Commentary

A Novel Exercise Template for Parkinson's Based on Energetic Dynamics

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Parkinson's symptoms often remain variable despite carefully regulated medication schedules and optimized DBS settings to stabilize motor-circuit firing. This suggests that regulating dopamine and circuitry alone cannot account for moment-to-moment movement instability. This manuscript proposes that characteristic muscular rigidity, slowed and hesitant movement, gait impairment, balance difficulties and freezing episodes arise from unstable mitochondrial ATP production that limits the real-time power available to basal ganglia circuits. This energetic-dynamics framework clarifies several clinical puzzles: why handwriting can deteriorate even when gross motor ability is preserved; why many exercise models do not produce the expected results; and why improvements triggered by rhythmic cues can arise and disappear within minutes. It also explains why the tango, with its short bouts of rhythmic movement followed by brief recovery intervals, stands out as unusually effective. Drawing on tango's energetic structure and mitochondrial dynamics, this manuscript proposes a home-based exercise template that may generate transient increases in ATP availability that support smoother movement, reduce chronic fatigue, and enable the slow, energy-dependent maintenance of remaining dopaminergic neurons. This model further predicts that brief meditation sessions may reduce competing cognitive load and preserve energetic resources for motor function. Together with short bouts of chair-based movement, these practices offer a coherent, person-centered strategy for improving daily movement that merits formal testing to confirm their effectiveness.

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Introduction

Parkinson's is the second most common neurodegenerative disease after Alzheimer's and is characterized by motor symptoms that may include muscular rigidity, slowed and hesitant movement, gait impairment, balance difficulties, and resting tremor. Non-motor symptoms such as chronic fatigue, depression, and anxiety also significantly affect quality of life [1]. Although the cause of Parkinson's disease is incompletely understood, mitochondrial dysfunction is widely recognized as a central contributor because it limits the energy available to dopaminergic neurons. These neurons require exceptionally high metabolic support to generate smooth, fluid movement, and when their energy needs are not met, the resulting metabolic stress first impairs motor function and ultimately contributes to progressive dopaminergic neuronal loss. This energetic vulnerability is compounded by neuropathology showing misfolded α-synuclein aggregating into Lewy bodies. These aggregates overwhelm proteostasis and further increase ATP demand in neurons already operating near energetic limits [2].

Medication that either increases dopamine or slows its breakdown remains the foundation of Parkinson's treatment, but many individuals also benefit from complementary procedures such as deep brain stimulation (DBS) or high-intensity focused ultrasound. Although both approaches modulate abnormal neural firing in motor circuits, they do so through different mechanisms and are used in different clinical situations [1]. DBS delivers continuous electrical pulses to specific motor regions, helping to regularize firing patterns and reduce fluctuations in movement control. High-intensity focused ultrasound, by contrast, uses precisely targeted acoustic energy to create a small lesion in an overactive circuit, interrupting pathological signaling that contributes to tremor or rigidity. These procedures can support symptom control when medication alone provides inconsistent relief [3], yet none slow the underlying progression of neuronal loss [1].

Adjacent to these treatments is exercise therapy, which is known to temporarily improve motor function [4]. Yet responses to exercise are inconsistent: some individuals improve dramatically, others minimally, and even responsive individuals may fluctuate from day to day. These variations are typically attributed to changes in dopamine availability and to circuit-level disruptions that increase cognitive load, impairing the signals necessary for fluid movement. However, a closer look at the mechanics of movement in Parkinson's disease suggests a complementary explanation. When dopamine levels are sufficient to initiate movement, the remaining motor difficulties may arise from a more fundamental problem: insufficient moment-to-moment energy availability provided by mitochondria. This energetic

perspective provides a coherent starting point for understanding why motor performance can vary so widely and why certain exercise structures may be more effective than others.

What the tango reveals about energetics

A 2024 Cochrane review comparing 10 exercise modalities across 154 randomized trials was unable to identify a unifying feature, yet it consistently highlighted tango as one of the most effective in improving motor skills [4]. On the surface, the success of dancing the highly complex, stylized Argentine tango seems improbable for the typical senior population that develops Parkinson's. The explanations for this remarkable effect range from rhythmic cueing and partner support to motivation and social engagement, which some have suggested may increase dopamine levels, but the proposal put forth most prominently claims that its complex choreography induces neural retraining. Yet this explanation does not fully account for the dramatic short-term gains in functional movement, such as improvements in standing, walking, turning and sitting, that can be found in just one class [5]. Neural retraining is an energy-intensive process requiring receptor synthesis, dendritic strengthening, and synaptic remodeling — all of which unfold over extended periods of time [6]. Improvements that occur within a single session are more consistent with a rapid increase in available cellular energy [7].

With this understanding, the structure of tango becomes relevant. Classes typically consist of sets of a *tanda* — three to four dances of approximately three minutes each — separated by a brief *cortina*, a one-minute musical interlude that functions as a natural recovery period [8]. This pattern of brief bouts of moderate movement followed by short rests closely matches the conditions known to stimulate mitochondrial activation and biogenesis [2] while avoiding energetic collapse. In this sense, tango may be effective not just because of its complexity, rhythmic cuing, or partner support, but because it delivers an energetically optimized sequence of activation and recovery that increases energy availability in real time.

A new interpretation of symptom variability

Motor dysfunction in Parkinson's is typically framed as the consequence of dopamine deficiency and disrupted circuitry: dopaminergic neurons die, basal ganglia firing patterns become abnormal, and motor programs degrade. Yet many clinical observations are difficult to reconcile with a model that focuses solely on chemical and network dynamics. People can walk poorly one hour and far better the

next; freezing can occur with no warning, triggering abrupt falls; rhythmic cues can produce immediate improvements; and stress or fatigue can worsen movement almost instantly. These fluctuations are hard to explain, especially when adjustments to medication doses and timing that may have worked previously do not always reduce symptoms [1]. However, fluctuations in physical abilities make sense when energetic limitations are present, which can produce motor issues even when medication provides enough dopamine.

Mitochondrial insufficiency offers a coherent explanation for this moment-to-moment variability. ATP is delivered continuously, not unlike energy passing through an electrical current. When demand rises — even during a single muscle contraction — the "voltage" of available ATP dips briefly, triggering the cell's mitochondrial repair and rebuild program: biogenesis (making new mitochondria); mitophagy (removing damaged ones); enzyme upgrades (better efficiency); and stronger membrane potential (needed for stable ATP production) [9]. Mitochondrial dysfunction interferes with this delivery system, making these dips in energy availability deeper and more disruptive. Even with sufficient dopamine to send the signal and optimized DBS settings to reduce network "noise," energetic fluctuations may contribute to variability.

Neurons rely on a continuous supply of ATP to sustain the countless micro-adjustments required for postural integrity and fluid movement. When mitochondrial efficiency declines, as in Parkinson's, these networks become vulnerable to even small drops in ATP [2]. Prolonged or intense exertion may exceed production capacity, making these dips in energy availability deeper and more disruptive. In contrast, short, manageable bouts of movement can transiently increase ATP availability through acute mitochondrial activation [7]. This energetic profile naturally explains why some exercise sessions produce dramatic improvements, why these benefits fade within hours, and why individuals fluctuate so widely in their responsiveness.

Since motor performance depends not only on dopamine to signal motion but also on energetic capacity, the structure and duration of an exercise session become as important as the activity itself. Brief rhythmic bouts followed by short recovery intervals — the pattern seen in tango — provide repeated metabolic stimulation without tipping the system into energetic depletion. These transient windows of improved ATP availability may not only stabilize movement in the moment but also create the conditions necessary for slower neuroplastic processes to occur. In this view, the short-term effect of exercise is not to retrain circuits but simply to power them, while longer-term adaptation may be possible if daily ATP supply is sufficient to support maintenance and gradual strengthening of remaining pathways.

The energetic dynamics framework also suggests a complementary hypothesis. Increasing ATP availability through brief interval exercise is only one means of improving the energetic resources available to motor circuits. Interventions that reduce competing neural demands, including meditation, may represent a second, largely unexplored approach. Although meditation has primarily been studied for its effects on mood, anxiety, and quality of life in Parkinson's disease [10], the present model predicts that reducing competing cognitive and emotional processing could preserve energetic resources for motor function. Whether this translates into measurable improvements in gait, freezing, or postural stability remains an important question for future investigation.

How rhythm cuts through the noise

The basal ganglia — a set of interconnected subcortical nuclei — sit at the center of this dynamic. Their role is not to generate movement but to select, sequence, and scale it, functions that depend on both dopamine and a stable energy supply [11]. Dopamine provides the instruction that allows a movement to begin [1], but the basal ganglia must then orchestrate a continuous stream of micro-adjustments to keep that movement fluid [11]. When mitochondrial output is unstable, this cascade of adjustments becomes noisy or incomplete, producing the characteristic hesitations, freezing episodes, and abrupt fluctuations that people with Parkinson's experience [1].

Crossing a room, for example, requires the basal ganglia to estimate distance, select the muscles needed, determine movement amplitude, and continuously update posture and balance —each step requiring dopamine to cue the action and ATP to power the series of necessary commands [11]. The mechanical work of movement is handled by the mitochondria within muscle fibers, but the basal ganglia must supply the energetic resources needed to generate and coordinate the underlying control signals. External rhythm, whether counting aloud or following a metronome, reduces this computational burden by providing an external timing scaffold [12]. This allows the basal ganglia to synchronize movement to an outside signal rather than generating timing internally, which is especially helpful when internal energy supply is fragile. For many individuals, this shift makes walking easier because it stabilizes the system at the very moment when ATP availability is most limited. This helps explain why soundtracks make exercise easier for almost everyone — and why it can be especially useful for people with Parkinson's.

A novel exercise template for Parkinson's

If the tango's therapeutic mechanism depends on short bouts of rhythmic movement followed by brief recovery but the resulting gains are temporary, then the most effective exercise model is one that can be done daily at home to repeatedly generate small increases in mitochondrial ATP. Because these energetic boosts fade quickly, the intervention must be simple enough to perform consistently. Since balance can be an issue, the exercises should be performed seated for safety and to reduce the energetic "posture tax" — the continuous activation of axial and postural muscles — that standing imposes. Seated intervals minimize this background drain, allowing more of the available energy to support basal ganglia signaling rather than postural maintenance.

A practical starting "dose" might be a 15-minute session comprised of four rhythmic movement intervals, each lasting approximately three minutes, separated by one-minute rests, which would respect mitochondrial limits while still providing meaningful metabolic stimulation [9]. This structure mirrors the energetic architecture that makes tango effective: brief activation, brief recovery, gently repeated several times in succession.

As chair-based intervals, these micro sessions are accessible to people with balance challenges, freezing, fatigue, or limited mobility. They eliminate the logistical barriers that make not just tango but many types of traditional exercise difficult: travel time, instructional availability, fall risk, and the unpredictable fluctuations of Parkinson's symptoms. Chair exercises are also something that can be used throughout disease progression, even in advanced cases, providing an activity anchor that does not lead to a sense of failure.

Because the benefit arises from rhythm and the timing of effort rather than the type of movement, the protocol can be adapted to almost any cultural or personal preference. The goal is to replicate tango's successful formula: manageable bursts of effort followed by recovery, repeated enough times to activate mitochondria without overwhelming capacity. The same three-minute pattern can be paired with hip hop, salsa, disco, country music, or any other genre. It can also be adapted to movement styles that feel more athletic or traditionally masculine, such as boxing-inspired or martial-arts-based sequences. This flexibility allows individuals to choose movements that feel culturally familiar, personally authentic, and physically safe, while still delivering the mitochondrial benefits necessary for symptom improvement. Phone apps could make these variations portable and easily accessible to anyone.

How to test the model

A straightforward way to evaluate this energetic-dynamics model is through a small, pragmatic pilot study. The goal would be to test the structure that tango reveals: short bouts of rhythmic movement separated by brief recovery intervals. A feasible design would enroll individuals with mild to moderate Parkinson's disease and assign them to a home-based protocol consisting of four seated movement intervals of three minutes each, separated by one-minute rests, performed twice daily. Participants would choose their preferred movement style and soundtrack, ensuring cultural and personal relevance while preserving the energetic structure.

Primary outcomes could include changes in gait speed, stride variability, freezing frequency, and patient-reported energy levels, measured both immediately after sessions and across days via a structured checklist to capture fluctuations. Secondary outcomes might include fatigue, mood, and ease of daily activities. Because the proposed mechanism involves acute increases in ATP availability, the study should incorporate same-day assessments to detect rapid changes, along with longer-term follow-up to explore whether repeated energetic windows support gradual improvements in motor stability over weeks and months. This simple design would allow investigators to test whether short, rhythmic, interval-based sessions produce consistent, measurable benefits and whether adherence is feasible in real-world conditions.

Discussion

When exercise is misaligned with a person's energetic capacity, the result is not improvement but exhaustion: fatigue that lasts the rest of the day, early dropout from classes, and a demoralizing sense of failure. This is why the standard model of pushing through 30- or 45-minute sessions of aerobics, treadmill walking, or cycling may defeat the purpose entirely. For people with compromised mitochondrial function, prolonged exertion can stress the very energy system the exercise is meant to support. The prevailing fitness culture — longer sessions, higher intensity, pushing through discomfort — is built on assumptions that do not apply to a nervous system running on fragile mitochondrial reserves. A surprising implication of this model is that some everyday tasks involving fine motor skills — such as handwriting, buttoning a shirt, or using a keyboard — may impose greater ATP demands on the basal ganglia than tasks involving gross motor skills, such as crossing a room.

Many exercise protocols are built on the premise that neural circuits need to be retrained by putting them through a series of motor patterns, but it is likely that the motor circuits themselves are not faulty — they remain encoded in distributed basal ganglia networks. In stroke or traumatic injury, motor circuits are destroyed and must be rebuilt through intensive practice. In Parkinson's, the circuits remain intact, but retrieving and executing them becomes harder as dopaminergic neurons are lost and surviving neurons must carry more of the energetic and signaling burden. As the number of dopaminergic neurons decreases and fewer cells must support more of the load, accessing these stored patterns becomes slower, less reliable, and more vulnerable to moment-to-moment fluctuations in ATP availability.

This distinction has important implications for exercise therapy. Much of Parkinson's rehabilitation has been modeled on stroke recovery, which is designed to rebuild motor circuits after they are destroyed. But in Parkinson's, the circuits remain intact; what is impaired is the energetic capacity needed to retrieve and execute them. Applying a stroke-style retraining model to Parkinson's misaligns therapy with the underlying biology, emphasizing repetition and intensity when the real limitation is energetic fragility.

However, since Parkinson's involves the loss of dopaminergic neurons, the remaining neurons that must compensate for such losses would benefit from structural support to execute motor patterns reliably. That support requires energy-intensive maintenance: enough dopamine receptors to offset the loss of signaling capacity, enough dendritic surface to integrate incoming signals, and enough synaptic strength to propagate the motor sequence without signal degradation. To maintain function, the brain must continually repair and reinforce the structure of the remaining dopaminergic neurons taking on this additional energetic and signaling burden.

These maintenance demands are metabolically expensive. Receptor synthesis, dendritic stabilization, and synaptic protein turnover all require abundant ATP. When mitochondrial output is unstable — as in the case with Parkinson's — these key repairs cannot be reliably completed. Short, daily sessions of chair-based interval exercise may create brief opportunities during which ATP availability rises enough for this maintenance work to proceed. More is not better: the sessions must be short because prolonged exertion drains the very energy reserve they are meant to create. Repeated over days and weeks, these micro-sessions may support the functional stability of remaining circuits. If this occurs, such sustained efforts could plausibly slow disease progression.

Understanding these energetic constraints also clarifies why identifying energy bottlenecks matters. The basal ganglia participate not only in motor control but also in cognitive and limbic circuits. Executive function — including working memory, task switching, and inhibition — depends on basal-ganglia

gating and is metabolically demanding. Under conditions of unstable mitochondrial energy production, these cognitive operations cannot be reliably supported, and they compete with motor demands for limited ATP. As a result, executive dysfunction often emerges early, leaving movement planning to occur within a system already struggling to maintain stability.

The same energetic instability helps to explain the rapid decay of benefits that many individuals report after exercise or dance sessions. Just as improvements can appear within minutes when ATP availability rises, they can fade just as quickly when energetic reserves fall. The sudden return of stiffness, hesitancy, or freezing after a period of fluid movement is not evidence that motor circuits failed to retain new patterns — it is evidence that they were never adequately powered to sustain them. In an energy-limited system, gains are transient unless the underlying ATP instability is addressed. Had rapid improvements primarily reflected structural neuroplasticity, one might expect them to persist longer than the transient gains often reported after individual sessions or even a series of classes.

Energetic fluctuations may also help explain why some individuals experience variability in their responsiveness to DBS. When the energetic capacity of motor circuits fluctuates, the same stimulation parameters may produce different effects from day to day, reflecting changes in ATP availability rather than changes in the underlying circuitry. This variability suggests that fluctuations in the energetic state may sometimes be mistaken for changes in stimulation needs, even when the underlying parameters remain appropriate.

The model also points to a complementary hypothesis. While brief exercise intervals may potentially increase ATP availability, strategies that reduce ATP demand may also have a beneficial effect. Meditation — even in forms as minimal as focused breathwork or calmly repeating a single word for a few minutes — may support motor function by reducing competing cognitive and emotional demands and preserving energetic resources for motor function. Exercise and meditation can be used singly or combined within the same short chair-based session, depending on what fits an individual's schedule and energy. Although this second possibility remains speculative and has not yet been directly tested with motor outcomes in Parkinson's, it represents a logical prediction of the energetic-dynamics model: both practices may support the energetic stability needed for symptom relief.

Since Parkinson's disease is not uniform, different clinical subtypes may respond differently to this proposed exercise structure. Very broadly, people with akinetic-rigid symptoms (more stiffness and slowness) may benefit most from short rhythmic intervals because this form is especially impacted by irregular ATP availability [13]. Those with tremor-dominant symptoms, which have more oscillatory loops

that interfere with voluntary movement, may find that meditation is more helpful because it reduces the number of signals vying for priority in the basal ganglia, which can reduce tremor intensity. Individuals whose symptoms center on gait and balance problems — sometimes grouped under “postural instability and gait disorder” — may respond well to the interval structure itself, which provides repeated timing anchors without exhausting their limited energy reserves. Because chronic fatigue in Parkinson’s may also reflect unstable ATP availability, these brief interval sessions could improve not only movement but subjective energy levels throughout the day. These possibilities are exploratory and should be evaluated in future studies.

Conclusion

It takes two to tango, but with the right exercise structure, individuals can design their own movement practice that can be done safely at home, alone, in just a few minutes a day. Short, seated sessions of rhythmic movement performed twice daily have the potential to temporarily increase ATP availability, providing the energetic support needed for smoother, more fluid movement. A complementary strategy is brief meditation sessions, which may reduce the cognitive load on the basal ganglia and make more ATP available for the complex signaling required for locomotion.

Time and energy are limited resources for anyone, but for people with Parkinson’s they are especially precious. An exercise program that respects both may offer meaningful improvements in daily function. When short exercise sessions with built-in pauses are paired with meditation on a regular basis, they may combine to allow the brain to make the most of its available dopamine and ATP. Over time, these windows may support the slow, energy-dependent maintenance and strengthening of existing neural circuits, offering the possibility not only of better movement in the moment but of slowing the trajectory of disease progression.

About the Author

Caroline C. Rodgers is an independent science theorist whose peer-reviewed work centers on neurodegeneration, with additional contributions in the fields of autism and maternal–neonatal health. She develops biologically grounded frameworks that challenge prevailing explanations for complex public health problems, offering alternative models where existing theories fall short.

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