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Designing personalized levodopa regimens using minimal inputs and hybrid simulation modelling

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Abstract: Levodopa is the gold-standard medication used to treat conditions such as Parkinson's disease and dopa-responsive dystonia. Despite its widespread use, levodopa has also been described as a "pharmacological nightmare:" the therapeutic window for individuals is narrow and varies based on sex, disease progression, and interactions with existing medications; its half-life is short, which can lead to unstable accumulations; delivery is impeded by dietary composition; efficacy is altered by illness and menstrual cycles; and medication side effects are easily confused for symptoms of the treated condition. These problems can result in persistent fluctuations between improved function and distressing symptoms and side effects. Most individuals are left to self-experiment between specialist appointments, which may be separated by months or years.

Over the last 30 years various methods, including pharmacokinetic/pharmacodynamic models, reinforcement learning, and sensor-based systems have been used to adjust levodopa regimens. Most of these models and methods have not gained widespread adoption likely owing to their complexity, data requirements, specialised hardware, and/or focus on a narrow set of symptoms and side effects. The exceptions are pump-based technologies, but these are not universally available and are not always compatible with an individual's preferences.

Complex solutions have not delivered widespread benefit to those dependent on levodopa. This paper therefore presents a purposefully simple simulation-based method to identify an individual's therapeutic window using data from a single representative day. It describes how optimization can design a regimen from this data and how calibration might be performed to tailor a minimal set of parameters to users. A UI prototype is presented to demonstrate how this technology can be delivered to populations dependent on levodopa, which frequently experience reduced fine motor control or may have limited experience with technology. Two case studies are presented to demonstrate the tool's clinical value. Strategies for widespread testing and inclusion of other dopaminergic medications such as agonists and antagonists are also discussed.

By bridging the gap between theoretical modelling, clinical practice, and population-appropriate design, this work lays the foundation for a precise, individual-centric, and scalable approach to managing levodopa-dependent conditions.

Keywords: *Parkinson's disease, Levodopa, Dopa-responsive dystonia, Pharmacokinetics, Pharmacodynamics*

1. INTRODUCTION

Levodopa is a dopaminergic medication used to treat conditions such as Parkinson's disease (Ghandi 2023) and dopa-responsive dystonia (Lee 2014). Levodopa is a precursor to dopamine and is rapidly converted into the neurotransmitter once absorbed. With a short half-life (Nyholm 2012) and the need for an inhibitor to prevent peripheral metabolism (i.e. use outside of the brain) (Ghandi 2023), levodopa has been described as a pharmacological nightmare (Tambasco 2018). This is compounded by its narrow therapeutic window: too little or too much levodopa can lead to side effects worse than the disease itself, including nausea, dyskinesia, hallucinations, and impulsivity (Parkinson's Foundation 2025). Levodopa's rapid absorption, distribution, and metabolism exacerbates this problem, with individuals often experiencing the gamut of symptoms and side effects within the span of a few hours. Identifying and staying within an individual's therapeutic window to prevent these fluctuations is therefore desirable but challenging: the window may narrow as conditions progress (Stocchi 1995); it is affected by sex and disease progression (Cattaneo 2024); symptoms may vary by time of day (Lee 2014); menstrual cycles and illness can alter levodopa's efficacy (Sprenger 2014); it is impacted by diet (Agnieszka 2022); and many common medications are dopamine antagonists. Once the window is identified, a suitable regimen must be designed to match. Pump-based technologies (e.g. Vyalev 2025) can deliver a constant dose to match the window, but as of writing are not universally available nor always suitable due to personal needs and preferences.

Various methods have attempted to identify an individual's therapeutic window and/or suggest regimen alterations: examples include detailed mathematical models (Baston 2016), reinforcement learning (Watts 2020), and sensor-based dosing systems (Thomas 2019). Despite regimen optimizers existing for at least 30 years (Hacisalihzade 1989), few if any of these tools have been widely adopted. While barriers such as awareness and cost are present when scaling a solution, the complexity of existing approaches plays a role: some methods, for example, are clinically impractical (Hacisalihzade 1989) and people with Parkinson's can struggle to log symptoms due to mobility impairments. A user-friendly tool is more likely to meet the needs of this demographic.

This paper presents a simulation-based tool that determines an individual's therapeutic window using symptom records from a representative day. It then designs a regimen minimizing deviation from the window to help mitigate symptoms and side effects. It is purposely built with minimal assumptions and uses widely available parameters to model levodopa accumulation. A prototype UI is presented to demonstrate a viable interface for mobility-impaired users. Two case studies show that the approach can meaningfully reduce symptoms and side effects in levodopa-responsive conditions.

2. MODELLING BACKGROUND

Models to help optimize levodopa regimens have existed for over 30 years. Hacisalihzade et al (1989) created a device to track the the end of the thumb after administering levodopa, which allowed a model based on Fourier transforms to predict the individual's optimal dose. Their method resulted in strong agreement between modelled and self-assessed motor abilities but required an individual's arm to be strapped to an armrest with a plaster cast for three hours. As the authors note, this is not clinically practical. It also measures a narrow picture of Parkinson's disease, excluding symptoms and side effects such as constipation, nausea, and mood swings.

Baston et al (2016) provide a more recent example. They combined a simulation model of the basal ganglia (i.e. the area of the brain implicated in Parkinson's disease) with a traditional two-compartment pharmacokinetic model of levodopa. The model produced accurate predictions of both levodopa blood plasma levels and patient movement characteristics, but it was unclear how easily the approach could scale due to challenges in parameter estimation. Nevertheless, the ability to accurately predict symptoms is clearly valuable when optimising a medication regimen.

Watts et al (2020) utilized sensor data and deep reinforcement learning (DRL) to produce levodopa regimens. They focussed on reducing bradykinesia and dyskinesia, which are proxies for too little and too much levodopa, respectively. Their models approximated real-world regimens when administration times were fixed, designed more effective regimens when timing was flexible, and greatly increased dose frequency when set to minimize symptoms. This third model outperformed clinical recommendations on virtual patients, but at 1200 mg per day could result in side effects like pronounced nausea in real patients or require hourly administration, which can conflict with an individual's preferences. Other DRLs produced even higher daily totals, suggesting that models optimizing a narrow set of metrics could negatively impact the broader patient experience.

Thomas et al (2019) developed a simulation-based method using movement sensor data to reduce Parkinson's symptoms while avoiding medication side effects. Their method proved effective in a clinical trial but required a special-purpose wearable device. It also assumed that a patient's therapeutic window was constant, but an individual's required levodopa dose can change as the day progresses (Hacisalihzade 1989).

These studies reveal a theme: regimen optimization is possible but has so far faced barriers to adoption such as requiring specialized hardware or focussing on a narrow set of easily measured symptoms. The former is not

universally available; the latter misses the broader experience of living with a chronic condition. What is missing is a simple solution that empowers individuals to optimize the outcomes important to them.

3. METHOD

3.1. Inception and development of prototype

In 2024 an individual known to the author was diagnosed with dopa-responsive dystonia (DRD) (Knowles 2024). DRD is a rare disorder that affects 1/1,000,000 to 1/200,000 people worldwide (Orphanet 2025). Similar to Parkinson's disease, DRD is caused by impaired dopamine production that eventually leads to dystonia. DRD does not feature degenerative progression, and symptoms generally improve in the morning and worsen throughout the day. Clinicians familiar with DRD are rare and timely access can be elusive if they are locally accessible at all. This often means that individuals with DRD are prescribed levodopa following diagnosis and are left to refine their regimen via trial and error. This is problematic as dystonia can be extremely painful: finding an appropriate levodopa regimen can take months of agonizing self-experimentation. We concluded that a "good enough" solution was preferable to waiting for expert advice or a perfect model. The prototype was successful and satisfactorily managed the individual's dystonia and levodopa side effects.

3.2. Updated model

Arbitrarily complex medication regimens were challenging to represent in the prototype's pure system dynamics model. A hybrid model in which each dose was an agent produced a much more flexible structure. Each agent in the updated model contains a system dynamics model of levodopa accumulation (Figure 1) with parameters appropriate to the dose type (i.e. instant or controlled). Total levodopa accumulation is calculated by simply summing stock *R* across all doses.

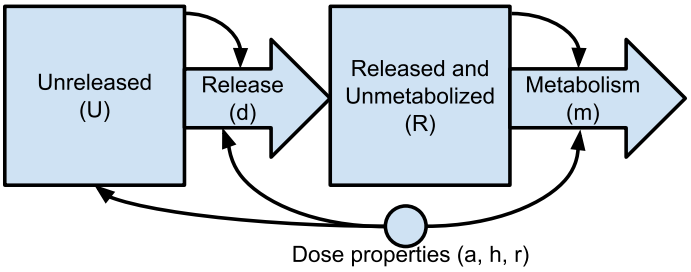

Figure 1 The updated model. Each dose is administered, released into the body, and then metabolized. See Table 1 for details.

While more accurate models exist (e.g. Baston 2016), the properties of levodopa that make it a frustrating drug for individuals also make a simple model fit for purpose: confounds are minimized as individuals avoid taking it with interacting substances (Agnieszka 2022) and its short half-life means it does not accumulate in large quantities between days. Our minimal assumptions allow parameters to be based on widely published sources (e.g. AA Pharma Inc. 2020) and individually tuned. The model's output is a theoretical accumulation of levodopa as blood serum levels have, until recently, been challenging to measure in a decentralized manner (Longardner 2024). The model purposely excludes dopamine as this system is complex and would require substantially more assumptions.

Component	Definition	Value or Formula
a	Levodopa contained in dose	Dose-dependent. E.g. 50 mg
r	Release speed: instant or uniform rate	Dose-dependent. E.g. 25mg per hour
h	Levodopa half-life	2.5 hours
U	Dose remaining	Initial value: $U = a$
R	Levodopa released but unmetabolized	Initial value: $R = 0$
d	Release rate	$d = if\ U > 0\ then\ r\ else\ 0$
m	Metabolism rate	$m = R / h$

Table 1 Model parameters and formulae calibrated to volunteers. Population half-lives have a wide range with an average of ~1.5 hours. Controlled release characteristics vary (Arav 2023), but a uniform assumption worked sufficiently well for our volunteers.

3.3. Mapping ON/OFF times to theoretical levodopa accumulations

Individuals using levodopa describe periods with and without symptoms as OFF and ON time. While OFF time can be challenging to assess as many are not informed of the difference between symptoms, side effects, and normal ageing, it is identifiable once reported due to its distressing or frustrating nature (e.g. constipation, tremor, frozen gait, and dyskinesia). ON times can be inferred from periods between OFF times.

Because the model can calculate the theoretical accumulation of levodopa across the day, ON and OFF times can be used to calculate three regression curves: the target accumulation (i.e. a curve that passes through ON time accumulation levels), the therapeutic window's upper limit (i.e. a curve that passes through OFF time accumulations *above* the target), and the therapeutic window's lower limit (i.e. a curve that passes through OFF time accumulations *below* the target). A single representative day can be enough to estimate the range; in practice,

not all individuals can readily identify ON/OFF periods. A symptom journal is therefore valuable to produce a representative average given transient confounds (e.g. illness, stress, menstrual cycles, comorbid conditions).

3.4. Deriving an improved regimen from the model

To use the model an individual needs to supply their current regimen and ON/OFF times (either a single day or derived from journaled averages). The individual can then specify their regimen evaluation period: for example, they may need to have their medication "work" between 09:00 (first time symptoms typically emerge) and 23:00 (mid-sleep where their current regimen produces pain and discomfort); a default evaluation period can also be used. The theoretical levodopa accumulation for each time point within the evaluation period is then calculated. This allows a score to be derived for later optimization: for example, the current regimen's levodopa accumulations can be scored against the target accumulation curve using a simple sum of residuals. Preference-based penalties can also be applied: for example, regimens with more administration times are harder to adhere to successfully and thus may be less preferable than a simpler regimen with slightly worse results. Regimens that interfere with daily life (e.g. established mealtimes, family activities) can also be penalized.

With a scoring function established for the individual's existing regimen an optimizer can be applied to test and score alternatives. Our tool is built in AnyLogic, so the standard OptQuest algorithm is currently applied. The model's optimizer adjusts three main parameters: the number of administration times per day; the spacing between each administration time (which may vary); and the combination of doses at each administration time (chosen from a list of known dosages, formulations, and combinations thereof).

Note that the model assumes non-levodopa medications to be constant. For example, an individual may take a dopamine antagonist whose effects vary across the day due to a long half-life: because the model couples subjective experience with levodopa accumulation, this simply produces a therapeutic window that curves.

4. CASE STUDIES

Our tool has been successfully applied to two individuals.

4.1. Individual with dopa-responsive dystonia

Our first volunteer (Knowles 2024) was prescribed three instant release levodopa/carbidopa tablets per day. As these tablets are rapidly absorbed, metabolized, and excreted, this resulted in a predictable sawtooth accumulation pattern. The first iteration of our tool suggested that controlled release tablets would result in greater stability. It became apparent, however, that the volunteer had varying levodopa needs as the day progressed: regular administration of controlled release levodopa to maintain stable accumulation resulted in an afternoon "crash." With iterative refinement the model suggested a more complex regimen (Table 2). This alleviated the afternoon crash and allowed their dystonia to be managed. As the dystonia had caused widespread spinal disc injuries, consistent symptom management has been imperative to facilitate recovery and restore mobility.

Time	Dose
09:00	50 mg levodopa / 12.5 mg carbidopa instant release (IR) 100 mg levodopa / 25 mg carbidopa controlled release (CR)
13:00	50 mg / 12.5 mg IR 100 mg / 25 mg CR
17:00	100 mg / 25 mg CR
21:00	100 mg / 25 mg CR

Table 2 Volunteer's regimen. Doses are irregular as needs change with time of day.

4.2. Individual with Parkinson's disease

Our second volunteer was a knowledgeable member of Saskatoon's Parkinson's community. Their original regimen consisted of four administrations of 1.5 levodopa/carbidopa instant release tablets per day (totalling 150 mg levodopa / 37.5 mg carbidopa). These produced ON/OFF periods at predictable times that largely matched our tool's predictions: exceptions included mirror movements and stress-induced dyskinesia, which do not have a straightforward relationship with levodopa dose and are thus not naturally identified by our tool (Espay 2006). The volunteer's therapeutic window appeared stable for much of the day, which allowed the tool to design a regimen composed entirely of 200 mg levodopa / 50 mg carbidopa controlled release tablets. The optimized regimen resulted in sustained reductions in nausea and dyskinesia compared to their original regimen. The volunteer reports that their day "is more comfortable, nausea-wise, and more controlled" and that they are no longer "rushing home from [the] gym to eat something" before their medication, which was previously required to manage side effects.

5. DELIVERING A USER-FRIENDLY TOOL

For the model to be widely adopted it must be made easy to access (e.g. via a mobile-friendly website) and expressly designed for its audience's needs. People with Parkinson's disease, for example, are typically elderly and may struggle to use mobile devices due to their symptoms: after consulting our local support group (Saskatoon Support Group 2025) we discovered that this is particularly true when tools require fine motor control like typing. Change or loss of fine motor control is one of the earliest symptoms of Parkinson's noticed by individuals and their carers (Eklund 2022); with many people with Parkinson's experiencing tremor, complex apps and websites are unviable. Inputs must be extremely simple with large buttons and minimal steps. More complex inputs, if required, must fall to carers and medical practitioners: this indicates 1) a need to link these carers to an individual and 2) that medical practitioners will almost certainly see a substantially expanded view of the delivered system, which is reinforced by the fact that many countries have strict regulations for software directly advising patients.

We constructed a UI prototype to assess the feasibility of coupling these goals with our tool. The main components, viewed from the individual's or a carer's perspective, are: 1) a dashboard containing the individual's current regimen; 2) a means by which to update this regimen and log ON/OFF times; and 3) a list of linked carers and medical practitioners. Recommendations are only provided to practitioners as practitioner-focussed decision support systems have fewer regulatory requirements, increasing availability.

The prototype was presented to a group of 50 attendees. Initial feedback was positive with a consistent request: the ability to journal symptoms, exercise, illness, and sleep as these factors can make ON/OFF times inconsistent. Attendees also stressed the importance of non-levodopa medications prescribed for comorbid conditions or for managing symptoms (e.g. blood pressure changes).

While the prototype UI and model are not connected – a non-essential requirement when the goal is rapid feedback on interface design – the UI was designed to be web-first to ensure widespread availability. It and its database are therefore naturally hosted on a web server: as our model is constructed in AnyLogic, the UI can be connected to the model via the AnyLogic Cloud API (AnyLogic 2025) once middleware such as authentication is in place.

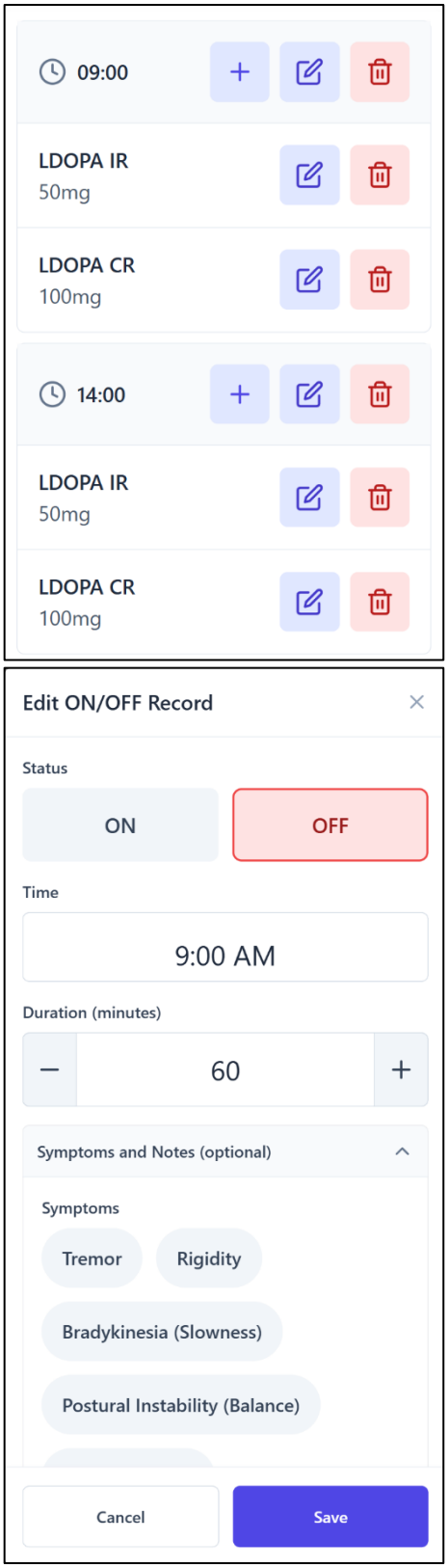

Figure 2 Mock ups of medication schedule (top) and ON/OFF modal (bottom). Large buttons help facilitate mobility impairments.

6. LIMITATIONS

The model has several limitations that must be addressed for a larger audience. The first complication is levodopa's half-life. While our two volunteers appear to have similar levodopa metabolism, their half-life estimate of 2.5 hours is larger than reported (AA Pharma Inc. 2020). This said, it is estimated that an equivalent dose of levodopa provides a 2.2 times higher exposure in women than men (Nishikawa 2020): simple math using reported averages, sex ratios in the Parkinson's community, and the experience of our female volunteers could be used to estimate sex-specific half-life values. Calibration is also needed for the delay between ingestion and absorption but should be estimable given that instant release tablets often result in sudden side effects once absorbed.

The model assumes levodopa/carbidopa tablets as these are the first line of Parkinson's treatment in the author's locale. Other pairings such as levodopa/benserazide are also available: as benserazide is substantially more potent than carbidopa (Prada 1987), the model may need to account for its unique properties. Dopamine agonists, such as amantadine, are used by individuals who do not respond well to levodopa, but it is unclear if our simplifying

assumptions can be used for this class of medications. Two obvious alternatives exist to handle these challenges: only recommend dopaminergic medications from a single family (e.g. only amantadine or levodopa/carbidopa but not both) or incorporate literature on dose equivalents and interactions (e.g. Rinne 1979).

Finally, the model assumes non-dopaminergic medications to be constant. This is a flawed assumption if a new regimen results in the removal of other medications, especially dopamine antagonists. The model can handle this by simply iterating until a new solution is found (e.g. trial new regimen for two weeks, recommend changes, and repeat until satisfied), but a more intelligent approach is likely ideal if the goal is to reduce self-experimentation.

7. CONCLUSIONS AND RECOMMENDATIONS

This paper presented a deliberately simple model built to identify an individual's therapeutic window for levodopa and design a suitable regimen. Existing tools were too complex, costly, or inaccessible at scale: our aim was to therefore build a tool that was low input and user friendly yet still clinically useful. The result was a hybrid system dynamics and agent-based model that simulates levodopa accumulation, identifies an individual's therapeutic window using minimal inputs, and applies an optimizer to design an improved regimen. The prototype achieved its goals and has been successfully applied to two individuals, demonstrating its potential to reduce symptoms and side effects in individuals with levodopa-responsive conditions. A web-based prototype UI was developed with feedback from the local Parkinson's community, which produced a clear roadmap for product development.

Scaling this tool involves challenges, most of which are readily overcome. Some are purely technical: calibration should be performed to determine levodopa parameters for each user; a journaling system must be developed; and the platform must be hosted on the web with resulting compliance challenges such as GDPR and HIPAA for European and American audiences, respectively. Others are clinical: broader studies should be performed to ensure results are generalizable and theoretical levodopa accumulations should be validated. Co-prescribed medications, such as dopamine agonists and antagonists, should be included. The final and perhaps most challenging hurdle is simply earning the trust of clinicians and pharmacists: individuals using levodopa are regularly encouraged to participate in cutting-edge research (e.g. Michael J. Fox Foundation 2025), but practitioners require substantial evidence and peer adoption before taking advice from recommendation systems. This creates a negative feedback loop in which the tool is not adopted due to lack of adopters, which suggests a bottom-up, product-led growth strategy will be valuable (Wildlund 2021). This would require a frictionless system for individuals using levodopa (i.e. designed to be painless given their likely mobility constraints) that heavily incentivizes them to invite practitioners. A natural incentive for this action is requiring a medical practitioner to see detailed benefits identified by the model. This contrasts with a top-down, sales-led growth strategy that targets practitioners or Parkinson's organizations directly. While most practitioners may simply read the individual's symptom journal used to generate model results, which itself is valuable, the availability of a decision support tool will inevitably nudge some early adopters to evaluate the tool and invite their patients, which can help spread the tool further.

With calibration, validation, and iterative development based on user feedback this tool could offer a practical way to personalise levodopa regimens at scale. Journaling, invite features, web hosting, and tailoring UIs by user type are straightforward engineering tasks. Detailed pharmacokinetic models and drug equivalency information can be used to expand the tool. Rapid, decentralized blood testing can help validate and adjust the model's levodopa predictions. Bottom-up pressure from excited patients can nudge practitioners to try the tool.

By addressing model limitations, implementing UI enhancements, and validating with a wider audience the tool has the potential to significantly streamline levodopa regimen design and improve quality of life. This work offers a path toward making personalized medicine available to those with levodopa-responsive disorders and could meaningfully reduce the frustrating and lengthy self-experimentation undertaken by millions worldwide.

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