The Human Energy Switchboard

A Symptom-Based Personal Guide to Fatigue, Loss of Drive, and the Prevention of Depression

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Preamble

For a long time I have imagined the human body through a lens that is half mechanical and half science fiction. Not as a cold machine, but as an astonishing system of interrelated elements, coupled to different degrees, working in remarkable coordination to sustain the body's built-in desire-to-live. When that system functions well, life does not merely continue. It comes online, recovers, acts, adapts, repairs, and keeps moving toward engagement with the world. When part of that system begins to fail, the first signal may not be sadness or philosophical despair. It may be fatigue.

This paper grew from that intuition. I wanted to look at fatigue not as a vague complaint, but as a warning light - a sign that somewhere in the body's energy network a gate is no longer opening clearly, a channel is no longer delivering reliably, or a local site of energy handling is no longer supporting life in the way it should. My suspicion has long been that if such physical malfunctions persist, the mind begins to carry their consequences. Loss of drive, apathy, flattening, and eventually depression may follow.

Medical practice often has to begin where suffering is most visible: mood, thought, behavior, diagnosis, neurotransmitters, treatment categories. That approach is necessary and often lifesaving. But it may also begin too late in the sequence. The approach taken here begins one layer earlier. It asks whether, in a meaningful class of cases, the route into depression runs first through disturbance in energy distribution, then through fatigue, and only afterward into the mental and emotional language by which the person experiences the state.

That is why I visualize this guide as a physical switchboard. Each symptom is a clue. Each clue may point to a stuck gate, a weak channel, a threshold problem, or a lagging local energy site. Each suspected malfunction suggests a Point of Physical Engagement - a place where one can try to reopen flow, restore activation, improve delivery, stabilize recovery, or interrupt decline before a physical deviation hardens into a mental collapse categorized as depression.

What follows is not a finished doctrine and not a replacement for medical or psychiatric care. It is a personal guide to a possibility: that by learning to read fatigue earlier, more mechanically, and more

locally, we may sometimes prevent a deeper descent. The governing hope of this paper is simple - to help people re-enter life before the body's loss of power becomes the mind's loss of hope.

Governing chain: Energy Distribution -> Fatigue -> Depression

Introduction

With that personal frame in mind, this paper develops one focused working chain: disturbance in energy distribution comes first, fatigue emerges next, and depression may follow as the mental and behavioral expression of a body-brain system that can no longer reliably support life from within.

This is not a universal theory of depression. It is a focused exploration of depressive states in which fatigue, apathy, psychomotor slowing, low initiative, poor recovery, bodily heaviness, and loss of drive are prominent. In those states, fatigue is not a side symptom. It may be the key warning signal and the most practical point of early intervention.

The paper continues the earlier Human Energy Flow framework, which treated the body as a distributed energy-access system rather than as a simple fuel container. That framework emphasized access, timing, delivery, control, and recovery rather than gross energy quantity alone. The present paper sharpens that view around one proposed chain: Energy Distribution -> Fatigue -> Depression. [1]

That possibility matters because it changes where we look and how we intervene. If fatigue is the bridge between bodily malfunction and depressive state, then early, targeted response to fatigue may do more than improve comfort. It may interrupt the slide into depression.

1. Reverse the Order of Explanation

The usual story about depression starts high in the hierarchy. Thoughts darken, mood falls, motivation decreases, and the body follows with slowed movement, poor sleep, low appetite, and exhaustion. That description is often true, but it may also be incomplete.

The body may lead before the mind explains.

A person may first lose the ability to wake fully, recover after normal demand, tolerate standing, remain clear after meals, or sustain effort through the day. What emerges at first may look like ordinary tiredness, but it is not ordinary tiredness. It is a change in the organism’s ability to bring usable energy online. When that state persists, the mind does what it always does: it interprets. It looks for meaning, blame, loss, threat, and explanation. The resulting mental experience is real, but the origin of the state may already be physiological.

This paper therefore proposes a different order of inquiry. When fatigue and loss of drive are prominent, one should ask not only, “What does the person think and feel?” but also, “What part of the energy system may be failing?” That question does not reduce depression to physiology. It broadens the frame.

2. The Core Chain: Energy Distribution -> Fatigue -> Depression

The body does not live on stored energy alone. It lives on energy that can be absorbed, buffered, transported, admitted into cells, converted into ATP, allocated to the tissues under demand, and

restored after use. Every stage in that sequence contains regulatory points. In this paper, those points are called **gates**.

A gate is any local structure or process that regulates whether energy moves forward clearly, slowly, unevenly, or not at all. Gates may sit in the intestine, the liver, the microcirculation, the cell membrane, the mitochondria, the endocrine system, or the neural supervisory system. They do not all behave like physical barriers. Some are biochemical, some vascular, some neural, some hormonal, and some are local response thresholds.

The main **channels** of distribution are easier to picture. The blood circulatory system is the dominant rapid-delivery network. The gut-to-liver portal route is a critical buffering and redistribution path. The lymphatic system plays a quieter but still important supporting role in returning interstitial fluid to the circulation and in transporting absorbed dietary fats through intestinal lacteals. [5] [9]

Fatigue is the felt signature that something along this chain has gone wrong. It is not merely the wish to rest. It is reduced ability to mobilize usable energy for action, thought, posture, effort, or recovery.

Depression, in the present framework, begins to appear when fatigue is no longer occasional but structural. Once the organism repeatedly fails to mobilize, recover, and restore itself, initiative becomes risky. Effort loses its promise. Reward loses force. The future no longer feels inviting because the body no longer trusts its own capacity to fund engagement. Depression then emerges not only as sadness, but as a state of underpowered life.

This chain does not say that all fatigue becomes depression or that all depression begins in fatigue. It says something narrower and more practical: in a substantial class of cases, fatigue is the bridge through which energy-system dysfunction becomes depressive state.

3. Neurotransmitters Matter, but They May Not Always Be First

A paper like this should be careful here. It should not pretend that neurotransmitters are unimportant. They are clinically important, mechanistically important, and often therapeutically important.

But neurotransmitter imbalance may not always be the first failing layer.

Synapses are energy-demanding sites. Neurotransmitter synthesis, vesicle loading, vesicle release, ion pumping, receptor resetting, and signal recovery all require continuous metabolic support. Mitochondria positioned near synapses help provide ATP and regulate calcium handling, both of which influence neurotransmission. Astrocytes are also deeply involved in brain energy regulation and in the metabolic support of neurons. [14] [15]

That changes the conceptual picture. The neurotransmitter story can remain valid while moving one level down. Disturbed neurotransmission may sometimes be a downstream expression of disturbed energy support rather than the solitary initiating cause. If synaptic function is energy-expensive and energy delivery is unstable, then transmitter balance may become unstable as well.

This is one of the most important possibilities in the paper. It does not reject brain chemistry. It places brain chemistry inside a larger energy framework.

The key question then becomes: when we see apathy, loss of drive, flattening, slowed initiation, and depression, are we always looking at a primary neurotransmitter disorder? Or are we sometimes looking at neurotransmitter imbalance that has been pushed off balance by upstream failures in energy distribution, gating, and local cellular conversion?

This paper argues that the second possibility deserves much more attention than it usually receives.

4. The Body-Side Architecture in Brief

A fully technical version of this model could become dense very quickly. The main essay should stay broad enough to remain readable. Still, the structure matters.

The **intake and buffering layer** begins in the digestive tract. The small intestine absorbs nutrients; absorbed sugars and amino acids enter the blood; the liver stores, processes, and redistributes them; absorbed fats also move through intestinal lymphatics before entering wider circulation. The pancreas and biliary system influence how well digestion and nutrient handling proceed. [5] [9]

The **delivery layer** is dominated by the heart, lungs, blood, vessels, and microcirculation. This is the fast-distribution system. It decides whether oxygen and substrate arrive where and when they are needed.

The **endocrine gating layer** sets pace and access. Thyroid hormone regulates metabolic speed and thermogenic tone. Insulin helps govern whether glucose enters tissues appropriately. Adrenal function affects blood pressure, salt-water balance, and the organism's ability to tolerate stress and maintain perfusion. Official clinical sources list fatigue, depression, or both among the common symptom patterns in hypothyroidism and adrenal insufficiency. [6] [7]

The **local conversion layer** sits inside cells, especially in mitochondria. Mitochondria are not separate cells; they are organelles within cells, and they are central to ATP production. When delivery is adequate but conversion is slow, narrow, or slow to recover, fatigue can become severe even in the absence of obvious structural disease. [14]

The **neural supervisory layer** includes arousal systems, hypothalamic timing systems, autonomic control, reward and action-initiation circuits, and higher networks that determine when and how the organism comes online. This layer does not create energy from nothing. It governs access, timing, prioritization, and effort.

The argument of this paper depends on one simple fact about this architecture: a person may have energy in the body in the gross sense, and still fail to function well because the relevant gates do not open clearly, quickly, locally, or in synchrony.

5. Thresholds, Reserve, and the Sudden Return of Power

This part of the argument arose from a lived observation: sometimes fatigue vanishes almost instantly. A person can feel flat, dim, and underpowered, and then—within minutes—become clear, alert, warm, and fully functional.

That kind of change is hard to explain if all fatigue is imagined as a slow, linear process. It is easier to explain if at least some gates operate with **threshold-like behavior**.

The threshold idea should be used carefully. The paper is not proposing one universal fatigue threshold or one universal depression threshold. It is proposing something more modest and more plausible: some gates of the energy system appear to have operating thresholds, reserve margins, or switch-like behavior.

The clearest candidate is the **arousal gate**. Sleep-wake regulation is not purely gradual. Sleep inertia is a recognized post-waking transitional state of lowered alertness, and research on sleep inertia and wake-promoting countermeasures suggests that state can shift materially with caffeine, light, timing, and other factors. [10]

A second candidate is the **perfusion gate**. Orthostatic intolerance and related states can worsen quickly in the upright position and improve when the person lies down, contracts large muscle groups, hydrates, or otherwise improves delivery. NINDS notes that POTS may include dizziness, fainting, palpitations, exhaustion, and brain fog, especially when upright. [4]

A third candidate is the **substrate gate**. Official endocrine references describe symptom thresholds in hypoglycemia, with adrenergic symptoms often appearing below about 55 mg/dL and neuroglycopenic symptoms below about 48–50 mg/dL. [11] That is a genuine threshold example inside the energy system.

A fourth candidate is **local reserve**. Cells do not merely produce enough energy for the present instant; they also maintain reserve capacity. Bioenergetic literature describes spare respiratory capacity as a reserve that can be called on when demand rises. [16] That idea fits a familiar experience: some people do not fail gradually. They appear adequate until demand exceeds reserve, and then they collapse quickly.

This threshold perspective matters because it supports the practical idea of **quick recovery interventions**. If some fatigue states are threshold-governed, then simple actions may sometimes return the person to a more functional side of the threshold before a deeper mental collapse consolidates.

6. Quick Recovery from Fatigue: Points of Physical Engagement

This is one of the most practical parts of the paper.

In this paper, these symptom-triggered actions are treated as Points of Physical Engagement: practical ways of acting on the body's energy switchboard before a physical malfunction consolidates into a depressive state.

The aim here is not to promise an instant cure for depression. The aim is more modest and, in some ways, more important: identify recognizable fatigue states early, apply simple corrective actions aimed at the most likely failing gate, and try to restore enough usable energy to prevent the fatigue state from deepening into apathy, collapse, or depressive interpretation.

If the chain is **Energy Distribution -> Fatigue -> Depression**, then there is a powerful preventive possibility: interrupt the fatigue phase early and often.

6.1 The morning dead-state: an arousal-gate problem

Some people wake not merely sleepy but inert. They are technically awake but not online. The body feels unavailable, initiative is absent, thinking is dim, and mood can turn dark before the day has even begun.

In a threshold model, this may reflect a poorly opened arousal gate. The fast tools here are simple: immediate light exposure, hydration, brief movement, getting out of bed without repeated snoozing, and—when appropriate and tolerated—timed caffeine. Bright light and wake-promoting routines fit the physiology better than waiting passively for life to return. [3] [10]

If this pattern is frequent, circadian misalignment, sleep deficiency, sleep apnea, medication effects, or mood disorder itself may be contributing. The quick tools are useful, but repeated morning dead-state patterns deserve evaluation.

6.2 The upright crash: a perfusion-gate problem

Another person feels relatively normal lying down or sitting, but worse when standing still or remaining upright. Brain fog, heaviness, low drive, dizziness, palpitations, and sudden fatigue may appear together.

Here the likely issue is not abstract motivation. It is delivery. The body is struggling to maintain adequate return and perfusion when upright.

Fast responses can be surprisingly practical: sit or lie down, elevate the legs if possible, contract the calves and gluteal muscles, walk gently rather than standing frozen, hydrate, and in some people use compression garments as a preventive tool. [4] [17]

This pattern matters for the paper because it shows how quickly a “depressed” state can sometimes change when blood delivery changes. If a person’s mental darkness lifts when delivery improves, that does not prove the whole case—but it strongly supports the need to look below mood description.

6.3 The desk-flattening or inactivity crash: a movement-activation problem

Some fatigue states are produced or intensified by immobility itself. Long sitting periods can produce flattening, low alertness, fog, and low mood.

Research on interrupting prolonged sitting suggests that brief walking breaks can reduce fatigue and help preserve cerebral blood flow. [12] [18] In practical terms, a person whose energy and mood deteriorate with prolonged sitting may benefit from an extremely simple rule: do not let immobility become the default state when a movement-gate intervention is available.

The fast tool here is not heroic exercise. It is short bouts of walking, stepping, stair use, calf pumping, or gentle whole-body activation. The point is to reopen distribution and local activation before the flat state deepens.

6.4 The meal-linked crash: a substrate-gate problem

Some people reliably crash when meals are delayed, when meals are heavily refined, or in the post-meal period after certain food patterns. Symptoms may include shakiness, irritability, fog, weakness, sudden fatigue, low drive, or a strange emotional collapse that improves after eating.

This cluster should be handled with caution because not every crash is hypoglycemia. Still, the substrate gate is real. If symptoms are clearly meal-linked, the immediate task is to restore stability: eat, hydrate, and then review timing and composition rather than treating the episode as purely emotional. Recurrent patterns deserve metabolic evaluation, especially in people with diabetes, insulin resistance, or suspected reactive hypoglycemia. [11]

For the broader paper, the significance is conceptual. The mind may tell a despairing story while the body's immediate problem is simply that fuel delivery and entry have become unstable.

6.5 The push-through crash: a recovery-gate problem

Some people can perform in the moment and then pay later. They are not merely tired after exertion; they are physiologically punished by it. Fatigue, fog, malaise, low mood, and loss of function appear hours later or the next day.

This is not a system asking for motivation. It is a system warning that recovery gates are failing.

In such cases, the quick intervention is not added effort but intelligent retreat: stop the push-through, reduce stimulation, respect the signal, and use pacing. CDC guidance for ME/CFS describes post-exertional malaise as symptom worsening after even minor activity and recommends activity management to avoid flare-ups. [8]

This is especially important for the prevention argument in the paper. Some depressive states may not be prevented by "trying harder" but by preventing repeated physiological penalties that teach the organism not to engage.

6.6 Where massage, manual work, and diet fit

The original impulse behind this paper included targeted exercise, massage, and diet. Those still belong here, but they should be placed honestly.

Exercise has the strongest evidence base of the three as a depression intervention, including a 2024 BMJ systematic review and network meta-analysis that found exercise effective for depression, with walking or jogging, yoga, and strength training among the more effective modalities. [13] Within the present model, exercise is not just a mood tool. It is also a way to improve delivery, local production, autonomic tone, and confidence in recovery.

Diet belongs in the model most clearly when it stabilizes energy handling rather than being treated as a vague lifestyle virtue. A diet that reduces meal-linked crashes, improves protein adequacy, moderates extreme glycemic swings, and supports sleep and inflammation control fits directly into the energy-distribution framework. Broader evidence for diet and depression is promising but still mixed enough to justify caution. [19] [20]

Massage and manual techniques fit best as adjunctive tools, not as primary claims. They may help some people through bodily relaxation, comfort, local circulation, or reduced physical shutdown, but official NCCIH sources describe the evidence base for depression-specific benefit as limited or modest, depending on context. [21] They are worth exploring where they clearly improve bodily state, but they should not be oversold.

7. What Changes in Prevention and Treatment

If this paper is right even part of the time, then prevention and treatment should change in tone and sequence.

First, fatigue should be treated as a localization clue, not merely as a complaint. The question becomes: what kind of fatigue is this? Morning inertness? Upright collapse? Meal-linked crash? Post-exertional worsening? Diffuse low-power slowing with cold intolerance? Exertional breathless fatigue with poor concentration? Each pattern points to different gates.

Second, treatment should become **whole-system but still local**. A holistic approach does not mean a vague approach. It means addressing the person as a whole while still trying to identify where the chain is most likely failing.

Third, quick tools should be taken seriously. If a fatigue state can be interrupted by light, posture change, hydration, food, pacing, movement, or other low-cost means, then these are not trivial self-help gestures. They are potential protective actions taken at the fatigue stage before the person falls further into a depressive state.

Fourth, standard psychiatric care remains in the picture. This paper does not reject therapy, medication, or established depression care. It argues that these should sometimes be joined to, and informed by, better physiological localization.

The more precise ambition is this: not “treat depression somehow,” but “identify the failing gate, reduce the fatigue produced by it, and thereby reduce the chance that fatigue hardens into depression.”

8. Boundaries, Red Flags, and Honest Limits

This paper is an idea-exploration, not a finished doctrine.

Not every depression belongs inside this framework to the same degree. Bipolar depression, psychotic depression, grief, trauma-related states, substance effects, primary psychiatric syndromes, and severe melancholic conditions may require additional frameworks or completely different priorities.

The quick-recovery idea also has limits. A fast intervention that helps a person come online after poor sleep, upright strain, or a delayed meal should not be mistaken for proof that all depression is reversible by simple actions. The claim is narrower: some fatigue states are physiologically structured, some appear threshold-sensitive, and interrupting them early may have preventive value.

Certain symptoms should immediately override any exploratory model: suicidal intent, psychosis, chest pain, fainting, severe shortness of breath, rapidly progressive weakness, major unintentional weight loss, GI bleeding, severe dehydration, or any abrupt and dangerous change in function. These require professional care.

The threshold idea also needs humility. It is credible to speak of gate-specific thresholds, reserve margins, and switch-like transitions. It is not credible to claim one universal threshold for depression across all people.

Conclusion

The proposal of this paper is simple enough to remember and strong enough to matter:

Energy Distribution -> Fatigue -> Depression

It asks us to take fatigue much more seriously than we usually do. Fatigue may be the first felt sign that the organism is losing access to usable energy. Once that state persists, drive weakens, initiative falls, reward loses force, and depression becomes more likely.

This perspective does not discard neurotransmitters. It re-situates them. Brain chemistry remains important, but some of its disturbance may be downstream of upstream dysfunction in energy delivery, gating, and local conversion.

It also changes the practical question. Instead of waiting until a depressive state is fully organized, one can intervene earlier at the fatigue stage. If the right gate is identified, even simple actions may sometimes restore enough energy access to prevent further slide.

Seen this way, the paper is not only an exploration of depression. It is also a guide for reading fatigue as the body's early warning light and for responding before loss of power becomes loss of hope.

That is the heart of the paper. Not a denial of psychology. Not a rejection of psychiatry. A wider and more physically grounded way of thinking about how depression may begin, and how some cases might be interrupted earlier, more practically, and more humanely.

Appendix A. Switchboard Companion: Gates, Channels, and Points of Physical Engagement

The main essay stays broad and exploratory. This appendix is the switchboard companion: a compact map of where the likely gates are, how fatigue patterns help localize them, and where the main Points of Physical Engagement are. Use it as a guide for structured thinking, not as a substitute for diagnosis.

A1. Major channels, organs, and gates

Gate or channel	Main location	Main job	Typical clue when strained	Examples of first influence
Arousal gate	Hypothalamic timing systems; brainstem arousal networks; sleep-wake control	Bring the system online and coordinate wakefulness	Morning dead-state, long sleep inertia, delayed clarity	Light exposure, hydration, brief movement, sleep timing repair, caffeine when appropriate
Perfusion gate	Heart, vessels, autonomic control, venous return, microcirculation	Maintain delivery to brain and muscle, especially upright	Worse standing still, better lying down, dizziness, palpitations, upright brain fog	Recline, leg muscle pumping, walking, hydration, compression, orthostatic evaluation
Oxygen-delivery channel	Lungs, hemoglobin, red blood cells, circulation	Carry oxygen for oxidative metabolism	Exertional fatigue, breathlessness, pallor, headaches, poor concentration	Check anemia, pulmonary or cardiac causes; treat underlying oxygen-delivery problem
Absorption gate	Small intestine, pancreas, bile, gut wall	Digest and absorb nutrients	Bloating, nausea, appetite loss, meal intolerance, weight loss, bowel disturbance	Dietary review, GI workup, meal structure, treat malabsorption or inflammation
Portal-liver buffer	Portal blood flow, liver	Store, process, buffer, and redistribute absorbed nutrients	Meal-linked instability, poor endurance, low resilience to fasting or overfeeding	Meal regularity, metabolic workup, liver and GI review when indicated
Glucose-entry gate	Insulin signaling, cell transport, liver-muscle-adipose regulation	Move glucose out of blood and into tissue appropriately	Post-meal crash, hunger-linked irritability, shakiness, reactive fog	Meal composition, regular timing, diabetes or insulin-resistance evaluation
Endocrine pace gate	Thyroid and adrenal systems	Set metabolic speed, thermogenesis, salt-water balance, stress tolerance	Cold intolerance, slowing, constipation, chronic fatigue, low blood pressure, low drive	Formal endocrine testing and treatment when indicated
Local conversion gate	Cellular mitochondria	Convert delivered substrate into usable ATP	Disproportionate fatigue, poor recovery, effort cost too high, delayed rebound	Graded activity only when tolerated; pacing if post-exertional worsening; treat upstream causes
Synaptic energy gate	Synapses, astrocyte-neuron metabolic coupling	Sustain signaling, release, reuptake, and network stability	Cognitive fog, slowed initiation, flattened response, reduced reward pull	Support upstream sleep, perfusion, metabolic stability, psychiatric treatment as needed
Lymphatic support channel	Intestinal lacteals, peripheral lymphatics, thoracic duct	Return interstitial fluid; transport absorbed dietary fats; support tissue fluid balance	Heaviness, swelling tendency, poor tolerance of immobility, GI fat-handling issues	Walking, muscle contraction, manual support in selected cases, treat underlying fluid or inflammatory issues

A2. Symptom signatures and likely gate localization

Symptom pattern	Most likely gate(s) to consider first	Why it matters for the fatigue -> depression chain	Useful next step
Fatigue plus morning inertia	Arousal gate, sleep quality, circadian timing	The person may start each day below functional threshold; low drive follows quickly	Review sleep schedule, light exposure, snoring or apnea risk, medication timing
Fatigue worse upright, better when lying down	Perfusion gate / orthostatic intolerance	Delivery failure can look like anxiety, apathy, or fog	Check orthostatic vitals; try posture and hydration strategies; evaluate autonomic causes
Fatigue plus cold intolerance and slowing	Thyroid pace gate	Under-speed physiology can feel like emotional shutdown	Check thyroid function and related symptoms
Fatigue plus pallor, breathlessness, headache, poor concentration	Oxygen-delivery channel / anemia	Low oxygen delivery lowers power before mood fully changes	CBC, iron studies, B12 and related evaluation as indicated
Fatigue plus abdominal symptoms or meal intolerance	Absorption gate, liver buffer, glucose-entry gate	Gut underpowering can lower both bodily and mental resilience	Review meals, weight change, bowel symptoms, glucose pattern, GI history
Fatigue plus delayed crash after effort	Recovery gate, local conversion gate	Repeated physiological penalties teach withdrawal and reduce drive	Use pacing; assess for post-exertional symptom patterns
Fatigue plus shakiness or abrupt hunger-linked fog	Substrate gate / glucose instability	Rapid underfueling can produce instant mental darkness	Immediate food if appropriate; recurrent episodes need evaluation
Fatigue plus diffuse low drive but clear improvement with movement	Movement-activation / perfusion gate	Inactivity may be locking the system below threshold	Introduce frequent light activity breaks rather than waiting for motivation
Fatigue plus low interest after chronic poor sleep	Arousal gate and synaptic energy gate	Prolonged sleep deficiency affects mood, motivation, decision-making, and coping	Prioritize sleep repair before assuming purely psychological origin

A3. Rapid Fatigue Recovery tools: symptom-triggered preventive actions

Pattern to recognize	Simple actions	Why these actions make sense in this model	Important cautions
Morning dead-state	Open curtains or go outside; hydrate; brief walk; avoid repeated snooze cycles; caffeine if appropriate	Tries to push the arousal gate above threshold	Persistent morning failure warrants sleep and circadian evaluation
Upright crash	Sit or lie down; raise legs if possible; contract calves and glutes; walk gently; hydrate	Improves venous return and delivery	Fainting, chest pain, or severe symptoms need medical care
Desk-flattening	Every 30-60 minutes, take 2-5 minutes of walking or muscular activation	Reopens movement and perfusion gates; helps oppose sitting-related fatigue	If activity predictably causes next-day collapse, use pacing instead

Meal-linked crash	Eat; hydrate; then stabilize future meals with protein/fiber and avoid long gaps if they trigger symptoms	Addresses the substrate gate before the mental state deepens	Recurrent episodes, especially in diabetes, need formal assessment
Push-through crash	Stop escalation; rest; reduce stimulation; use pacing diary or energy-envelope tracking	Protects a failing recovery gate rather than punishing it further	Not every patient should "exercise through" fatigue
Cold, heavy, slowed state	Gentle movement, warmth, food if delayed, review thyroid-like symptom pattern	Can distinguish simple under-activation from endocrine slowing	Help distinguish simple under-activation from endocrine slowing
Diffuse shutdown with bodily tension	Gentle stretching, short walk, breath settling, manual or massage adjunct if clearly helpful	May reduce physical shutdown and support re-entry into movement	Massage evidence for depression itself is limited; use as adjunct, not doctrine

A4. How to correlate intervention with likely malfunction

1. Name the fatigue pattern before naming the depression pattern.
2. Ask which gate is most likely failing.
3. Use the simplest plausible action first if it is safe.
4. If the same pattern repeats, escalate from self-observation to formal evaluation.
5. Use psychiatric care and physiological care together when both are needed.

The appendix does not replace the main essay. It grounds it. It suggests that prevention of depression may partly begin with learning to notice fatigue patterns early and treating them as clues to specific gates of failure rather than as vague signs of low mood.

Appendix B. Selected Sources and Pointers for Follow-Up

[1] Neymotin, L. A Probabilistic Risk Assessment Framework for Human Energy Flow Systems. Foundation paper for this continuation.

[2] National Institute of Mental Health. Depression. https://www.nimh.nih.gov/health/publications/depression

[3] National Heart, Lung, and Blood Institute. How Sleep Affects Your Health; Circadian Rhythm Disorders. https://www.nhlbi.nih.gov/health/sleep-deprivation/health-effects ; https://www.nhlbi.nih.gov/health/circadian-rhythm-disorders/symptoms

[4] National Institute of Neurological Disorders and Stroke. Postural Tachycardia Syndrome (POTS). https://www.ninds.nih.gov/health-information/disorders/postural-tachycardia-syndrome-pots

[5] National Institute of Diabetes and Digestive and Kidney Diseases. Your Digestive System & How It Works. https://www.niddk.nih.gov/health-information/digestive-diseases/digestive-system-how-it-works

[6] National Institute of Diabetes and Digestive and Kidney Diseases. Hypothyroidism (Underactive Thyroid). https://www.niddk.nih.gov/health-information/endocrine-diseases/hypothyroidism

[7] National Institute of Diabetes and Digestive and Kidney Diseases. Adrenal Insufficiency & Addison's Disease. https://www.niddk.nih.gov/health-information/endocrine-diseases/adrenal-insufficiency-addisons-disease/symptoms-causes

[8] Centers for Disease Control and Prevention. Manage Myalgic Encephalomyelitis/Chronic Fatigue Syndrome. https://www.cdc.gov/me-cfs/management/index.html

[9] StatPearls / NCBI Bookshelf. Physiology, Lymphatic System. https://www.ncbi.nlm.nih.gov/books/NBK557833/

[10] Hilditch CJ, McHill AW. Sleep inertia: current insights. https://pmc.ncbi.nlm.nih.gov/articles/PMC6710480/

[11] Endotext / NCBI Bookshelf. Hypoglycemia. https://www.ncbi.nlm.nih.gov/books/NBK279137/

[12] Bergouignan A, et al. Effect of frequent interruptions of prolonged sitting on self-perceived energy and mood. https://pmc.ncbi.nlm.nih.gov/articles/PMC5094084/

[13] Noetel M, et al. Effect of exercise for depression: systematic review and network meta-analysis of randomised controlled trials. BMJ 2024. https://pubmed.ncbi.nlm.nih.gov/38355154/

[14] Lee A, et al. Emerging roles of mitochondria in synaptic transmission and neurodegeneration. https://pmc.ncbi.nlm.nih.gov/articles/PMC6178220/

[15] Beard E, et al. Astrocytes as Key Regulators of Brain Energy Metabolism. https://pmc.ncbi.nlm.nih.gov/articles/PMC8787066/

[16] Marchetti P, et al. Mitochondrial spare respiratory capacity: mechanisms, regulation, and significance. https://pubmed.ncbi.nlm.nih.gov/32808332/

[17] Stewart JM. Common Syndromes of Orthostatic Intolerance. https://pmc.ncbi.nlm.nih.gov/articles/PMC3639459/

[18] Carter SE, et al. Regular walking breaks prevent the decline in cerebral blood flow associated with prolonged sitting. https://pubmed.ncbi.nlm.nih.gov/29878870/

[19] Bizzozero-Peroni B, et al. The impact of the Mediterranean diet on alleviating depressive symptoms. https://pubmed.ncbi.nlm.nih.gov/38219230/

[20] National Center for Complementary and Integrative Health. Depression and Complementary Health Approaches. https://www.nccih.nih.gov/health/providers/digest/depression-and-complementary-health-approaches

[21] National Center for Complementary and Integrative Health. Massage Therapy for Health: What the Science Says. https://www.nccih.nih.gov/health/providers/digest/massage-therapy-for-health-science

This manuscript is an exploratory working paper. It is not a substitute for urgent medical or psychiatric care, especially when there is suicidal intent, psychosis, chest pain, fainting, severe shortness of breath, GI bleeding, or rapidly progressive systemic illness.