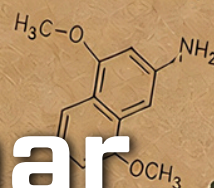
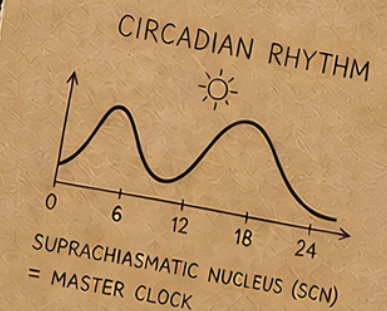
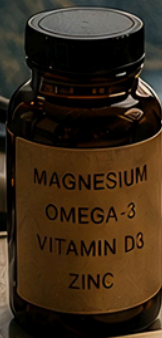
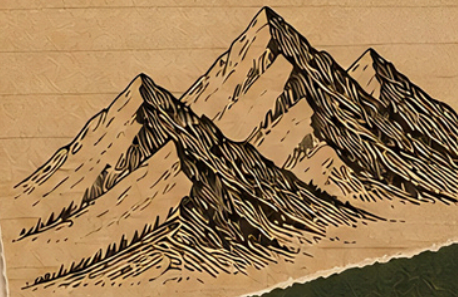


TRAVELING BIOHACKER EXCERPT

A PRACTICAL GUIDE TO
STAYING FUNCTIONAL ON
THE MOVE

Michal Kodnar



Traveling Biohacker (Excerpt)

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.

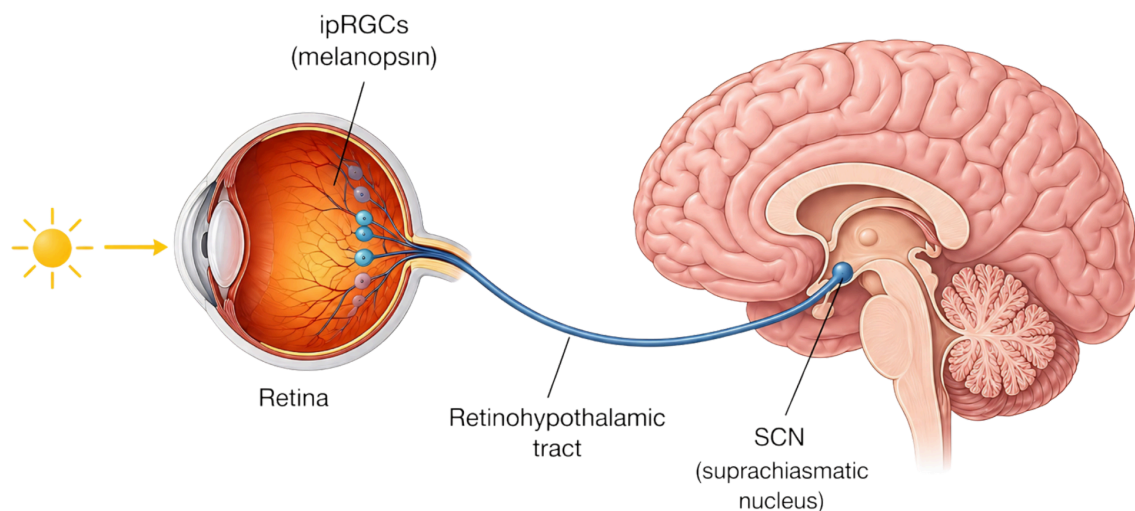
When light enters the eye, it does not simply generate visual perception. Part of that signal is routed into a separate neural pathway dedicated to biological timekeeping. Light information from the retina travels through a direct connection known as the **retinohypothalamic tract**, which carries timing 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.

But, this is just one piece of the puzzle.

If you want the full system for staying sharp, energized, and biologically aligned while traveling, the complete guide is in The Traveling Biohacker 😊