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Research on the post-operative outcomes of Parkinson's disease using a neural network optimized with a genetic algorithm

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ABSTRACT

Parkinson's disease is a progressive neurodegenerative pathology characterized by impairment of motor function. Dynamic monitoring and neurological assessment of the patient's clinical condition after surgical interventions used for treatment, especially after deep brain stimulation procedures, are of particular importance. This study proposes a hybrid approach that integrates a recurrent neural network with a stochastic model for analyzing post-operative outcomes related to Parkinson's disease. Through a mathematical model constructed based on the Ornstein-Uhlenbeck process, changes in tremor, motor functions, and dopamine levels over time have been imitated. Clinical biomarkers, medication protocols, and biosignal data have been analyzed using a neural network optimized with a genetic algorithm to identify prognostic indicators. Simulation and test results show that the proposed approach is capable of discriminating between stable and deteriorating post-operative patient conditions with high accuracy. This model can also serve as an effective tool in developing personalized therapy strategies by playing a supportive role in neurological decision-making.

1. Introduction

Parkinson's disease is a progressive neurodegenerative disorder of the central nervous system, primarily manifesting through motor symptoms such as tremor, movement slowness, muscle rigidity, and postural instability. It mainly affects motor (movement-related) functions and occurs as a result of the destruction of dopamine-producing nerve cells in the area of the brain called the "black substance". This disease is particularly

widespread among the elderly population and negatively affects their quality of life. Age-related neurodegeneration is the most common cause hereditary in 10-15% of case. While pharmacological agents used in the treatment of Parkinson's disease, especially levodopa preparations, are effective in managing symptoms during the initial stages, long-term use leads to motor fluctuations and reduced therapeutic effectiveness. In this context, surgical interventions such as deep brain stimulation (DBS) are applied as

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important alternatives in more advanced stages of the disease. DBS was successfully applied by Alim-Louis Benabid (1987) to treat the tremor symptoms of Parkinson's disease. Benabid and colleagues showed that tremor can be reduced by continuous electrical stimulation and that this is an alternative to destructive (e.g., thalamotomy) methods.

The objective of this study is to describe how tremor, motor functions, and dopamine levels change over time in the post-surgical phase of Parkinson's disease through mathematical modeling and to predict these processes using neural networks. The proposed approach serves both for more accurate monitoring of disease dynamics and for optimizing clinical decision-making processes.

Our main scientific innovation in this study was to obtain the prognosis of dopamine, tremor, and motor function parameters of Parkinson's patients based on the data of the last two years, after surgery, for the next month, day by day, with recurrent neural networks (RNNs) optimized with genetic algorithms (GA) (Brown, R. G, et al., 1998). In the study, a stochastic differential equation system was proposed to express the dynamics of symptoms for Parkinson's disease, and was carried out based on the Ornstein-Uhlenbeck process.

This paper proceeds as follows. Section 2 depicts the literature review. The stochastic model based on the Ornstein-Uhlenbeck process that describes the symptom dynamics of Parkinson's disease in patients is given in Section 3. Section 4 investigates the prediction of parameters symbolizing neuro-reparative and clinical fluctuation factors using a built-in Long Short-Term Memory (LSTM) model optimized with a GA. Semi-analytical solution of the stochastic model under consideration is given in Section 5, and the obtained stochastic integrals are discretized using the Euler-Maruyama scheme in Section 6. Section 7 analyzes the experimental results, as well as a comparison of numerical methods for solving stochastic integrals and estimating the semi-analytical solution of the equations. Section 8 summarizes the findings and results for further investigation.

2. Related works

There are several studies on mathematical modeling of Parkinson's disease and the application of artificial intelligence for the postoperative period: e.g, Charles S.Venuto et al. (2017) reviewed studies evaluating how UPDRS

(Unified Parkinson's Disease Rating Scale) indicators used in clinical trials change over time with traditional and mathematical models. Here, both the progressive course of the disease and the treatment effects are modeled. In addition, Qingyun Wang et al. (2020) have conducted research on the improvement of deep brain stimulation through control systems and computer models, and patient-specific open and closed-loop stimulation strategies. M.A.Elfouly (2024) conducted a study on dynamic models described by "Forward-Backward" for the occurrence of synchronization disorders and tremor in Parkinson's disease. Jeroen. G.V. Habets et al. (2020) built a machine learning model to predict the motor response one year after subthalamic nucleus DBS surgery, and as a result, the model was able to predict poor motor responses with high accuracy.

However, the majority of the studies related to the outcomes of the disease under consideration so far lack sufficient accuracy in stochastic modeling and semi-analytical solutions. RNN model was built using the values of tremor, motor, and dopamine recovery rate, as well as average values and variability of these biomarkers that are obtained by statistics of the patient's postoperative milestones. The recorded parameters of a Parkinson's patient for last two years were measured and applied in the artificial intelligence model. The prepared model gave the prognosis of dopamine, motor function, and tremor indicators for the next months.

The key research findings of our study can be summarized as follows:

- Using a stochastic differential equation system to express the dynamics of symptoms;
- An extensive validation process through direct comparison of numerical and analytical solutions to ensure the reliability of the models;
- Obtaining the prediction of dopamine, motor Function, and tremor indicators with minimal errors through the developed artificial intelligence model.

3. Stochastic model of symptom dynamics for Parkinson's disease

This section defines the stochastic model of the symptom dynamics for Parkinson's disease after post-operation (Obeso, J. A, et al.,2000). The model under consideration has been constructed

according to the Ornstein-Uhlenbeck process. A stochastic process, the Ornstein-Uhlenbeck process finds use in both the physical sciences and evolutionary biological mathematics (Gillespie, D. T. 1996). It was initially used in physics as a model to simulate the velocity of a big Brownian particle when friction was present. It bears the names of George Eugene Uhlenbeck and Leonard Ornstein (1930). The Ornstein-Uhlenbeck process is mathematically established on the basis of the Wiener process — a continuous-time stochastic process proposed by Norbert Wiener in 1923, which formalized the idea of Brownian motion first explained by Albert Einstein in 1905. The Wiener process reflects pure stochastic movement devoid of memory or inclination to revert to equilibrium, whereas the Ornstein-Uhlenbeck process incorporates mean-reversion, representing systems where variables oscillate around a consistent long-term average (Benabid et al., 1991). This process is expressed by the following differential equation:

$$dX(t) = \theta(\mu - X(t))dt + \sigma dW_t \quad (1)$$

For this process, $X(t)$ - is a stochastic process, μ - is the long-term mean, $\theta > 0$ - is the rate of mean reversion, σ - is the volatility and W_t - is the Wiener process.

In Parkinson's disease, the unstable change of symptoms over time and their tendency to stabilize in response to treatment create suitable conditions. Therefore, in the model describing the dynamics of the disease, the behavior of each symptom is structured like this process (Aron, A. R. et al., 2014). For this approach, let us introduce the following notation: We should formulate a stochastic model that relates the recovery rates, clinical equilibrium values for each symptom, and intensity of stochastic fluctuations parameters as the following (2)-(3) system of stochastic differential equations:

$$\begin{cases} dT(t) = \alpha(\mu_T - T(t))dt + \sigma_T dW_t^{(1)}, T(t) \in [3, 7]Hz, t \geq 0 \\ dM(t) = \beta(\mu_M - M(t))dt + \sigma_M dW_t^{(2)}, M(t) \in [0.2, 1.0], t \geq 0 \\ dD(t) = \gamma(\mu_D - D(t))dt + \sigma_D dW_t^{(3)}, D(t) \in [0.1, 1.0], t \geq 0 \end{cases} \quad (2)$$

$$T(t_0) = T_0, M(t_0) = M_0, D(t_0) = D_0 \quad (3)$$

$$\alpha \in [0.3, 1.2], \beta \in [0.2, 1.0], \gamma \in [0.5, 1.5] \quad (4)$$

$$\mu_T \in [4, 6], \mu_M \in [0.4, 0.8], \mu_D \in [0.3, 0.7] \quad (5)$$

$$\sigma_T \in [0.05, 0.3], \sigma_M \in [0.02, 0.2], \sigma_D \in [0.01, 0.1] \quad (6)$$

where $T(t)$ is a finite continuous function expressing the patient's tremor intensity over time

in hertz (Halliday, D. M., 1995). $M(t)$ is a finite, continuous function shows the motor function index in a normalized scale. $D(t)$ is a finite, continuous function expressing the patient's dopamine level over the postoperative milestone. $W_t^i, i = \overline{1,3}$ functions follow a normal distribution as

$$W_0^i = 0, W_t^i - W_s^i \sim \mathcal{N}(0, t - s) \quad (7) \\ 0 \leq s < t$$

serves as an individualized stochastic driver capturing latent neurophysiological variability, allowing the system to realistically emulate the irregular, patient-specific fluctuations observed in Parkinson's disease progression (Kotz, S., & Nadarajah, S. 2000). Note that, $T(t), M(t), D(t)$ functions are the only differentiable in the Itô sense. Also, $W_t^i, i = \overline{1,3}$ can be processed via the Itô integral (Itô, K., 1944) in stochastic analysis.

The parameters that we have used in this model, α, β, γ are the recovery rates for each respective symptom. Other constant coefficients in the Eqs.(2)-(3), μ_T, μ_M, μ_D - long-term equilibrium values representing the asymptotic health targets over post-operation. Intensities of stochastic fluctuations are expressed by $\sigma_T, \sigma_M, \sigma_D$.

In next section, we investigate the prediction of parameters symbolizing neuro-reparative and clinical fluctuation factors using a built-in LSTM model optimized with a GA.

4. Predicting parameters by using a neural network model optimised with a genetic algorithm

For the semi-analytical solution of the stochastic model under consideration Eqs.(2)-(3), we need to find predicted values of the coefficients. $\alpha, \beta, \gamma, \mu_T, \mu_M, \mu_D, \sigma_T, \sigma_M, \sigma_D$, which are used in the modeling the postoperative outcomes of Parkinson's disease by constructing a recurrent neural network model.

When preparing the dataset required for the modeling and prediction of Parkinson's disease symptom dynamics, several biomedical and neurophysiological factors were carefully considered. The tremor frequency range $T(t)$ was selected based on clinical observations (Benamer et al., 2000), with values typically ranging between 3–7 Hz, which correspond to common Parkinsonian rest tremor frequencies. Motor function index $M(t)$ was normalized between 0.2 – 1.0, informed by Unified Parkinson's Disease Rating Scale (UPDRS) assessments.

Dopamine activity levels were scaled within the interval 0.1 – 1.0, in accordance with relative concentrations observed in dopaminergic imaging and positron emission tomography (PET) scan studies. PET imaging allows for the in vivo quantification of dopamine synthesis, receptor availability, and synaptic activity by using radiolabeled tracers such as flurodopa or raclopride, providing a reliable biomarker for dopaminergic function in neurological conditions such as Parkinson's disease (Abdullayev et al., 2025).

The stochastic variability of these clinical variables was modeled using Ornstein–Uhlenbeck processes to realistically capture physiological noise and intra-patient fluctuation, with stochastic intensity coefficients (DeLong et al., 2007). $\sigma_T, \sigma_M, \sigma_D$ informed by neurobiological data on signal variability in basal ganglia circuits. Recovery rate parameters α, β, γ were initialized and later optimized to reflect the regenerative and compensatory processes in neurodegenerative conditions.

The choice of these variables, their physiological ranges, and model coefficients was grounded in neurological literature, physiological system modeling, and medical cybernetic approaches to modeling disease progression.

Based on these scientific research works, a dataset consisting of last two years (730 days) relevant parameter values of a patient suffering from Parkinson's disease during the postoperative treatment and recovery period, synthesized from research studies on the subject, was constructed, and based on this dataset, the patient's neuro-reparative values were predicted for the following day. The additional parameters for the training and testing datasets are illustrated in Table 1.

Table 1. Description of the experimental datasets

Dataset	Number of samples	Number of features
Training dataset	560	9
Testing dataset	70	9

After preprocessing the dataset, considering the temporal dependencies present in the data, a multivariate LSTM network was chosen as the main architecture for the recurrent neural network. LSTM was proposed by Sepp Hochreiter and Jürgen Schmidhuber (1997). This model was developed to overcome the "vanishing gradient" problem that classical recurrent neural networks RNNs have in learning long-term dependencies.

The LSTM model introduced "cell state", input, output, and forget gates. This architecture allows decisions to be made about whether information is stored or forgotten over time. To overcome the difficulties of manual selection of hyperparameters, an evolutionary optimization method GA (John Holland, 1992) was applied to systematically and efficiently search for the most optimal hyperparameter combination. The model was trained using the Adam (Adaptive Moment Estimation) optimizer, a highly efficient and widely adopted optimization algorithm specifically designed for training deep learning models (Yamins et al., 2016). This allows Adam to adjust the learning rate dynamically for each parameter, ensuring more precise and stable updates. Introduced by Diederik P. Kingma and Jimmy Ba in 2015, Adam is particularly well-suited for time series prediction tasks and sequential data modeling, as it efficiently handles non-stationary objectives and noisy or sparse gradients that are common in real-world datasets. One of the key features of Adam is its use of bias-correction terms, which compensate for the initialization of the moment estimates and provide better convergence, especially during the early stages of training.

In the context of our study, Adam's ability to adaptively scale the learning rate helped stabilize the learning process and accelerate convergence, even under the complex dynamics of stochastic differential equations and neural network parameter optimization (Särkkä et al., 2019). Its robustness and minimal need for manual tuning make Adam an ideal choice for optimizing LSTM networks and other deep learning architectures involved in modeling physiological processes (Mutallimov et al., 2025). Given these strengths, Adam is considered a de facto standard for a wide range of machine learning problems.

The main goal of the GA was to automatically find three critical hyperparameters that directly affect the performance of the model: the number of neurons in the LSTM layers, the learning rate, and the dropout rate for regularization. Through the processes of selection, crossover, and mutation, the GA iteratively improves these combinations (Shalev-Shwartz et al., 2014).

In the LSTM model, the *tanh* function was used as the activation function for the hidden layers. In the output layer, a *linear* activation function was used, which allows the model to predict sequential and continuous quantitative values. Such a choice supports the real numerical nature of the

implemented parameters and the performance of the neural network as a regression function. The additional information for the model is illustrated in Table 2.

Table 2. Description of the LSTM model

Layers	Activation function	Units
Input layer	-	30 x 9
LSTM layer 1	Tanh	115
Dropout layer 1	-	0.15
LSTM layer 2	Tanh	57
Dropout layer 2	-	0.08
Output layer	Linear	9

The final optimal configuration was determined as 115 neurons for the main LSTM layer, a learning rate of exactly 0.0005 , and a dropout rate of 0.15 . It was these specific values that were used in building the final forecasting model, ensuring its reliability and accuracy (Brockwell et al., 2016). Performance of the final model was rigorously evaluated on a stripped-down test set that was not used in any of the training or optimization stages.

The evaluation was based on the Mean Absolute Error (MAE) and Mean Squared Error (MSE) metrics for each predicted parameter. The error rates calculated for each coefficient on the test set are presented in Table 3.

Table 3. Predicted values and errors of the coefficients

Parameters	Predicted value	MAE / MSE
α	0.801233530	0.054646 / 0.004261
β	0.570061624	0.018373 / 0.000536
γ	1.000880241	0.038808 / 0.002463
μ_T	5.038064003	0.090973 / 0.002463
μ_M	0.609865963	0.012660 / 0.000222
μ_D	0.486436784	0.012811 / 0.000230
σ_T	0.179216072	0.010059 / 0.000158
σ_M	0.104413509	0.004714 / 0.000039
σ_D	0.050313860	0.008457 / 0.000096

Additionally, the efficiency of the model training process itself is shown in Fig. 1. This graph depicts how the training loss and validation loss errors decrease over epochs (training periods).

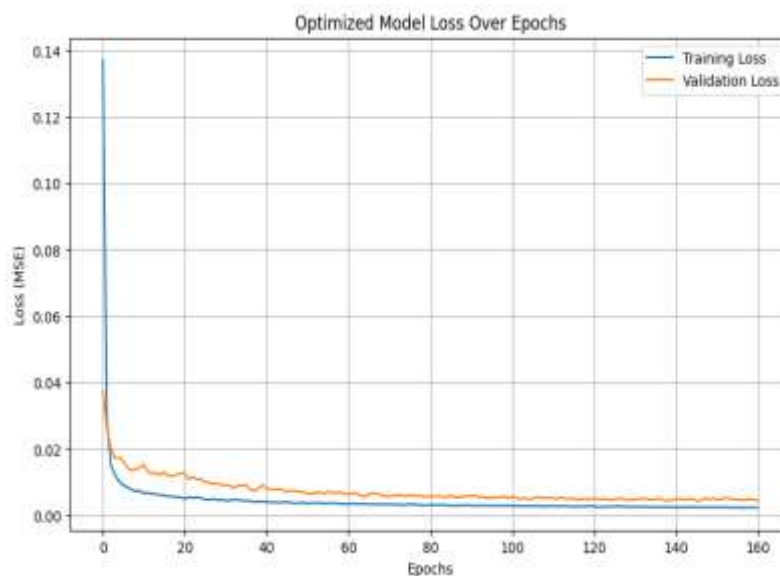


Fig. 1. Optimized model loss over epochs

5. Semi-analytical solution of the stochastic differential equation system

In classical deterministic differential equations, the function sought is mainly determined as a function of time. However, in real biological and medical systems, random variability, physiological noise, and environmental factors make modeling

the system with classical deterministic approaches inaccurate and unreliable. For this reason, stochastic models, in particular Itô stochastic calculus, are resorted to.

Before using stochastic methods to equation system, the first equation in the system of stochastic equations (2) - (3), we rearrange the equation to group like terms:

$$dT(t) + \alpha T(t)dt = \alpha \mu_T dt + \sigma_T dW_t^{(1)} \quad (8)$$

that we got a non-homogeneous linear stochastic equation. Then we use the integrating factor method just like in deterministic ordinary differential equations. Thus, according to Itô's lemma (Itô, K., 1951), we are defining the integrating factor as:

$$I(t) = e^{\alpha t} \quad (9)$$

For the next step, multiply both sides of the Eq.(8) by $I(t)$:

$$\begin{aligned} e^{\alpha t} dT(t) + \alpha e^{\alpha t} T(t)dt &= \\ &= \alpha \mu_T e^{\alpha t} dt + \sigma_T e^{\alpha t} dW_t^{(1)} \end{aligned} \quad (10)$$

The left-hand side of the equation (10) is the total differential of the product (11):

$$d(e^{\alpha t} T(t)) = \alpha \mu_T e^{\alpha t} dt + \sigma_T e^{\alpha t} dW_t^{(1)} \quad (11)$$

After integrating both sides of equation (11), the following equations (12) -(14) are obtained:

$$\int_0^t d(e^{\alpha s} T(s)) = \int_0^t \alpha \mu_T e^{\alpha s} ds + \sigma_T \int_0^t e^{\alpha s} dW_s^{(1)} \quad (12)$$

$$e^{\alpha t} T(t) - T_0 = \alpha \mu_T \int_0^t e^{\alpha s} ds + \sigma_T \int_0^t e^{\alpha s} dW_s^{(1)} \quad (13)$$

then,

$$e^{\alpha t} T(t) = T_0 + \mu_T (e^{\alpha t} - 1) + \sigma_T \int_0^t e^{\alpha s} dW_s^{(1)} \quad (14)$$

If we divide both sides of the equation (14) by $e^{\alpha t}$, then we got the initial version of the semi-analytical form of the $T(t)$ function.

$$T(t) = T_0 e^{-\alpha t} + \mu_T (1 - e^{-\alpha t}) + \sigma_T \int_0^t e^{-\alpha(t-s)} dW_s^{(1)} \quad (15)$$

By performing the same operations for the other two functions in the system of stochastic differential equations (2) - (3), we obtain the following results:

$$\begin{aligned} M(t) &= M(0)e^{-\beta t} + \mu_M(1 - e^{-\beta t}) + \\ &+ \sigma_M \int_0^t e^{-\beta(t-s)} dW_s^{(2)} \end{aligned} \quad (16)$$

$$\begin{aligned} D(t) &= D(0)e^{-\gamma t} + \mu_D(1 - e^{-\gamma t}) + \\ &+ \sigma_D \int_0^t e^{-\gamma(t-s)} dW_s^{(3)} \end{aligned} \quad (17)$$

In Eqs.(15)-(17), the differential equations governing $T(t)$, $M(t)$ and $D(t)$ were expressed in a semi-analytical form, incorporating stochastic integrals of the type $\int_0^t e^{-K(t-s)} dW_s^{(i)}$, it is well known that such integrals generally do not possess closed-form solutions. Consequently, to enable simulation and parameter forecasting, it becomes necessary to approximate these stochastic integrals numerically.

6. Discretization of stochastic integrals using the Euler–Maruyama scheme

In this study, the Euler–Maruyama scheme was used to discretize stochastic convolution equations. This method is known as one of the most fundamental and widely used numerical approaches to stochastic differential equations and is particularly effective in systems controlled by the Wiener process. The Euler–Maruyama method – (G.Maruyama, 1955) is a simple and widely used approach for numerically solving stochastic differential equations (SDEs). It is used to calculate approximate solutions to stochastic differential equations based on the Ito definition. The application of this approach when modeling the random variability of Parkinson's disease symptoms is justified both from a mathematical point of view and from the point of view of the nature of the biological system.

As shown in the literature, the Euler–Maruyama method provides sufficiently accurate results for models of simple and moderate complexity, representing biophysical and physiological processes (Higham, 2001; Kloeden & Platen, 1992). This is explained by its low computational complexity, high computational speed, and practical ease of application.

Alternatively, stochastic integration methods of the Milstein and Runge–Kutta types provide higher accuracy, but these methods are preferable in cases where the diffusion coefficients depend on the state. However, in this model, the stochastic variability of the system is expressed by fixed parameters.

Therefore, the Euler–Maruyama method is considered to be the optimal choice from a theoretical and practical point of view. In addition, the simplicity of the Euler–Maruyama method also provides an advantage in its hybridization with deep learning. Thus, this method is suitable for building models that take into account the stochastic nature of neurodegenerative processes in terms of both computational efficiency and mathematical stability.

Table 4 below shows a comparative analysis of numerical solution methods for stochastic integrals.

Table 4. Description of the analysis of numerical solution methods for stochastic integrals

Method	Accuracy	Stability	Suitability for stochastic integrals
Euler-Maruyama method	$O(\sqrt{\Delta t})$	Moderate	Suitable
Milstein method	$O(\Delta t)$	High	Depending on the diffusion state
Runge-Kutta method	$O(\Delta t^2)$	High	Not suitable

The Euler–Maruyama method offers a first-order strong approximation to Itô integrals and is particularly effective for processes driven by Brownian motion with Lipschitz-continuous coefficients. Specifically, for a time grid $t_n = n\Delta t$ The integral,

$$\int_0^{t_n} e^{-K(t_n-s)} dW_s^{(i)} \quad (18)$$

is approximated as,

$$\sum_{z=0}^{n-1} e^{-K(t_n-t_z)} \Delta W_z^{(i)}, \Delta W_z^{(i)} \sim \mathcal{N}(0, t-s) \quad (19)$$

This approximation allows us to simulate the time evolution of each physiological variable while preserving both the deterministic trend and the stochastic variability inherent in Parkinsonian symptom progression. Thus, the stochastic integrals in Eqs.(15)-(17) should be found by the following approximate method:

$$\int_0^t e^{-\alpha(t-s)} dW_s^{(1)} \approx \sum_{z=0}^{N-1} e^{-\alpha(t-t_z)} \Delta W_z^{(1)} \quad (20)$$

$$\int_0^t e^{-\beta(t-s)} dW_s^{(2)} \approx \sum_{z=0}^{N-1} e^{-\beta(t-t_z)} \Delta W_z^{(2)} \quad (21)$$

$$\int_0^t e^{-\gamma(t-s)} dW_s^{(3)} \approx \sum_{z=0}^{N-1} e^{-\gamma(t-t_z)} \Delta W_z^{(3)} \quad (22)$$

If we substitute the approximate forms (20)-(22) of the stochastic Itô integrals found by the Euler-Maruyama method into Eqs.(15)-(17), we obtain the following final results.

$$T(t) = T(0)e^{-\alpha t} + \mu_T(1 - e^{-\alpha t}) + \sigma_T \sum_{z=0}^{N-1} e^{-\alpha(t-t_z)} \Delta W_z^{(1)} \quad (23)$$

$$M(t) = M(0)e^{-\beta t} + \mu_M(1 - e^{-\beta t}) + \sigma_M \sum_{z=0}^{N-1} e^{-\beta(t-t_z)} \Delta W_z^{(2)} \quad (24)$$

$$D(t) = D(0)e^{-\gamma t} + \mu_D(1 - e^{-\gamma t}) + \sigma_D \sum_{z=0}^{N-1} e^{-\gamma(t-t_z)} \Delta W_z^{(3)} \quad (25)$$

7. Experimental results

The proposed approaches were implemented on a 13th Gen Intel(R) Core(TM) i7-13700H processor with quad-core architecture running at 2.50 GHz, supported by 16 GB RAM and an NVIDIA GeForce RTX 3050 graphics card with 16 GB dedicated memory. The experiments were conducted in the IPython Notebook environment using the Python 3 kernel, leveraging libraries such as NumPy, SciPy, TensorFlow, and Matplotlib for numerical computation, machine learning, and visualization. All simulations were executed under a Windows 11 operating system, ensuring a stable and efficient computational framework. The described setup provided sufficient computational resources to handle stochastic simulations, deep learning model training, and optimization processes within a reasonable timeframe.

The values of the parameters predicted by the LSTM model were substituted into the Eqs.(23)-(25) obtained during the semi-analytical solution of the considered stochastic differential equations system (2)-(3). Here, the values of $T(t)$ - a lower and upper bound finite continuous function expressing the patient's tremor intensity, $M(t)$ - a lower and upper bound also finite continuous function shows the motor function index in a normalized scale, and $D(t)$ - expressing the patient's dopamine level over the postoperative milestone were found within the initial conditions $T(t) = 5.0$, $M(t) = 0.6$ and $D(t) = 0.5$. As a result of the semi-analytical experiment, we have obtained $D(t) = 0.526011$ final values that contain a more exact solution.

During the solution by numerical methods for stochastic integrals, the results were realized in the iterative process with a step that is shown below:

$$\tau = \Delta t = 0.01, t_z = z \cdot \tau \\ \varpi_\tau = \{t_z \in [0, T_{max}], z = 0, \dots, N-1\} \quad (27)$$

Table 5 shows the values of the functions found by the numerical method and the values found by the analytical method after 1000 iterations, and then the error was estimated.

Table 5. Semi-analytical solution of the system of stochastic differential equations.

Step	Functions	Semi-analytical solution	Absolute error
t=9.96	T(t)	4.956602	0.081449
	M(t)	0.616377	0.006545
	D(t)	0.510663	0.024226
t=9.97	T(t)	4.976234	0.061817

	$M(t)$	0.612500	0.002667
	$D(t)$	0.516077	0.029639
$t=9.98$	$T(t)$	5.005971	0.032080
	$M(t)$	0.605987	0.003846
	$D(t)$	0.515431	0.028994
$t=9.99$	$T(t)$	5.025779	0.012273
	$M(t)$	0.614055	0.004222
	$D(t)$	0.526320	0.039883
$t=10.00$	$T(t)$	5.016415	0.021637
	$M(t)$	0.607107	0.002726
	$D(t)$	0.526011	0.039574

In Fig. 2, the graphical representation of the $T(t)$ – is a finite continuous function expressing the patient’s tremor intensity, $M(t)$ - is also a finite

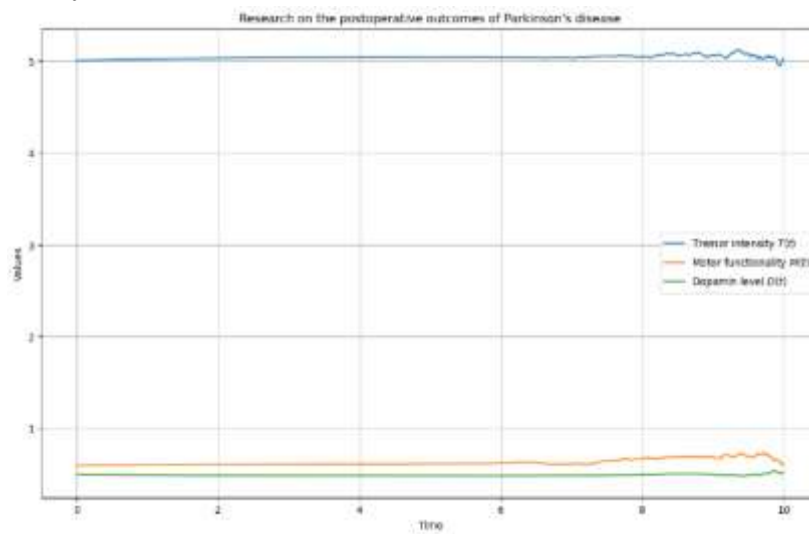


Fig 2. Graphical representation of symptom dynamics

8. Conclusion and future works

In this study, a novel hybrid modeling approach was developed to predict the key symptomatic parameters of Parkinson’s disease – including tremor intensity, motor functionality, and dopamine levels – using a genetically optimized LSTM neural network. The predicted parameters were integrated into a system of stochastic differential equations based on Ornstein–Uhlenbeck dynamics, which were then solved using a semi-analytical method where the stochastic integrals were approximated via the Euler–Maruyama scheme. The results demonstrated reduced stochastic fluctuations over time, indicating a stabilizing trend in the disease progression, which can be interpreted as a favorable physiological response to treatment. The combined use of machine learning and stochastic modeling provides a powerful and interpretable

continuous function, shows the motor function index in a normalized scale, and $D(t)$ - expressing the patient’s dopamine level over the postoperative milestone is given below. When analyzing the graphical representation, we can see that the stochastic fluctuations in the solutions of the differential equations have significantly diminished over time. This indicates a desirable outcome, as it reflects a trend toward physiological stability, suggesting that the modeled symptoms – such as tremor intensity, motor function, and dopamine level – are gradually stabilizing. Such behavior is consistent with effective therapeutic intervention and supports the reliability of the parameter estimation process used in the model.

framework for analyzing the post-treatment dynamics of neurodegenerative diseases.

In future work, we aim to extend the proposed software into a fully integrated medical device, tentatively named *NeuroTrack+*, which combines wearable biosensors and embedded AI. This device would continuously collect real-time data on motor activity, tremor frequency, and neurochemical markers through non-invasive monitoring. The embedded version of our AI-based forecasting model will process the data onboard to provide personalized parameter estimation and real-time disease progression analysis. Such a system would not only assist clinicians in evaluating the effectiveness of treatment but also offer early warnings for potential relapse or symptom aggravation. Additionally, further research will explore the use of higher-order numerical methods and alternative stochastic modeling frameworks to improve accuracy and interpretability in patient-

specific applications.

As a result, in the future, this approach will stimulate the development of innovative, patient-centered neurodiagnostic devices and engineering solutions in both medicine and biomedical technology, laying the foundation for intelligent systems that can significantly enhance the quality of life and long-term care of individuals affected by Parkinson's disease.

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