes. This fermentation process produces a group of molecules known as **short-chain fatty acids (SCFAs)**, primarily acetate, propionate, and butyrate.

However, these molecules are not metabolic waste products. They are biologically active signaling compounds with major effects on intestinal physiology.

Butyrate in particular plays a central role in **gut barrier integrity**. Colonocytes – the epithelial cells lining the colon – use butyrate as a primary energy source. When butyrate availability is high, epithelial cells maintain stronger tight junctions and healthier turnover rates. When butyrate production falls, epithelial metabolism becomes less efficient and barrier integrity can weaken (Koh et al., 2016).

Butyrate also influences inflammation. It interacts with regulatory pathways such as histone deacetylase inhibition and G-protein coupled receptor signaling, which modulate immune responses and reduce inflammatory cytokine activity (Makki et al., 2018).

In simple terms, butyrate helps maintain a **stable intestinal barrier and a calmer immune environment**.

Many people around the world are fiber-deficient for this reason, but it's an especially big problem when traveling, when even people with otherwise exemplary diets struggle to get enough fiber.

Airport food, convenience snacks, and restaurant meals often contain far less fiber than a typical home diet. Moreover, some countries are simply not progressive enough when it comes to fiber. For this reason, I personally struggle quite a bit in India, for example, where green salad seems to be almost an illegal food and is nearly impossible to find in supermarkets, let alone in restaurant meals.

From the perspective of the microbiome, it is a **sudden shift in ecological resources**.

When fiber intake drops sharply, microbial species that depend on complex carbohydrates lose their primary energy source. Other organisms capable of metabolizing proteins or simple sugars may temporarily expand in their place. These shifts can occur surprisingly quickly.

Controlled dietary experiments have demonstrated that microbial composition can change measurably within **48 hours** of major dietary alterations (David et al., 2014).

This rapid responsiveness is one reason travelers sometimes experience digestive instability within the first days of a trip even when no obvious pathogen is involved.

Maintaining some degree of **fiber continuity** during travel can therefore act as a stabilizing factor for the microbiome. Sometimes, though, you just have to improvise. While I can't find green lettuce in India, I can find chia seeds almost everywhere there. It's not exactly a culinary delight, but by adding a few to my water every day, I can at least boost my fiber intake a little.

This does not require obsessive dietary control. Inhale, exhale and relax. It simply means that when possible, including foods such as vegetables, legumes, fruit, or whole grains can provide fermentable substrates that support microbial metabolism and short-chain fatty acid production.

A Small Note on Coffee, Fiber, and Microbial Metabolism

There is also an interesting observation related to fiber that does not always receive much attention.

Coffee contains small amounts of soluble fiber and a large number of polyphenolic compounds that interact with the gut microbiome. These compounds pass through

the upper digestive tract largely intact and reach the colon, where they become substrates for microbial metabolism.

Several microbial species appear to respond strongly to regular coffee consumption and one organism that has attracted particular attention in microbiome research is *Lawsonibacter asaccharolyticus*. This bacterium appears to be strongly associated with habitual coffee intake and seems particularly adapted to environments where coffee-derived polyphenols are present (Vieira-Silva et al., 2020).

Unlike many gut microbes that rely primarily on carbohydrates as substrates, *Lawsonibacter asaccharolyticus* participates in metabolic pathways linked to **polyphenol transformation and fermentation networks within the gut ecosystem**. In other words, compounds that originate in coffee may serve as ecological fuel for this organism.

What makes this particularly interesting is the downstream metabolic effect.

The microbial fermentation networks in which *Lawsonibacter* participates contribute to the production of short-chain fatty acids, especially **butyrate**.

If you remember the previous section, you already know what that means for the gut barrier and immune environment.

In addition, several large microbiome studies have reported that regular coffee drinkers show distinct microbial signatures associated with increased abundance of organisms involved in fermentation and polyphenol metabolism (Mills et al., 2019). While coffee is obviously not the only dietary factor influencing these microbial pathways, it appears to be one of the most consistent nutritional variables associated with the presence of *Lawsonibacter*.

In that sense coffee is not merely a delicious beverage. It may also function as a subtle **microbiome modulator**, providing substrates that shape microbial metabolism in the colon.

And this is also one of the reasons I enjoy drinking coffee regularly – even when I drink only decaffeinated coffee.

Over time I noticed something interesting. When I drink coffee regularly, my digestive tolerance to certain foods appears to improve. I cannot claim a precise mechanistic explanation for this observation. It may involve microbial shifts, polyphenol metabolism, intestinal motility, or several of these mechanisms interacting together.

Observations like this highlight an important point about physiology. Scientific studies provide population-level insights, but individual responses can vary significantly. Paying attention to how your own body reacts to variables such as food timing, coffee intake, fiber sources, or sleep patterns can reveal patterns that research has not yet fully mapped.

Cultivating this kind of careful curiosity – observing your responses and adjusting accordingly – is one of the core attitudes I hope this book helps you develop. Travel constantly introduces new variables, and the ability to read those signals becomes one of the most valuable biohacking skills you can build.

Zinc and Intestinal Barrier Repair

One micronutrient that becomes particularly relevant in this context is zinc.

Zinc participates in numerous biological processes, including immune signaling, enzyme activity, antioxidant defense, and cellular repair mechanisms. Within the intestine, zinc appears to play a particularly important role in **maintaining epithelial barrier function**.

We know that the intestinal barrier depends on tight junctions that regulate permeability. Experimental studies show that zinc influences the structure and regulation of several tight junction proteins. When zinc availability is insufficient, barrier integrity can weaken, allowing increased passage of bacterial products and inflammatory molecules (Sturniolo et al., 2001).

This mechanism becomes especially relevant during gastrointestinal illness.

Clinical research in children with diarrheal disease has demonstrated that zinc supplementation can **shorten the duration and severity of diarrhea episodes**. These findings led the World Health Organization to recommend zinc supplementation as part of the treatment protocol for pediatric diarrheal illness in many regions (Fischer Walker & Black, 2010).

The mechanism likely involves several overlapping processes.

Zinc supports epithelial regeneration, helping intestinal cells recover after inflammatory damage. It also influences immune signaling pathways and antimicrobial defenses within the gut environment. In other words, zinc does not simply act as a nutrient; it participates directly in the repair and stabilization of the intestinal barrier.

In some aspects, discussions about zinc can become a bit more technical – and this is where biohackers sometimes like to go one step deeper.

Some discussions about zinc focus not only on how much zinc you consume, but also on **how effectively zinc enters cells**, where many of its biological effects actually occur. Certain compounds known as **ionophores** can help shuttle zinc ions across cellular membranes, increasing intracellular zinc concentrations.

This idea has attracted attention both in research and among more detail-oriented biohackers.

One compound that has received attention in this context is **quercetin**, a flavonoid found in many fruits and vegetables. Experimental studies suggest that quercetin can act as a zinc ionophore under certain conditions, facilitating the movement of zinc into cells (Dabbagh-Bazarbachi et al., 2014).

Another molecule that appears to have similar – and in some experimental systems even stronger – ionophore activity is **epigallocatechin gallate (EGCG)**, a polyphenol abundant in green tea and particularly concentrated in fucoxanthin. Laboratory studies indicate that EGCG may enhance zinc transport across cellular membranes and influence intracellular zinc signaling pathways.

In practice this leads to a slightly nerdy but interesting biohacker trick: some people combine zinc supplementation with polyphenol-rich compounds such as quercetin or green tea in the hope of increasing intracellular zinc availability.

Is this necessary for normal physiology? Probably not. The human body already possesses specialized zinc transport systems – including the ZIP and ZnT transporter families – that regulate how zinc enters and exits cells.

But the topic illustrates an interesting principle: micronutrients rarely act in isolation. Their biological effects are often shaped by interactions with other compounds present in the diet.

And if someone chooses to take zinc with green tea and enjoy the feeling of doing something slightly more sophisticated, that is not the worst habit to develop.

From a practical perspective, zinc becomes most relevant when the system is already under stress – during gastrointestinal illness, dietary disruption, or exposure to unfamiliar microbes. In those situations, even small support for epithelial repair can make a noticeable difference.

When I travel, I'm always happy to find any supplement that contains at least a little zinc :)

Activated Charcoal

Activated charcoal is one of those remedies that almost everyone seems to recommend for digestive problems. If you mention diarrhea, food poisoning, or sudden gastrointestinal distress while traveling, there is a good chance someone – sometimes even someone's grandmother – will suggest taking activated charcoal.

And to be fair, there is a reason it has earned that reputation.

Activated charcoal is produced by heating carbon-rich materials to extremely high temperatures, creating an extremely porous structure with an enormous surface area. This structure allows charcoal to **adsorb** a wide range of molecules, meaning compounds can bind to its surface and remain in the gastrointestinal tract rather than being absorbed into the bloodstream.

Because of this property, activated charcoal has long been used in clinical medicine to treat certain types of poisoning and drug overdose. When administered early enough, it can bind toxins in the gastrointestinal tract and reduce their systemic absorption (Vale et al., 1985; Chyka et al., 2005).

In the context of travel, charcoal is often used as a short-term tool for **gastrointestinal discomfort, gas, or mild food-related illness**. The proposed mechanism is relatively simple: charcoal may bind bacterial toxins, fermentation byproducts, or other irritating molecules in the gut lumen, allowing them to pass through the digestive system without being absorbed.

Personally, it is something I keep available and occasionally use when gastrointestinal symptoms appear during travel.

However, it is important to understand what activated charcoal **does and does not do**.

Charcoal does **not kill pathogens**, and it does not repair the intestinal barrier or restore microbial balance. Its effects are largely mechanical – binding certain molecules within the digestive tract rather than altering the underlying biological processes responsible for the illness.

For that reason, the scientific evidence supporting routine use for gastrointestinal infections is limited. Some small clinical studies suggest that activated charcoal may reduce symptoms such as gas or toxin-related irritation, but it is generally

considered a **supportive measure rather than a curative treatment** (Holtmann et al., 2003).

There is also an important practical caveat.

Activated charcoal binds molecules **non-selectively**, which means it can also bind medications and reduce their absorption. For this reason, I personally prefer not to take it together with minerals, vitamins or other supplements.

Alcohol and Barrier Permeability

Most of the strategies discussed so far focus on supporting the microbiome and stabilizing the intestinal environment during travel. There is also a factor that pushes strongly in the opposite direction.

Alcohol.

By the time we reach this point in the discussion, we already know that the intestinal barrier is a regulated interface that normally prevents bacterial fragments and toxins from entering circulation. When that regulation is disturbed, permeability can increase – a phenomenon well documented in physiology, sometimes referred to in research literature as **increased intestinal permeability** (Bischoff et al., 2014).

And alcohol is undoubtedly one of the substances known to influence this process.

Ethanol and its metabolic byproduct **acetaldehyde** can disrupt the proteins that maintain epithelial barrier integrity. Experimental studies show that alcohol exposure alters tight junction proteins such as **occludin and claudins** (as illustrated earlier in the tight junction diagram in the section "Gut & Barrier Integrity: Microbial and Immune Interface"), increasing intestinal permeability and allowing bacterial endotoxins to cross the intestinal lining more easily (Bischoff et al., 2014).

This does not occur in isolation.

Alcohol also influences the microbiome itself. Both animal and human studies demonstrate that alcohol consumption can shift microbial composition and increase the translocation of **bacterial endotoxins such as lipopolysaccharides** into circulation (Mutlu et al., 2012).

During travel this becomes particularly relevant because several destabilizing variables are already present at the same time – disrupted sleep, circadian misalignment, dehydration, unfamiliar foods, and exposure to new microbial environments.

Adding alcohol into that mixture can amplify the disturbance.

Alcohol also has broader physiological effects that extend beyond the gut. Its metabolism increases oxidative stress, interferes with mitochondrial function, disrupts sleep architecture, impairs glucose regulation, and places additional burden on the liver through acetaldehyde detoxification.

Taken together, it is not just a gut disruptor – it is a systemic stressor.

For that reason, I personally avoid alcohol entirely. From a physiological perspective it is difficult to find a compelling argument for it – especially when the body is already adapting to environmental stress during travel. That does not mean every reader will suddenly become abstinent, but it is worth understanding what alcohol is doing to the system. Even if you skipped just one drink because of this chapter, I consider that a success.

Stress Physiology and the Gut Barrier

Another important variable influencing gut barrier stability is **stress physiology**.

The gastrointestinal system is closely connected to the central nervous system through neural, endocrine, and immune pathways collectively described as the **gut–brain axis**. Signals originating in the brain influence intestinal motility, epithelial secretion, immune activity, and microbial behavior.

Activation of the **sympathetic nervous system** – whether triggered by psychological stress, sleep disruption, or environmental uncertainty – produces several physiological shifts. Cortisol levels rise, gastrointestinal motility may change, and blood flow can be redistributed away from the digestive tract. These responses are adaptive in the short term, but persistent activation can influence barrier regulation and microbial dynamics.

Experimental research shows that psychological stress can increase **intestinal permeability** and alter microbial composition through neuroendocrine signaling pathways (Konturek et al., 2011).

Situations involving environmental novelty and logistical demands often produce mild but sustained sympathetic activation. This state does not necessarily manifest as overt anxiety. A person may feel curious or relaxed while the autonomic nervous system remains slightly vigilant.

From a gastrointestinal perspective, however, that vigilance still matters.

Subtle shifts in autonomic balance can influence intestinal motility, immune signaling, and epithelial barrier function. As a result, digestion may become temporarily more sensitive even when food quality remains unchanged.

In many cases the explanation is therefore not a specific meal but the interaction between **autonomic stress signaling and intestinal physiology**.

At this point, the practical question is obvious – what should be done about stress in this context?

The answer is not new. In earlier sections of this book, we already discussed multiple ways of influencing autonomic balance – from breathing patterns and light exposure to movement, supplementation, and sleep regulation.

This chapter does not introduce new tools. Its purpose is to show why those tools matter for the gut as well.

So, please, don't underestimate stress. Stress isn't just about having trouble falling asleep or having a lower HRV.

As one of my business professors at university said: "Everything is connected with everything else!"

So don't stress out, because that affects all your systems at once.

Hydration Revisited: Fluid Balance and Digestive Stability

Earlier we discussed hydration in relation to the mucosal barrier and environmental dryness during flights. However, hydration also plays an important role in digestive stability.

Fluid balance is a fundamental component of gastrointestinal physiology. The intestinal epithelium regulates the movement of water and electrolytes through tightly controlled transport systems. Under normal conditions, the small intestine absorbs several liters of fluid each day while maintaining osmotic balance between the intestinal lumen and the bloodstream. This process depends on coordinated transport of sodium, chloride, and glucose across epithelial membranes.

When intestinal inflammation or infection occurs, this regulation can become disrupted. Many pathogens associated with gastrointestinal illness produce toxins that stimulate chloride secretion into the intestinal lumen. Water follows these

electrolytes osmotically, producing the rapid fluid loss characteristic of diarrheal illness (Field, 2003).

From a physiological perspective, the most important intervention during acute gastrointestinal disturbance is therefore not antimicrobial therapy but **fluid replacement**.

Oral rehydration solutions work because they exploit one of the most robust transport mechanisms in intestinal biology: the **sodium–glucose co-transport system (SGLT1)**. Even when intestinal secretion is elevated, this transporter continues to absorb sodium and glucose together, allowing water to follow osmotically and restoring fluid balance (Binder et al., 2014).

For this reason oral rehydration therapy remains one of the most effective and widely used treatments for diarrheal illness worldwide.

Travel creates a situation where dehydration is easy to miss: reduced intake, altered meals, low cabin humidity, and fluid loss during illness.

In that situation, the priority is replacement.

Water alone is often not enough. When fluid loss becomes significant, electrolyte-containing fluids or oral rehydration solutions become far more effective because they actively support intestinal absorption rather than relying on passive intake.

Integrating the Pieces: A Gut Stability Strategy for Travelers

By now the main components are clear – microbiome stability, barrier integrity, hydration, and immune resilience shape how the gut responds to unfamiliar environments.

Rather than relying on a single “anti-diarrhea trick,” it is more useful to think in layers.

Before travel, a few preparations can improve baseline resilience. Starting *Saccharomyces boulardii* several days before entering high-risk regions has some of the strongest evidence among probiotic strategies (McFarland, 2010; Kollaritsch et al., 1993). Maintaining fiber intake supports microbial fermentation and SCFA production, strengthening epithelial barrier function (Koh et al., 2016). Adequate zinc

intake supports epithelial repair and immune competence (Fischer Walker & Black, 2010).

During travel, the objective is stability. Hydration supports mucosal function. Gradual introduction of unfamiliar foods allows the microbiome to adapt. Reducing (or avoiding) alcohol removes an additional destabilizing input. Continuing *S. boulardii* may help maintain microbial competition.

If symptoms appear, priorities become simple and physiological. Fluid and electrolyte replacement comes first. Zinc may support epithelial recovery and shorten diarrheal episodes in some cases (Fischer Walker & Black, 2010). Activated charcoal may reduce toxin-related irritation in mild cases, although it does not treat infection. Severe symptoms – fever, persistent vomiting, or bloody diarrhea – require medical evaluation (Connor, 2015).

Their effect is cumulative rather than singular.

They simply reduce the number of destabilizing variables acting on the gut at the same time.

And in travel physiology, reducing chaos is often the most effective strategy available.

Oxidative Load & Recovery: Mitochondrial & Antioxidant Support

Now we arrive at another layer of physiology that quietly shapes how the body handles stress: **oxidative load**.

Every cell constantly produces reactive oxygen species (ROS) as a byproduct of energy metabolism. These molecules are often described as harmful, but that description is incomplete. In moderate amounts, ROS function as signaling molecules that regulate immune responses, cellular adaptation, and mitochondrial remodeling.

The problem appears only when production exceeds the systems designed to neutralize them.

Under normal conditions, the body maintains a sophisticated network of antioxidant defenses. Enzymatic systems such as superoxide dismutase, catalase, and

glutathione peroxidase continuously convert reactive molecules into less harmful compounds. Small molecular antioxidants – glutathione, uric acid, vitamin C, and numerous plant polyphenols – support these enzymatic systems and help maintain cellular redox balance (Sies, 2017).

When metabolic demands fluctuate, environmental stress increases, or recovery becomes irregular, ROS production can temporarily rise above this buffering capacity. This state is commonly referred to as **oxidative stress**.

Sleep disruption alters mitochondrial efficiency. Air pollution introduces oxidizing compounds into respiratory tissues and circulation. Psychological stress increases catecholamine signaling and inflammatory activity. Changes in diet can reduce the intake of plant compounds that normally support antioxidant defenses.

Even dehydration can influence inflammatory signaling and metabolic efficiency.

None of these factors are catastrophic on their own. But when several of them converge, oxidative pressure can rise enough to influence fatigue, immune resilience, and recovery.

This layer of physiology explains why certain behaviors appear repeatedly throughout this book. Sleep protection, sunlight exposure, movement, and nutrient intake are not only about comfort or mood. They influence mitochondrial function and redox balance – two systems that quietly determine how well the body adapts to stress.

Mitochondria: Energy Production and Oxidative Byproducts

Mitochondria are often described as the “power plants” of the cell. The analogy is not entirely wrong, but it is also incomplete. Mitochondria are not passive energy generators quietly sitting in the background. They are dynamic metabolic regulators that integrate signals from nutrient availability, hormonal signaling, circadian timing, and cellular stress.

To understand why oxidative stress appears in the first place, we need to briefly revisit something that may sound like high-school biology.

Cells run on a molecule called **ATP – adenosine triphosphate**. ATP is essentially the universal energy currency of biology. Almost every process that requires energy

inside the cell – muscle contraction, protein synthesis, ion transport across membranes, neuronal signaling – ultimately draws from ATP.

Mitochondria are the structures responsible for producing most of this ATP.

Inside the mitochondria, nutrients derived from carbohydrates and fatty acids are processed through a sequence of metabolic reactions that ultimately feed electrons into the **electron transport chain**. This chain of protein complexes sits within the inner mitochondrial membrane and functions almost like a microscopic electrical circuit.

As electrons move through this chain, their energy is used to pump protons across the mitochondrial membrane, creating a gradient. That gradient then drives a remarkable molecular machine called **ATP synthase**, which uses the proton flow to assemble ATP molecules from ADP and inorganic phosphate.

It is an elegant system, but not a perfectly efficient one.

A small fraction of electrons escape the electron transport chain before reaching their final destination and react prematurely with oxygen. When this happens, **reactive oxygen species (ROS)** such as superoxide radicals are formed. Estimates vary, but a small percentage of mitochondrial oxygen consumption normally produces ROS as a byproduct of energy metabolism (Murphy, 2009).

Importantly, this is not simply a design flaw.

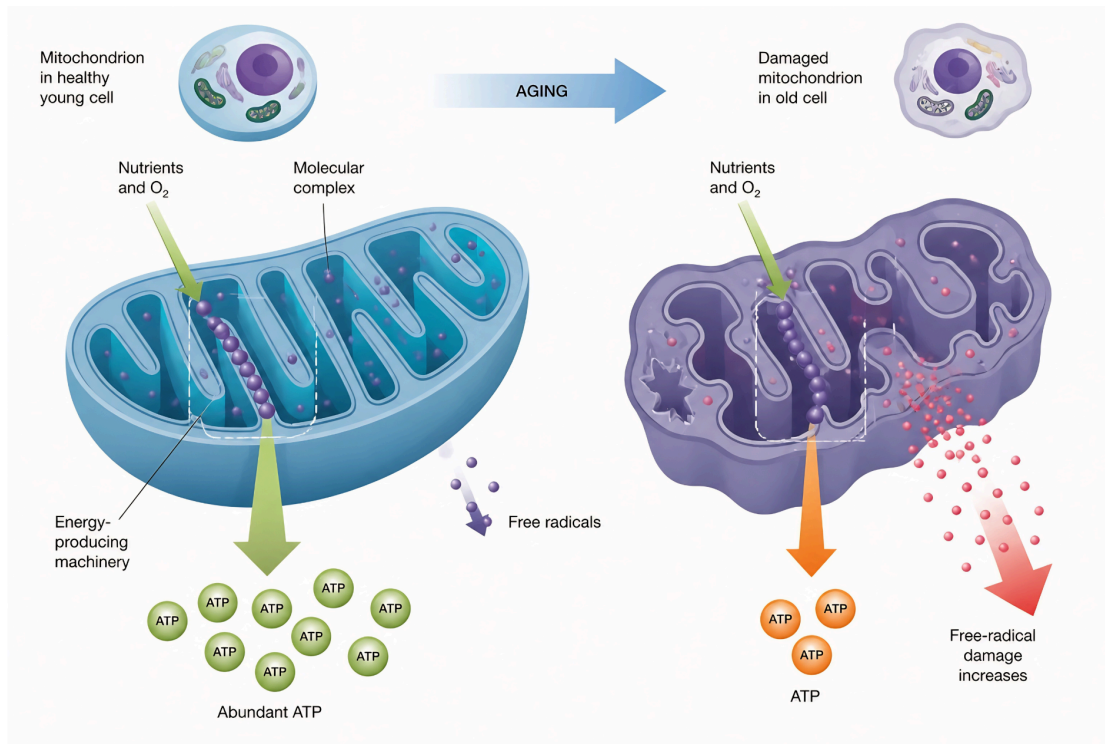
ROS act as signaling molecules that help regulate mitochondrial adaptation, immune responses, and cellular repair processes. The ROS generated during muscular activity, for example, help trigger some of the adaptations associated with physical training.

The real problem appears when ROS production rises beyond the capacity of the body's antioxidant defenses.

Sleep deprivation, inflammation, toxin exposure, and metabolic instability can all impair mitochondrial efficiency. When electron transfer becomes less controlled, more electrons escape the chain, increasing ROS formation.

Under those conditions, the signaling system that normally drives adaptation begins to resemble oxidative damage.

And environments that combine metabolic stress, environmental exposure, and disrupted recovery make this imbalance much more likely.



This schematic illustrates how mitochondrial energy production can shift under conditions of increased electron leakage, leading to elevated ROS. While ROS normally participate in signaling, excessive levels can contribute to cellular damage and are associated with aging-related processes. The diagram also highlights how mitochondrial dysfunction and ROS production can reinforce each other, creating a self-amplifying cycle under sustained stress.

Sleep Loss and Oxidative Stress

Sleep disruption is one of the most consistent physiological stressors associated with long-distance travel. Jet lag, early departures, unfamiliar sleeping environments, and nighttime noise frequently shorten sleep duration and fragment sleep architecture.

Experimental research shows that sleep deprivation increases markers of oxidative stress in multiple tissues, including the brain, liver, and skeletal muscle (Everson et al., 2005). These changes are closely linked to mitochondrial metabolism and the cellular repair processes that normally occur during sleep.

During healthy sleep cycles, metabolic activity shifts toward restoration. Antioxidant systems regenerate reduced glutathione, DNA repair pathways become more active, and inflammatory signaling declines. These processes help maintain redox balance after the metabolic demands of the waking day.

When sleep is shortened or repeatedly fragmented, this restorative window becomes less effective. Experimental sleep restriction has been associated with

reduced activity of antioxidant enzymes and increased **accumulation of oxidative damage markers** such as lipid peroxidation products (Villafuerte et al., 2015).

Across consecutive nights of insufficient sleep, these metabolic byproducts accumulate while mitochondrial efficiency gradually declines. The result is a measurable increase in oxidative pressure within cells, particularly in tissues with high energy demand.

In that sense, sleep is one of the main conditions under which redox balance is restored.

Sunlight, Circadian Biology, and Redox Regulation

Sunlight is one of the dominant environmental signals regulating oxidative physiology because it controls the timing of the circadian system.

Light entering the retina is transmitted to the suprachiasmatic nucleus in the hypothalamus, the central clock that coordinates daily rhythms throughout the body. Through hormonal and neural signaling, this system synchronizes oscillations in cortisol secretion, melatonin release, body temperature, immune activity, and metabolic pathways.

A large number of enzymes involved in oxidative phosphorylation and energy substrate utilization exhibit time-of-day variation in their activity. For example, enzymes such as **pyruvate dehydrogenase** – which regulates the entry of glucose-derived substrates into mitochondrial metabolism – and **carnitine palmitoyltransferase 1**, which controls the transport of fatty acids into mitochondria, show circadian variation in activity. Even components of the electron transport chain, such as cytochrome c oxidase, appear to be influenced by circadian timing. In other words, mitochondrial energy production is not constant across the day; it is rhythmically regulated (Peek et al., 2013).

When circadian timing becomes misaligned – for example after rapid time-zone shifts – this coordination begins to break down. Nutrient intake, hormonal signaling, and mitochondrial metabolism can temporarily fall out of phase with one another. Under these conditions, metabolic efficiency declines and mitochondrial electron transport becomes less tightly regulated, a state associated with increased production of reactive oxygen species.

Light exposure also influences several physiological systems intersecting with redox regulation. Ultraviolet radiation stimulates cutaneous vitamin D synthesis. Bright

light exposure affects nitric oxide signaling in vascular tissues. Retinal light input also modulates neurotransmitter systems that influence mood and inflammatory tone.

Taken together, these pathways make environmental light one of the most powerful regulators of metabolic timing in human physiology.

Regular exposure to natural daylight – particularly in the earlier part of the day – therefore provides a strong signal for stabilizing circadian alignment. And when circadian timing stabilizes, mitochondrial metabolism tends to stabilize with it.

Glutathione System

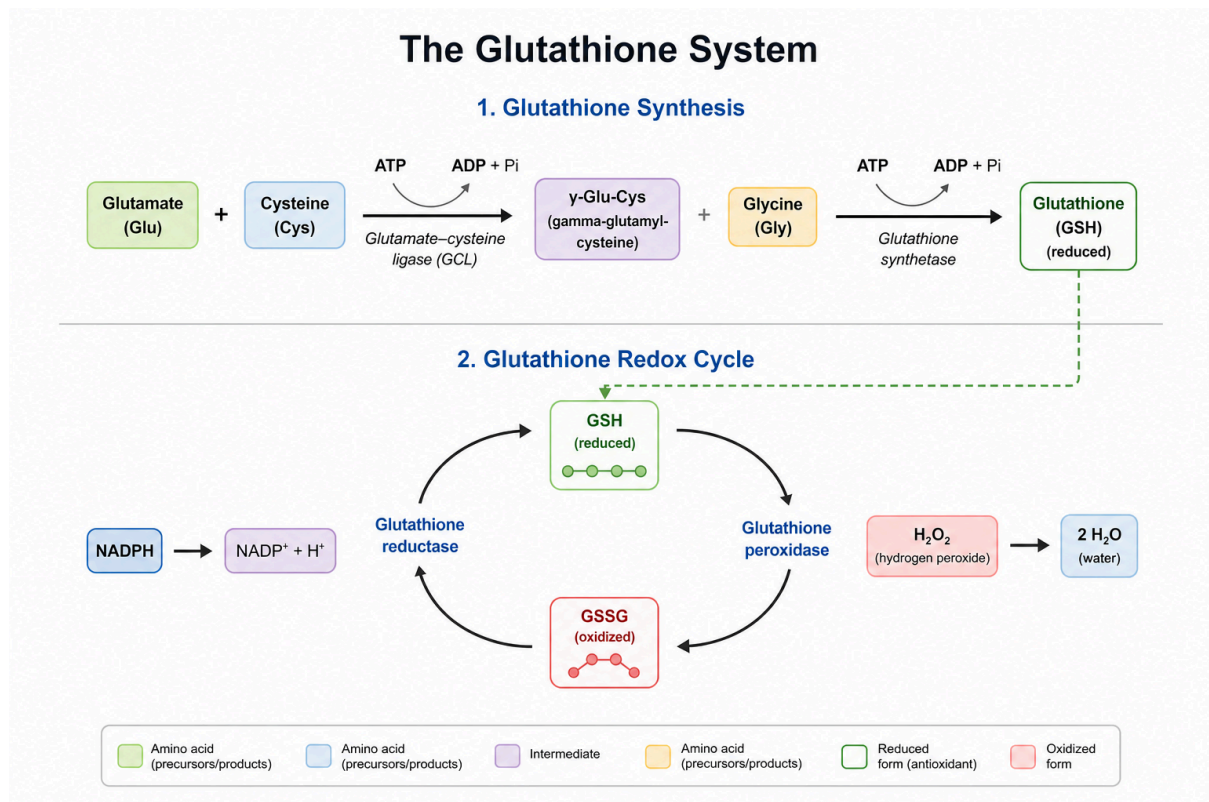
If sleep protection and circadian alignment help reduce the **production of reactive oxygen species**, the next layer of oxidative defense involves the systems that **neutralize and recycle those molecules once they are formed**.

Among the many antioxidant mechanisms in human physiology, one network stands out as particularly central: the **glutathione system**.

Glutathione is one of the central regulators of intracellular redox balance. It is a small tripeptide composed of three amino acids – cysteine, glycine, and glutamate – yet it participates in a wide range of metabolic processes that protect cells from oxidative damage.

Inside cells, glutathione exists primarily in two forms – reduced glutathione (GSH) and oxidized glutathione (GSSG). In its reduced state, glutathione can donate electrons to neutralize reactive oxygen species such as hydrogen peroxide and lipid peroxides. During this reaction, glutathione itself becomes oxidized, forming GSSG. Enzymes such as glutathione reductase then regenerate reduced glutathione using NADPH as an electron donor (Sies, 2017).

This redox cycle allows cells to buffer oxidative fluctuations without allowing reactive intermediates to accumulate. The ratio between reduced and oxidized glutathione is therefore widely used as a marker of cellular oxidative stress.



Glutathione is produced from three amino acids and functions as a central antioxidant by cycling between its reduced (GSH) and oxidized (GSSG) forms, allowing cells to neutralize reactive oxygen species and regenerate antioxidant capacity using NADPH.

Glutathione also plays a central role in detoxification. In the liver and many other tissues, enzymes known as glutathione S-transferases attach glutathione molecules to reactive compounds – including xenobiotics, heavy metals, and metabolic byproducts – making them more water-soluble and easier for the body to eliminate (Hayes et al., 2005).

These functions place the glutathione system at the intersection of antioxidant defense, metabolic regulation, and detoxification physiology.

In environments where oxidative exposure increases – particularly in polluted urban areas – the demand placed on this system can rise substantially.

Pollution Exposure and Oxidative Stress

Air pollution is one of the most significant environmental sources of oxidative stress encountered during travel. Urban environments with high concentrations of particulate matter, nitrogen oxides, sulfur compounds, and volatile organic pollutants introduce oxidizing agents directly into the respiratory tract.

These particles interact with lung tissue and immune cells, triggering inflammatory signaling and increasing ROS production. Fine particulate matter – especially particles **smaller than 2.5 micrometers (PM2.5)** – can penetrate deeply into the lungs and enter systemic circulation, contributing to oxidative stress in multiple organs (Kelly & Fussell, 2015).

Cities such as Delhi, Beijing, and parts of Southeast Asia frequently experience pollution levels that exceed recommended international safety thresholds.

When travelers enter these environments, their antioxidant systems suddenly face a higher oxidative burden than usual.

Studies examining pollution exposure consistently show increased markers of oxidative stress and inflammatory signaling in exposed populations. Elevated lipid peroxidation products, reduced glutathione levels, and increased inflammatory cytokines have all been observed following exposure to particulate air pollution (Pope & Dockery, 2006).

The body does possess mechanisms to cope with these exposures. Pulmonary macrophages remove particles from lung tissue, antioxidant enzymes neutralize reactive intermediates, and detoxification systems metabolize environmental toxins.

However, when pollution exposure becomes intense or prolonged, these systems can become temporarily overwhelmed.

For travelers spending several days in heavily polluted cities, supporting the glutathione system can therefore become relevant.

Glutathione Supplementation

Glutathione itself can be taken as a supplement, although the effectiveness of oral glutathione has been debated for many years. Earlier research suggested that glutathione might be broken down during digestion before reaching systemic circulation.

More recent studies indicate that oral glutathione may still influence intracellular glutathione status through indirect mechanisms, possibly by providing substrates for cellular synthesis or through partial absorption in the gut (Richie et al., 2015).

Other delivery forms, such as **liposomal glutathione**, have been developed in an attempt to improve bioavailability.

Mechanistically, increasing glutathione availability may help buffer oxidative stress and support detoxification pathways during periods of environmental exposure.

It is worth being precise about what “detoxification” actually means here. As we’ve already mentioned, the body is capable of dealing with airborne pollutants (so the body does not passively accumulate toxins until a supplement suddenly cleanses them away). But how exactly does the detoxification mechanism work throughout the entire system?

Detoxification is a continuous physiological process carried out primarily by the liver, kidneys, lungs, and gastrointestinal system. In the liver, especially, **Phase I and Phase II detoxification pathways** continuously modify and neutralize medications, pollutants, metabolic byproducts, and dietary compounds. Glutathione plays a central role in Phase II conjugation reactions, where reactive molecules are bound to glutathione and prepared for elimination through bile or urine (Hayes et al., 2005). So supporting glutathione availability does not activate some mystical cleansing mechanism. It supports biochemical systems that are already operating all the time.

In heavily polluted environments – such as large urban centers with persistent smog – some individuals therefore choose to take glutathione supplements as a precautionary measure.

When I spent time in some polluted cities in Asia, I took glutathione daily despite wearing a respiratory mask outdoors. The mask reduces particle exposure, but it cannot eliminate inhalation of ultrafine particles completely. Supporting antioxidant defenses seemed like a reasonable additional measure.

N-Acetylcysteine (NAC): Supporting Glutathione Production

While glutathione itself can be supplemented directly, another widely used strategy involves supporting the body’s own glutathione synthesis.

This is where **N-acetylcysteine (NAC)** becomes relevant.

NAC is a modified form of the amino acid cysteine, which serves as the rate-limiting precursor for glutathione synthesis. Because cysteine availability often limits the rate at which cells can produce glutathione, providing NAC can increase intracellular glutathione levels under certain conditions (Atkuri et al., 2007).

NAC has been studied extensively in medical contexts.

In clinical toxicology it is used as a treatment for acetaminophen overdose because it replenishes hepatic glutathione stores and prevents liver damage. It has also been investigated in respiratory diseases, where its mucolytic and antioxidant properties may improve lung function.

From a biochemical perspective, NAC offers two advantages.

First, it **provides cysteine for glutathione synthesis**, indirectly increasing intracellular antioxidant capacity.

Second, **NAC itself possesses mild antioxidant activity** and can interact directly with reactive species.

Several studies have shown that NAC supplementation increases glutathione levels and reduces oxidative stress markers in certain conditions (Atkuri et al., 2007).

So if you're a traveler looking to protect yourself from pollution and want to try something more comprehensive than glutathione, NAC may be a more practical option in this context.

Polyphenols

If mitochondrial metabolism produces reactive oxygen species and the glutathione system neutralizes many of them, the next layer of oxidative regulation involves something that is often overlooked when thinking about travel physiology – **dietary polyphenols**.

Polyphenols are a diverse group of plant-derived molecules found in coffee, tea, herbs, fruits, and many vegetables. Unlike vitamins such as vitamin C or E, polyphenols do not function primarily as direct antioxidants in the bloodstream. Their most important effects occur through **cellular signaling pathways**.

Many polyphenols interact with transcription factors such as **Nrf2**, a regulatory protein that controls the expression of antioxidant and detoxification enzymes. When Nrf2 is activated, cells increase production of protective enzymes including glutathione peroxidase, superoxide dismutase, and heme oxygenase (Kensler et al., 2007).

Polyphenols therefore do not merely neutralize reactive oxygen species themselves, but they stimulate the body's own **antioxidant defense machinery**.

Coffee, Cacao and Polyphenols

Coffee is one of the most significant sources of polyphenols in many modern diets. Although it is usually discussed in relation to caffeine, coffee contains large quantities of **chlorogenic acids and other phenolic compounds** that influence metabolism and redox signaling.

These compounds interact with oxidative pathways and inflammatory signaling networks. Several studies have shown that coffee consumption can increase antioxidant capacity in plasma and influence metabolic enzyme activity (Bakuradze et al., 2011).

Interestingly, many of the metabolic benefits associated with coffee appear to persist even when caffeine is removed, suggesting that polyphenols themselves play an important role.

As mentioned earlier in the book, coffee may also influence the microbiome. Certain bacterial species associated with coffee consumption participate in fermentation pathways that generate short-chain fatty acids, which support intestinal barrier stability and metabolic regulation (Vieira-Silva et al., 2020).

So yes – I’m promoting coffee again. It is, in many ways, a surprisingly complex plant extract.

Cacao represents another dense source of polyphenols, particularly flavanols such as **epicatechin**, which have been studied for their effects on vascular function and cellular signaling.

Mechanistically, cacao flavanols appear to influence **endothelial nitric oxide production**, improving vascular function and blood flow (Grassi et al., 2005; Schroeter et al., 2006). Emerging research also suggests activation of Nrf2-related pathways and potential effects on mitochondrial function (Ramirez-Sanchez et al., 2024).

The dose–response relationship, however, matters. Larger amounts of cacao – especially in more concentrated forms – can produce noticeable physiological effects, including cardiovascular stimulation in sensitive individuals like me. Lower doses tend to provide polyphenolic input without strong stimulation. Personally, I usually stay around 5 grams per day, which seems to strike a reasonable balance.

Sourcing becomes relevant for both cacao and coffee, although for slightly different reasons. In cacao, polyphenol content is strongly influenced by processing methods, particularly alkalization (“Dutch processing”), which can significantly reduce flavanol levels. Fermentation and roasting also modify the final composition,

but alkalization is one of the most decisive factors. Minimally processed cacao therefore tends to retain higher levels of bioactive compounds.

In coffee, polyphenol content is shaped primarily by bean variety, roasting, and preparation method. Lighter roasts generally preserve more chlorogenic acids than darker roasts, although darker roasting introduces other compounds with their own biological activity. Brewing method and filtration also influence the final chemical profile.

This topic could easily fill an entire book on its own, but the key idea is straightforward – the way coffee and cacao are processed and prepared meaningfully shapes their biological effects. I firmly believe that with your skills, you'll be able to find some really good coffee (decaf!) or cocoa :)

Greek Mountain Tea

Greek mountain tea is one of my favorite herbs, and I use it regularly. It would not be unreasonable to place it in the chapter on autonomic regulation and sleep, because it can have mild calming effects and is traditionally consumed in the evening. But there are literally thousands of herbs that could be placed in that category. Chamomile, lemon balm, lavender, passionflower, valerian, and countless others all have some relationship to relaxation or sleep support.

Greek mountain tea, however, seems to operate through mechanisms that are broader and more interesting than simple autonomic down-regulation, which is why I prefer to place it here, in the chapter on oxidative load and recovery.

Greek mountain tea is derived primarily from plants of the **Sideritis** genus – most commonly *Sideritis scardica*, *Sideritis raeseri*, and related species – and has been consumed for centuries in mountainous regions of the Mediterranean. Traditionally, it was used by shepherds and rural communities as a daily tonic beverage, especially during colder seasons, valued not only for its flavor but also for its perceived restorative and protective effects.

Modern phytochemical analysis has shown that *Sideritis* species contain a diverse range of biologically active compounds, including flavonoids such as apigenin and luteolin derivatives, phenylpropanoid glycosides, phenolic acids, and diterpenes. These compounds interact with oxidative and inflammatory signaling pathways in several ways, including modulation of antioxidant defense systems such as (our favourite) Nrf2 pathway that regulates the expression of endogenous antioxidant enzymes (González-Burgos et al., 2011).

What makes this plant especially interesting is that its potential effects appear to extend beyond general antioxidant activity.

A growing body of experimental work suggests that extracts from *Sideritis scardica* can influence processes associated with neuroinflammation and neural resilience. In cellular and animal models, these extracts have been shown to reduce markers of oxidative stress in neuronal tissue and to modulate inflammatory signaling pathways associated with neural damage (González-Burgos et al., 2011; Tadić et al., 2012).

Some research has also explored the plant's influence on processes related to **demyelination**, the degradation of the myelin sheath surrounding nerve fibers. Myelin acts as electrical insulation for axons, allowing signals to travel efficiently through neural circuits. Damage to myelin slows neural conduction and is involved in several neurological disorders. Experimental findings suggest that compounds from *Sideritis* plants may help protect neural tissue from oxidative and inflammatory mechanisms contributing to demyelination (Tadić et al., 2012).

This is one reason I personally find Greek mountain tea so interesting from a cognitive perspective. Its potential effects appear less related to direct stimulation and more related to stabilizing the biochemical environment in which neurons function. Oxidative stress and neuroinflammation are increasingly recognized as contributors to cognitive fatigue, neurological aging, and stress-related neural dysfunction. Compounds that reduce oxidative damage and inflammatory signaling in neural tissue may therefore support cognitive resilience indirectly by protecting neural structure and metabolism.

Some early human research points in the same direction. In a clinical study examining supplementation with *Sideritis scardica* extracts over several weeks, participants showed improvements in mood and certain cognitive parameters. Researchers suggested that these effects might be related to modulation of **monoamine neurotransmitter systems** and antioxidant signaling pathways (Wesnes et al., 2016). The study was small and should not be treated as definitive evidence of cognitive enhancement, but it fits well with the broader experimental literature showing neuroprotective and anti-inflammatory activity of the plant.

In travel contexts, this becomes relevant because many of the variables we've discussed – sleep disruption, circadian misalignment, pollution exposure, and stress – converge on oxidative and inflammatory pathways that also affect neural function.

Greek mountain tea is a truly wonderful and luxurious herb, especially after a hard and exhausting day. It makes you feel wonderfully calm, meditative, and alert at the same time.

And that is no coincidence.

I definitely recommend it to everyone.

That combination of **polyphenolic signaling, potential neuroprotective activity, and gentle physiological support** is why I consider Greek mountain tea one of the most elegant herbal tools in this entire book.

Movement as a Redox Signal

Physical movement increases the production of reactive oxygen species (ROS) inside muscle cells. At first glance, that sounds counterproductive. ROS are usually framed as harmful molecules associated with oxidative damage, so it is easy to assume that adding movement during periods of fatigue or stress only adds more strain.

But physiology is more nuanced.

In moderate amounts, ROS generated during muscular activity act as signals rather than damage. They initiate adaptive responses inside the cell, activating pathways that increase mitochondrial biogenesis and upregulate antioxidant enzyme systems. Over time, this shifts the system toward greater resilience – cells become better equipped to manage oxidative stress rather than more vulnerable to it (Powers et al., 2011).

This process is known as **hormesis**, and in the context of mitochondria more precisely as **mitohormesis** – a transient, controlled increase in oxidative stress that drives improvements in mitochondrial function and cellular defense capacity.

Small bursts of oxidative stress, in this sense, function as instructions. They push the system to upgrade its metabolic machinery.

This is reflected consistently in physiology. Regular physical activity correlates with improved mitochondrial efficiency, higher antioxidant enzyme activity, and greater metabolic flexibility.

Importantly – just as was the case with the circadian rhythm and circulation – the threshold for triggering these signals is not high.

Relatively small amounts of movement are sufficient. Walking, light mobility work, or short bouts of exercise stimulate muscular metabolism, support circulation, and maintain mitochondrial signaling without overwhelming the system.

The key variable here is intensity.

High-intensity exertion during dehydration, sleep deprivation, or illness can push oxidative stress beyond the adaptive range and become counterproductive. Moderate movement tends to have the opposite effect. It provides enough stimulation to keep energy systems active and responsive, without destabilizing them further.

After long flights, disrupted sleep, or environmental change, the body often feels slow and uncoordinated. In that state, gentle movement is one of the most reliable ways to restore metabolic rhythm.

Not because it removes stress, but because it recalibrates how the system responds to it.

Cold Exposure and Recovery Signaling

Cold exposure has already appeared earlier in the context of autonomic regulation. Here, it becomes relevant from a slightly different angle – not just as a nervous system stimulus, but as a signal influencing mitochondrial activity and oxidative balance.

When the body encounters cold temperatures, several physiological responses occur. Sympathetic nervous system activity increases, peripheral blood vessels constrict, and thermogenic pathways become active.

At the cellular level, cold exposure appears to influence mitochondrial activity and antioxidant responses.

Some experimental studies suggest that repeated cold exposure can stimulate mitochondrial biogenesis and improve metabolic efficiency, possibly through signaling pathways involving **PGC-1α** and other regulators of mitochondrial adaptation (Chung et al., 2017).

Cold exposure may also increase levels of certain antioxidant enzymes and improve resilience to oxidative stress in some experimental models.

In that sense, cold functions similarly to movement – a controlled stressor that, when applied in moderate doses, can stimulate adaptive responses rather than cause damage.

The practical application of this has already been discussed earlier – cold exposure does not need to be extreme to be useful.

Recovery Windows, Strategy, and the Bigger Picture

One of the most practical ways to understand oxidative load during travel is to look at what happens when the body **doesn't get a break**.

Flights, reduced sleep, artificial light at night, unfamiliar food, pollution, psychological friction – none of these are extreme in isolation. The problem is their convergence, and the fact that they often arrive without interruption. The system keeps adapting, but it does so continuously, without a phase where it can fully return to baseline.

This is where recovery windows become relevant.

A recovery window is not a technique or a protocol. It is a period where incoming stress is reduced long enough for regulatory systems to re-synchronize.

In practice, a recovery window can be surprisingly simple. It might be a full night of uninterrupted sleep in darkness after several fragmented nights. It can be a morning with natural light exposure and unhurried movement after a long travel day. It can be a period of several hours without constant stimulation – no screens, no noise, no metabolic overload – where the system is allowed to downshift.

It does not need to be perfect. It only needs to be long enough and coherent enough for regulatory processes to catch up.

You might be wondering why there's a whole chapter here on something so extremely obvious.

The point is, however, that some of the most obvious things are often overlooked, and yet they form the foundation for everything else.

When sleep is consolidated and occurs in darkness, antioxidant systems shift toward restoration – glutathione is regenerated, mitochondrial repair processes become more efficient, and inflammatory signaling declines.

When light exposure aligns with daytime, circadian signaling stabilizes, improving coordination between hormonal rhythms and mitochondrial metabolism.

When movement is present but not excessive, mitochondrial signaling remains active without pushing oxidative stress beyond the adaptive range.

When food becomes simpler and more predictable, metabolic variability decreases and digestive load stabilizes.

When hydration is maintained, circulatory and cellular processes operate with less constraint.

Without these periods, oxidative load accumulates quietly – slightly worse sleep, slower recovery, increased inflammatory tone, reduced metabolic flexibility.

When recovery windows are present, that drift tends to reverse.

At this point, it becomes difficult to treat individual interventions as separate.

It is tempting to isolate them – something for mitochondria, something for sleep, something for stress, something for the gut. But physiologically, that separation does not exist.

So the relevant question is not which single strategy works best.

It is whether the system is being guided in a coherent direction or continuously pulled apart by competing inputs.

Travel inevitably disrupts timing, environment, and routine. That cannot be eliminated, and it does not need to be. What matters is introducing enough structure for adaptation to remain efficient.

Oxidative balance sits at the center of that process.

Mitochondria generate energy and reactive oxygen species. Antioxidant systems regulate those signals. Circadian rhythms coordinate timing. Nutrient inputs shape metabolic capacity. Environmental exposures increase or decrease stress load.

When these elements remain even partially aligned, the system adapts with relatively low cost.

When they drift too far out of phase, recovery slows, and the same environment begins to feel heavier than it should.

And it is exactly where small, well-placed recovery windows start to matter.

Decision Layer

At this point, you have enough tools.

Sleep strategies, light management, movement, nutrition, circulation, oxidative balance, supplementation – there is no shortage of interventions. In fact, there is an

almost absurd number of things you *could* do. For every system discussed in this book, there are additional layers, more precise protocols, more compounds, more timing strategies, more edge cases.

You could keep adding.

And many people do.

At some point, the question is no longer *what works*.

It becomes:

What do you actually use when everything is happening at once?

Because that is the real condition of travel.

Not a clean protocol. Not a controlled environment. Not a situation where you can execute everything exactly as planned.

But a moving system with constraints, interruptions, and imperfect conditions.

This is where most people – and especially those who care about optimization – make a predictable mistake.

They try to manage everything.

They start thinking in terms of complete control. Light exposure must be perfect. Sleep must be engineered. Supplements must cover every pathway. Movement must be optimized. Food must be clean. Every variable becomes something to track, adjust, and refine.

It quickly turns into a form of identity signaling.

The person with the perfect stack. The perfect routine. The perfect protocol.

Waking up, chasing sunlight, managing caffeine timing, thinking about microbiome disruption before breakfast, considering circulation during a taxi ride, worrying about oxidative load while trying to fall asleep in a noisy room.

The system becomes overloaded – not because of travel itself, but because of the attempt to control every consequence of it.

There is a point where optimization stops being useful and it is the moment where you are no longer responding to the environment – you are reacting to your own expectations.

That is where the decision layer becomes necessary.

I've warned you throughout this book about the stress that comes with hyper-perfectionism and the urge to optimize everything. However, simply knowing this changes nothing. It is easy to say, "Pick only what is truly suitable from this entire book of biohacks." It sounds reasonable. It even sounds simple.

But it leaves the real problem untouched.

How do you actually choose?

Recognizing Breakdown

When multiple stressors accumulate, the body does not fail everywhere at the same time. It shifts unevenly. Certain systems destabilize earlier and more visibly than others.

And that is why understanding which system is drifting first is more useful than trying to correct everything simultaneously.

Most travel-related disruption tends to cluster around a few domains:

- circadian rhythm and sleep
- nervous system regulation
- metabolic stability
- circulation and physical movement
- oxidative load and environmental exposure

Each of these systems interacts with the others, but they do not collapse in the same order or at the same speed.

Sometimes the signal is obvious: poor sleep after crossing time zones, difficulty falling asleep in a new environment, waking too early or too late. Sometimes it appears as nervous system activation – restlessness, irritability, difficulty relaxing despite fatigue. In other cases, it is metabolic: unstable appetite, digestive irregularity, fluctuating energy. Long periods of immobility show up as stiffness, heavy legs, and sluggish circulation. Environmental stressors such as pollution or

sustained psychological load may not be immediately noticeable, but they increase background oxidative pressure.

The goal is not to analyze every sensation in detail – it is to recognize which system is drifting first.

Because that determines what actually needs attention.

Hierarchy: What Actually Matters

When everything feels slightly off, prioritization becomes more important than intervention.

Not all variables carry the same weight.

There is a clear hierarchy in how physiological systems respond to disruption, and respecting that hierarchy simplifies decision-making under pressure.

At the top:

Sleep and circadian alignment

This is where regulation begins. Sleep and circadian timing coordinate processes across the body – redox balance, immune activity, hormonal rhythms, cognitive function, and metabolism. When timing drifts and sleep fragments, coordination between systems weakens. Under those conditions, even well-chosen interventions tend to have smaller and less predictable effects.

Below that:

Hydration and electrolyte balance

Fluid and electrolyte status influences circulation, neuronal signaling, and cellular function. Changes here are often rapid and systemic. Even mild dehydration can increase fatigue, alter cardiovascular dynamics, and impair performance.

Then:

Movement

Not as performance, but as a signal.

Movement maintains circulation, supports endothelial function, and stimulates mitochondrial activity. It prevents the physiological stagnation that follows prolonged sitting.

After that:

Food

Composition matters, but consistency often matters more in unstable environments. Irregular meals, unfamiliar foods, and shifting timing increase metabolic variability. Simplifying inputs reduces that variability and lowers the burden on already stressed regulatory systems.

And only then:

Supplements

They can support specific pathways, but they do not replace the layers above. Their effect depends heavily on the state of the system they are entering. When foundational inputs are unstable, supplementation produces smaller and more variable outcomes. When those inputs are aligned, the same compounds become more effective.

This hierarchy answers a practical question:

What can be ignored?

If sleep is severely disrupted, adjusting micronutrients will not compensate for it. If hydration is poor, adding supplements will not stabilize the system. If movement is absent, circulation will not improve through intention alone.

Travel is not about doing everything. It is about doing the right things first.

Once that order is clear, decision-making becomes much simpler.

Stabilize vs Optimize

There is a difference between improving a system and keeping it from drifting.

Instead of asking, “How can this be perfect?” the question becomes:

“How can this stay within a functional range?”

That shift reduces the number of variables that need to be managed and makes decisions easier in real time.

A slightly imperfect system that remains stable will outperform a perfectly designed protocol that cannot be maintained.

Remove vs Add

When something feels off, the default reaction is to add.

Another supplement. Another tool. Another adjustment.

Sometimes that helps. Often it doesn't.

Because many travel-related disturbances are not caused by deficiency, but by excess input:

- too much light at the wrong time
- too much noise
- too many competing signals
- too much irregularity

In those conditions, adding more rarely restores stability. It usually increases complexity.

A simple example is sleep.

Someone sleeps poorly in a new environment and starts adding support – magnesium, then another form of magnesium, then glycine, then an herbal extract. The stack grows, each addition intended to push the system toward sleep.

At the same time, the room still leaks light. There is intermittent noise. The circadian signal is misaligned.

In that situation, the limiting factor is not the absence of another compound. It is the presence of disruptive inputs.

Reducing light, lowering noise, and stabilizing the environment often shifts the system more than adding another supplement.

The same pattern appears elsewhere.

Problems are addressed by adding support while the underlying disruption remains in place.

Removing destabilizing inputs changes the baseline. Adding interventions attempts to compensate for it.

Both approaches have value.

But during travel, removing friction is often the faster and more reliable strategy.

The Minimum Effective Travel Stack

At some point, theory runs into reality.

You already know how these systems work. You already know what matters.

But travel doesn't give you perfect conditions. It gives you constraints.

So the question changes:

Not *what works*. Not *what's optimal*.

What do you actually carry and use when things are messy?

Because you don't carry a protocol.

You carry a few things. And you use them well.

So if you just want the bare essentials and don't feel like thinking too hard, this brief section offers a handful of tools that every biohacker should definitely use (and why). Of course, something else might work better for your needs (mostly from the supplements section), but take this as a recommendation, not a blueprint.

1. Remove interference first

Start with what interferes directly with regulation – the physical environment.

What I bring:

- eye mask
- small piece of tape (for LEDs, standby lights, small light leaks)
- earplugs or a simple white noise solution

- blue light blockers

Light and noise are not subtle inputs. They directly influence circadian timing and sleep architecture. When that signal is unstable, coordination across systems degrades, and sleep disruption alone is enough to increase oxidative stress markers across tissues.

This is why this layer sits first – you are not improving just sleep. You are removing interference and taking care of a vast amount of variables at once.

2. Stabilize the system

Once the environment is not actively working against you, you stabilize the system.

What I bring:

- electrolyte packets (I often improvise and use whatever is available)
- magnesium (ideally Bisglycinate or L-threonate)
- compression socks (especially for long travel days)

And one rule that matters more than most supplements:

- **keep food simple when everything else is chaotic**

This layer is where most of the leverage is.

If hydration, circulation, and metabolic input are stable, the body tolerates a surprising amount of imperfection.

If they are not, everything starts to drift.

3. Situational Tools (only when something breaks)

Only now do you consider adding.

But not as a routine or some kind of cool toolkit. But as a response.

What I might carry:

- *Saccharomyces boulardii* (when gut instability appears)
- zinc (during illness or recovery)
- NAC or glutathione (in high pollution environments)

- glycine (for sleep – but only if environment is already handled)
- kava (for acute nervous system overactivation – for example after long travel days, time-zone shifts, or situations where the body feels “wired but tired”)

These are conditional tools, and if nothing is clearly failing, they stay unused.

The actual rule

Most tools are conditional.

They work when the system is already reasonably stable. They become unreliable when it isn't.

So you don't carry everything forward.

You filter:

- What removes disruption?
- What stabilizes quickly?
- What is actually needed right now?

Everything else is optional.

And under real conditions, **optional things are the first to go.**

When Things Go Wrong: Real Travel Scenarios

So, now we have everything we need. Or do we? We know the theory, we know how to make decisions, but still – something could always happen to derail even our wonderfully Zen and non-perfectionist plans. Flights get delayed. Cities hit you with air you're not supposed to breathe. Restaurants that look perfectly fine wreck your gut. Your nervous system gets pushed past what you thought was your limit.

The point isn't to avoid these situations. You won't. The point is still the same one I keep repeating in this book – to recognize what's actually breaking, and intervene at the right layer instead of guessing and trying randomly.

So let's take a look at some possible scenarios we might encounter as travelers. Things we didn't expect and things that can really get under our skin. I've mainly chosen situations I've experienced myself and wasn't exactly thrilled about.

1. Long-haul Flight Collapse

There's a specific kind of fatigue that only shows up after certain flights.

Not just tired. Not even jet lag in the usual sense. It's a dry, hollowed-out feeling. Eyes burning. Skin tight. Breathing slightly off. As if your whole body had been dried out from the inside.

I felt it hard on a flight from Brazil to Lisbon. Boeing 777. Nothing unusual on paper. Not even my longest flight.

But it felt worse than a much longer one I did before – Madrid to Asunción. That one was on a 787 by the way.

And that difference matters more than most people think.

The 787 isn't just a newer plane – it's built with a composite fuselage instead of aluminum, and that one design decision quietly changes what the cabin actually feels like.

Older aircraft keep humidity extremely low, sometimes down to 5%. That's desert-level dryness. The 787 can go higher – closer to 15–20%. Cabin pressure is also slightly different, with a lower effective cabin altitude. Small shift, but physiologically noticeable. And it shows up in actual data, not just anecdotes – controlled cabin environment studies report less eye dryness, less mucosal irritation, and lower perceived fatigue at higher humidity and lower cabin altitude, along with better overall comfort and sleep quality (Nagda & Koontz, 2003; Spengler et al., 2012). Similar patterns appear in field and manufacturer-supported comparisons between composite aircraft cabins and conventional aluminum designs (Boeing, 2014; Lee et al., 2015).

At 5% humidity, your body is constantly losing water through evaporation – eyes, airway, skin. You don't feel it as thirst first. You feel it as irritation. Your tear film destabilizes. Your mucosa dries out. Breathing gets harsher. Everything becomes slightly inflamed.

That Lisbon flight felt like that. My eyes were burning halfway through. Not tired – burning. Literally...

The Madrid to Asunción flight didn't do that. Same human, same habits. Different plane.

So when people talk about long-haul flights, they immediately jump to jet lag. That's not the first thing that hits you. The first thing is physical degradation – fluids, circulation, basic tissue condition.

Jet lag comes after. If you're already dried out and stagnant, your circadian system has less room to adapt.

The fix is not complicated, but it's easy to ignore.

You don't wait until you feel bad. You prevent the state from forming.

Drink regularly, not aggressively. Water alone is not enough if you're flushing electrolytes out. You don't need a formula – just don't combine liters of water with zero sodium. Eat something slightly salty. That's enough.

Move, even if it feels pointless. Stand up, walk a bit, contract your calves. It's not exercise. It's circulation maintenance. People underestimate how much just not moving for hours changes how they feel.

And protect your face from the environment. Those air vents blasting directly into your eyes – that's literally accelerating tear evaporation. It's a stupid detail, but it really matters a lot.

Circadian strategy is simpler than people make it.

Decide early: are you sleeping or staying awake?

Then commit.

Long-haul flights are not something you win. You just avoid losing too much.

2. Polluted City

When I went to Delhi after Diwali, it was extremely aggressive. I still remember the feeling.

AQI was around 300–400. That's not just “bad air.” At that level, you're inhaling a high concentration of fine particulate matter (PM2.5) with every breath – particles small enough to penetrate deep into the lungs, pass into the bloodstream, and trigger systemic inflammation.

You notice it quickly, but not always in the way people expect. It's not just coughing or obvious respiratory distress. Breathing feels slightly heavier, less clean. There's a

constant low-level irritation. But the more relevant effects are internal – oxidative stress, endothelial dysfunction, and a measurable increase in inflammatory signaling. You're not just breathing polluted air; you're loading the system continuously.

The default reaction when you arrive in a new city is to move. Walk, explore, spend time outside. Under these conditions, that instinct works against you. Ventilation rate increases with movement, which means you're pulling more air – and more particles – into the system per minute. What would normally be a healthy behavior becomes a multiplier of exposure.

So the strategy has to change.

In my case, that meant minimizing time outside and using a proper mask consistently. Not a loose covering, but an actual filtration mask that meaningfully reduces particulate intake. That alone changes the total load you're dealing with.

At the same time, removing outdoor movement creates another problem. After meals – especially higher-carbohydrate ones – you still get the usual metabolic response: glucose rises, insulin follows, and the body benefits from movement to stabilize that process. In a heavily polluted environment, going outside to “walk it off” is not a good trade.

So I kept the movement, but changed the environment.

Most of it happened indoors. Nothing complicated – just enough movement to keep circulation and glucose handling from drifting in the wrong direction. Sometimes that meant putting on music and dancing (yes, I tried that as well while staying in Delhi), sometimes simple animal movements, sometimes basic yoga flows. Nothing precise, nothing programmed – just enough to keep the system active without increasing exposure. It's a simple adjustment, but it prevents you from sacrificing one system while trying to protect another.

The supplement layer sits below that.

Compounds like NAC or glutathione support endogenous antioxidant systems, particularly through glutathione pathways that are directly involved in neutralizing reactive oxygen species generated by particulate exposure. There's evidence that air pollution increases oxidative stress and depletes these systems, so supporting them makes sense. But this is secondary. It does not offset continuous exposure.

If you keep inhaling high levels of pollutants, no supplement meaningfully compensates for that.

So the structure is straightforward.

Reduce exposure first.

Support internal defense second.

Maintain baseline functions – like movement – in a way that doesn't increase the problem.

And accept that in environments like this, you're not optimizing anything. You're just containing the damage.

3. Gut Instability

This one feels unfair when it happens.

You go to a good restaurant. Clean, well-rated, looks legitimate. You order something reasonable. Nothing extreme. And a few hours later, your body rejects everything.

Vomiting. Diarrhea. Full reset.

It happened to me in Delhi. Yes, Delhi is a truly uncompromising city.

The food itself wasn't the problem. I went back later, ate there again, and nothing happened.

The difference was one detail.

The first time, I had a salad.

Raw vegetables, washed in local water. That's enough.

Cooking reduces microbial load. Heat eliminates most pathogens. Raw food doesn't. So even in a well-run kitchen, you're exposed if the water behind it isn't something your system is used to.

Moreover, food poisoning is often framed as "bad hygiene" or "bad restaurants." But that's an extreme oversimplification. In practice, it's frequently just a mismatch between exposure and adaptation.

Your gut is not static. It's an ecosystem. And travel disrupts it quickly.

New location means a new bacterial environment – different strains, different ratios, different background exposure. Your existing microbiome isn't adapted, and your immune system has to respond in real time. If the incoming load exceeds what your system can handle, you get symptoms.

The mistake people make here is trying to maintain their usual idea of a “good diet.”

More fiber (in my case – this was especially tricky and that salad from Delhi really ruined my day...). More variety. Try local foods. Explore.

Under stable conditions, that makes sense. In an unstable gut, it creates more variables than the system can process.

So you simplify.

Cooked food. Repetitive meals. Minimal novelty.

If you want fiber, control the source. Something predictable – chia seeds, something packaged, something you trust. Not raw vegetables washed in unknown water.

Peel fruit. Wash what you can yourself. Reduce exposure where it actually matters.

After that first experience, I went back to the same restaurant, skipped the salad, and everything was fine.

Same place. Same kitchen. One variable removed.

That's usually how these situations work.

4. Nervous System Overload

There's a moment where things stop being logistical and start being internal.

And yes, I experienced something similar again with Delhi. All unpleasant roads lead to Delhi, as the new proverb you just learned says.

It started in a very concrete way. I arrived at an Airbnb that looked acceptable online, but in reality it wasn't. The fridge was full of mold, the space felt off, and it was immediately clear I couldn't stay there. So I canceled it on the spot.

That decision sounds simple, but it sets off a chain reaction.

Now you're outside, tired, slightly dehydrated, with no stable place to land. You start searching again. Booking quickly, because you just want the situation to end. You arrive at the next place – and it turns out to be a scam.

At that point, the external problem is no longer the main issue.

You can feel the internal shift. Noise becomes intrusive. The environment feels chaotic. Your tolerance drops. You start reacting faster, thinking less clearly, trying to regain control by making more decisions.

This is sympathetic dominance – heightened vigilance, elevated stress signaling, reduced cognitive flexibility. It's designed for short-term threats, not for prolonged uncertainty in a foreign environment.

The natural response is to push harder. Fix everything. Find the best option. Solve the situation completely.

That response backfires.

Because every additional decision adds load to a system that is already saturated.

What actually worked was narrowing the problem.

Find one place that is acceptable. Not ideal, not optimized – just stable enough to stop the search. Once that decision is made, stop looking. No comparisons, no upgrades, no second-guessing.

Then reduce input.

Sit down. Eat something simple. Limit exposure to noise and unnecessary stimulation. Give the system fewer things to process.

I took magnesium. It helps – through effects on neuromuscular tone and excitatory signaling – but it's not the main lever here. If anything, it just supports what the environment allows.

The real shift comes from removing pressure.

No constant switching between options. No scrolling through alternatives. No attempt to fix every variable at once.

Because in that state, the problem is not the hotel, or the city, or even the situation itself. It's the accumulation of unresolved inputs – fatigue, uncertainty, sensory overload, and continuous decision-making.

Once that input is reduced, the system starts to settle.

5. Sleep Disruption

Sleep doesn't usually collapse in one dramatic way.

It drifts apart, gradually exhausting you and taking away everything you need.

Timing shifts, light exposure is off, you eat at the wrong moment, the room is slightly too bright, slightly too warm, slightly too loud. None of it is extreme, but together it's enough to prevent deep recovery.

I experienced this in an extreme form on one of my flights, which I will probably never forget. I was supposed to fly at around 2 a.m., sleep through the flight, and arrive with at least some alignment. Instead, the plane was delayed, and we ended up boarding around 6 a.m.

That completely breaks the structure.

You stay awake through the night waiting at the airport. Your biological night passes without sleep. Then, just when your system is expecting light and activity, you're suddenly on a plane trying to rest in what is now your biological morning. If you start randomly eating, exposing yourself to bright light, dozing inconsistently – you reinforce the misalignment and make the next 24–48 hours worse.

At that point, you don't control the environment. You control which signals you follow.

Light is the dominant one.

I kept blue light blockers on consistently. That wasn't about comfort, it was about preventing the brain from fully switching into a daytime state. Short-wavelength light is a primary circadian input, and reducing it helps preserve the internal sense of night even when the external timing is off.

Food is the second signal.

I didn't eat during what was still my biological night, because I know that feeding acts as a strong peripheral cue for metabolic tissues, and eating at the wrong time can shift internal timing even if you try to control light. By not eating, I avoided reinforcing the disruption.

Then I slept when I could, without trying to impose a perfect schedule on a situation that clearly didn't allow it.

That was enough.

People often try to fix this with compounds – melatonin, sedatives, anything that promises sleep on demand. Sometimes they help, but only if the underlying signals are not working against them.

If the environment is bright, noisy, and temporally inconsistent (which is exactly what happened in my case), adding substances becomes unreliable. You're trying to override a system that is still receiving conflicting inputs, and while the best-case scenario might be that you fall asleep, you could wake up feeling confused, exhausted, and in a much worse mood than if you hadn't used any sleep aid at all.

So the order holds.

Reduce light.

Control the immediate environment as much as possible.

Keep food aligned with the phase you want to preserve.

Then sleep when the opportunity appears.

Everything else sits on top of that.

6. Chronic Travel Fatigue

This one doesn't arrive all at once.

There's no clear moment where something breaks, no obvious trigger you can point to. It builds gradually, almost quietly.

You move, adapt, adjust, repeat. New place, new bed, new rhythm. Again and again.

At the beginning, that variability feels stimulating. Everything is new, your attention is sharp, your system is engaged. Then it levels out. And after some time, it starts to cost you.

You notice it in small ways first. Less motivation. Less patience. Less enjoyment from things that would normally feel interesting – a persistent, low-grade depletion that doesn't fully resolve.

That's what it looks like when recovery never quite catches up.

Each individual stressor is manageable. A shift in sleep timing, a different environment, unfamiliar food, low-level social uncertainty. None of these are enough to cause a problem on their own. But they don't happen in isolation, and they don't stop. The system keeps adapting without a phase where it can fully return to baseline.

Over time, that matters.

The instinctive response is to push harder – to refine routines, add more structure, compensate for the drift. That usually misses the point.

Correction is simple, but not always easy to accept.

You reduce change.

Stay longer in one place when you can – personally, I prefer to stay in some accommodations for weeks rather than a few days. Then, give your system enough time to stabilize instead of constantly resetting it. Keep certain elements identical across locations – morning habits, movement patterns, basic food structure. Not because they are perfect, but because they are predictable.

Remove decisions where possible. Every new environment already introduces uncertainty; there is no advantage in adding more on top of it.

There's a phase where exploration works in your favor, where novelty sharpens perception and keeps you engaged. And there's a point where the same novelty becomes friction, where the system starts to lose efficiency instead of gaining it.

Don't push. Inhale. Exhale. Relax. And take your time.

Conclusion

So, I guess that's about it.

If you've made it this far and aren't tired, frustrated, or bored, congratulations. And if you've found even one travel hack, recommendation, piece of advice, or piece of

information here that caught your interest – or even inspired you to make a change you're currently enjoying in your life – I consider that a success.

There are very few people who care about their health. And there are even fewer people who care about their health and enjoy their health routines as a hobby. And the very fewest are those who keep all of this in mind even in changing environments, while staying in airport terminals and hotel rooms. Traveling is a challenge for our health hobbies, and this book was meant to be an inspiration on how to handle this challenge more easily – and, above all, with joy.

And if there is one thing to take from all of this, it's not a specific protocol, routine, or optimization – but a way of thinking. A way of moving through the world without completely disconnecting from your body, even when your environment is constantly changing.

Writing this book went very smoothly, and I enjoyed every line, but as is often the case, it wasn't an easy task. While AI made the work easier in many ways, it also made it more difficult in others, forcing me to rewrite things in my own words and engage in a variety of other activities. The content didn't just come up on its own, nor did the AI come up with it – the book isn't some random guide, but a collection of my personal experiences and insights that I had the privilege of testing and verifying while living my life as a digital nomad and traveler.

It took a lot of effort to figure out what to include in the book, what to leave out, and where to draw on my own knowledge and weave it into the text so that the whole thing makes sense and people holding this book in their hands don't think something like, "Oh my God, what kind of idiot wrote this!?"

So if you don't think that, I consider it a success as well. I hope that the energy I put into this has resulted in something you'll appreciate.

And maybe more importantly, I hope this book didn't just give you answers – but helped you ask better questions about how you move, how you travel, and how you take care of yourself when things are not ideal.

And so, with these slightly sentimental words, I bid you farewell. And since perfectionism has gotten me nowhere but into chronic stress, I want to give you one more little travel tip with which I began my book – don't be afraid to not be perfect. Don't be afraid of jet lag, poor sleep, food that isn't quite perfect, or a body that isn't absolutely amazing, strong, and fully regenerated in every possible situation. Most importantly – stay healthy and at ease at all costs.

Even though our journeys through the world will never be perfect, may they be well-managed, optimized as much as possible, and well-integrated. And more than that – may they still be enjoyable.

I wish you the best of luck on your travels – and in your everyday life as well.

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