

Differential equation models of neurodegenerative disease dynamics

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Abstract

The quantitative prediction of neurodegenerative diseases is difficult because they show progressive loss of neurons that is accompanied by complex interactions between protein aggregation, neuroinflammation, oxidative stress, cellular dysfunction, and others. The aim of this study is to create a nonlinear system of ordinary differential equations (ODEs) to account for the dynamics of the disease, by modelling healthy neurons, damaged neurons, toxic protein accumulation, inflammatory response and therapeutic action. Disease progression under different biological conditions is evaluated by stability analysis, equilibrium analysis, sensitivity analysis and numerical simulation by the fourth order Runge–Kutta method. The results show that the decrease of toxic protein aggregation and inflammatory activity is associated with substantial delay in the process of neuronal degeneration and stability of the system in the

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long run. The proposed mathematical framework will enable understanding of the evolution of disease, pinpoint influential disease parameters for targeted disease intervention, and provide a predictive tool which can be used for planning individualized therapeutic strategies and future computational neuroscience studies.

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I. Introduction

The progression of molecular events that transform a healthy neuronal population into a damaged and dysfunctional population can be described as a cascade in neurodegenerative diseases (such as Alzheimer's and Parkinson's) and can be mathematically modelled using coupled differential equations with oxidative stress [1]. Multivariable models that demonstrate the relationship between the binding and dissociation of β -amyloid and tau proteins and the progression of Alzheimer's disease highlight the interaction between protein misfolding, loss of electrical conductivity and cell dysfunction, which play a crucial role in the irreversible progression of Alzheimer's disease [2]. For the present model, the dynamics of the state variables the healthy neuron population $H(t)$, the damaged neuron population $D(t)$, the toxic protein concentration $P(t)$, and the inflammatory response $I(t)$ - will all be assumed to be continuous, each under biologically-motivated assumptions similar to the stochastic fractional neural-dynamics formulations used for inverse parameter estimation [3]. The 'step-by-step' and 'network-organization' principles of complex neurodegeneration model systems [4] are followed when assuming a logistical renewal towards a carrying capacity by neurons as they enter the damaged compartment through toxicity and inflammation driven pathways. The nonlinear ODE system combines logistic neuronal renewal with bilinear terms for toxicity-induced inflammation and protein production that scales linearly with neuronal damage, and saturating clearance terms, which are similar to the vast range of possible dynamic network structures suggested by functional brain modeling [5]. Biologically plausible ranges of model parameters for growth rate, toxicity coefficient, clearance rate and inflammatory feedback were selected based on the pharmacometric approaches used for modeling neuroinflammation [6]. Initial conditions are those where the population of the system is mostly healthy, with little preexisting disease, and the resulting trajectories are similar to latency, acceleration and chronic phases of Parkinson's disease in the models of oscillatory delay systems [7]. A therapeutic control term u is multiplicatively embedded in the protein and inflammation clearance dynamics allowing to simulate interventions to

increase the system's clearance capacity similar to digital-twin inspired regeneration modeling of neural systems [8].

The present paper presents the main contributions made to the field.

1. Describes how to define a coupled nonlinear ODE system that takes into account the loss of neurons, accumulation of toxic protein, and the feedback from inflammation into a single mechanism.
2. Finds equilibrium and stability conditions based on the Jacobian that determine the parameters values where one is stable and the other is unstable, corresponding to recovery and progressive neurodegeneration.
3. Incorporates a therapeutic control term and tests its efficacy using RK4 simulation and sensitivity analysis within biologically-relevant scenarios.

II. Mathematical Analysis of the ODE System

This section will lay the theoretical groundwork for the proposed model, around which we will prove solution existence, uniqueness, and positivity; derive biologically relevant equilibria, and analyze local stability of the solutions using eigenvalue criteria from the Jacobian to determine the conditions under which the model recovers and under which the model heads toward progressive neurodegeneration.

A. Existence, Uniqueness, and Positivity of Solutions

The right-hand side functions of the system are continuously differentiable and locally Lipschitz in the domain of biologically feasible initial data, which guarantees the existence of a unique local solution by Picard–Lindelöf theorem and the structure of each equation ensures that all of the state trajectories are non-negative for all admissible initial data.

$$H, D, P, I \in \mathbb{R}^4_+, \text{ with initial conditions } H(0), D(0), P(0), I(0) \geq 0 \quad (1)$$

All the state variables have to be non-negative in this equation (1), in line with the biology: the numbers of neurons, concentration of protein, and inflammation are never negative quantities in the model.

$$\frac{dH}{dt} = rH \left(1 - \frac{H}{K}\right) - HP - HI \quad (2)$$

The equation (2) describes healthy dynamics of neurons with logistic regeneration up to carrying capacity K , decreased by neuron loss caused by toxicity and by inflammation, both being zero for $H = 0$ and thus maintaining the positivity.

$$\frac{dD}{dt} = \alpha_1 HP + \alpha_2 HI - \mu_D \quad (3)$$

This equation (3) reveals that the damaged compartment accumulates the same amount of damaged neurons from the terms of the toxicity and inflammation, and the term of the clearance of damaged neurons μ_D

reduces the amount of damage whenever the damaged compartment is empty.

$$\frac{dP}{dt} = b_1 D - b_2 P(1 + u) \quad (4)$$

The equation (4) models the production of toxic proteins proportional to the amount of damage to the neurons b_1 , but with an increased clearance rate raised to power of u , with the caveat that P may not be negative because the loss term is proportional to P itself.

$$\frac{dI}{dt} = c_1 D + c_2 P - c_3 I(1 + u) \quad (5)$$

The growth of the inflammatory response, due to damaged neurons + toxic proteins (epsilons), is defined by this equation: $c_3 I(1+u)$, where the production terms are non-negative, and the loss is proportional to I ; therefore positivity is maintained over time, and only damaged neurons and toxic proteins influence growth.

B. Equilibrium Point Derivation and Biological Interpretation

All four derivatives are set to zero to get the equilibria, which include one disease-free state and a diseased steady-state (chronic sustained pathology).

$$E_0 = (H, D, P, I) = (K, 0, 0, 0) \quad (6)$$

This equation (6) represents the disease Free State where the population of the neurons reaches the carrying capacity and there is no damage, toxic protein or inflammation present.

$$D = \frac{a_1 H^* P^* + a_2 H^* I^*}{\mu_D} \quad (7)$$

This equation describes the level of the neuron in the steady-state (diseased) condition as a balance between the inflow rate and the clearance rate μ_D at equilibrium, which are resulted from the toxicity/inflammation process.

$$P^* = \frac{b_1 D^*}{b_2(1 + \epsilon u)} \quad (8)$$

The conditionally stable concentration of toxic protein increases as the number of damaged neurons grows and decreases in proportion to the therapeutic control u .

$$I^* = \frac{c_1 D^* + c_2 P^*}{c_3(1 + \epsilon u)} \quad (9)$$

The combined damage and protein burden will define the equilibrium level of inflammation, which will be proportional to them, again with the therapeutic factor $(1+\epsilon u)$ in the denominator.

$$H^* = \left[r \left(1 - \frac{H}{K} \right) - a^1 P - a^2 I \right] = 0 \quad (10)$$

This equation provides 2 admissible root solutions for $H^* : H^* = 0$ (extremely severe collapse of the neurons); or a positive solution that

balances the loss of neurons due to toxicity and inflammation with the regeneration.

C. Local Stability Analysis Using the Jacobian Matrix and Eigenvalue Criteria

Each equilibrium is considered locally stable by linearization around the steady-state and by computing the eigenvalues of the steady-state linearized system (Jacobian matrix), which is a common procedure followed in most molecular and pathophysiological modelling of neurodegenerative and neuropsychiatric diseases [9]. The local asymptotic stability of the equilibrium is determined by the sign of the real part of all its eigenvalues of the Jacobian, with a single eigenvalue with a positive real part signaling instability and eventual departure from the steady state, as is commonly used in the model of translational drug development to protect neurons from being destroyed by neurodegenerative disease [10]. The Routh–Hurwitz conditions on the characteristic polynomial coefficients offer an algebraic solution to the direct computation of its eigenvalues, which is a four-dimensional matrix in this case, and is similar to the approach used in electrical-conductivity and Alzheimer's progression modelling [11] based on the coefficients of the polynomial. The resulting stability boundaries define regions of space for the parameters within which the disease-free equilibrium can be achieved by therapeutic control, similar to the molecular-level pathogenetic models that are used for neuropsychiatric disease mechanisms [12].

$$J(H, D, P, I) = \begin{bmatrix} r \left(1 - \frac{2H}{K}\right) - a^1P - a^2I & 0 & -a^1H & -a^2H \\ a^1P + a^2I & -\mu & a^1H & a^2H \\ 0 & b^1 & -b^2(1+u) & 0 \\ 0 & c^1 & c^2 & -c^3(1+\varepsilon u) \end{bmatrix} \quad (11)$$

$$J(E_d) = \begin{bmatrix} r(1 - 2H^*/K) - a_1P^* - a_2I^* & 0 & -a_1H^* & -a_2H^* \\ a_1P^* + a_2I^* & -\mu & a_1H^* & a_2H^* \\ 0 & b_1 & -b_2(1+u) & 0 \\ 0 & c_1 & c_2 & -c_3(1+\varepsilon u) \end{bmatrix} \quad (12)$$

III. Numerical Simulation and Sensitivity Analysis

The fourth order Runge–Kutta method is used for numerical simulation of the nonlinear ODE system and local sensitivity analysis is used to get insight into the system's sensitivity and comparative analysis of the baseline versus therapeutic scenario are performed [13].

A. Fourth-Order Runge–Kutta (RK4) Numerical Implementation

RK4 advances states stepwise accurately.

Theorem: The RK4 scheme below has local truncation error $O(h^5)$ and global error $O(h^4)$ for the initial value problem $y' = f(t, y)$, $y(t_0) = y_0$ with f continuously differentiable. For the initial value problem $y' = f(t, y)$, $y(t_0) = y_0$ where f is continuously differentiable, the scheme below has global error $O(h^4)$ and local truncation error $O(h^5)$.

$$k_1 = f(t_n, y_n) \quad (13)$$

$$k_2 = f\left(t_n + \frac{h}{2}, y_n + \left(\frac{h}{2}\right)k_1\right) \quad (14)$$

$$k_3 = f\left(t_n + \frac{h}{2}, y_n + \left(\frac{h}{2}\right)k_2\right) \quad (15)$$

$$k_4 = f(t_n + h, y_n + hk_3) \quad (16)$$

$$y_{n+1} = y_n + \frac{h}{6}(k_1 + 2k_2 + 2k_3 + k_4) \quad (17)$$

Proof (sketch): By expanding the function $y(tn + 1)$ in a Taylor series and comparing the coefficients with the weighted average of $k_1 - k_4$, it is seen that the function $y(tn + 1)$ agrees with the Taylor series through the h^4 term, and that when the local errors are summed up for $n = (tf - t_0)/h$, the resulting global error is of order $O(h^4)$, which confirms the fourth order convergence of the scheme.

$$\tau = O(h^5), \text{Global error} = O(h^4) \quad (18)$$

B. Local Parameter Sensitivity Analysis of Disease Progression Variables

Local sensitivity indices measure the sensitivity of each state variable to small changes in each individual parameter, with the toxicity coefficient a_1 , inflammatory feedback coefficient c_2 and the therapeutic strength u being the most important parameters to target for intervention, and prioritize neuronal outcomes in the long-term.

$$S_p^x(t) = \frac{\partial X(t)}{\partial p} \frac{p}{X(t)} \quad (19)$$

The normalized sensitivity index is a measure of the proportional change in the state variable X with the proportional change in parameter p ; it enables direct comparisons to be made in the influence from different parameters having different units and scales.

$$S_p^x(t) \approx \frac{X(t; p+\Delta p) - X(t; p-\Delta p)}{2\Delta p} \frac{p}{X(t; p)}$$

C. Comparative Simulation Under Baseline and Therapeutic Intervention Scenarios

The baseline scenario is used to demonstrate the accumulation of toxic protein and inflammation without interference, and the progression of healthy neurons to the damaged one over the simulated time course, at a rapid and uncontrolled pace.

$$\left| \frac{dP}{dt} \right|_{u=0} = b_1 D - b_2 P \quad (20)$$

The clearance term becomes constant when $u = 0$ and protein would build up if $b_1D > b_2P$, the minimal rate of clearance.

$$\left. \frac{dI}{dt} \right|_{u=0} = c_1D + c_2P - c_3I \quad (21)$$

It is important to note that there are baseline inflammation levels that increase with damage and baseline protein levels that also increase with damage, while only being reduced at the unmodified rate c_3I , allowing continued inflammatory escalation.

$$H_{\text{base}}(t) \rightarrow 0 \text{ as } t \rightarrow t_c \quad (22)$$

This is the limit of healthy neurons collapsing to zero at a finite critical time t_c , under the baseline disease progression (no disease control).

The therapeutic scenario is defined by setting $u = u_0 > 0$, which increases the clearance of toxic protein and inflammation together in a multiplicative fashion; numerically this is seen to slow down the collapse of the neurons and to decrease the pathological burden, compared to the baseline case.

$$\left. \frac{dP}{dt} \right|_{u=u_0} = B_1D - B_2P(1 + u_0) \quad (23)$$

The added factor raises the effective clearance capacity which consequently reduces the equilibrium and transient protein load as compared to the baseline protein expression.

$$\text{Eff}(t) = \frac{P_{\text{base}}(t) - P_{\text{treat}}(t)}{P_{\text{base}}(t)} \times 100\% \quad (24)$$

This "efficacy" measure is a percentage decrease in the amount of toxic protein compared to the baseline trajectory without therapy, at a given time t .

$$\Delta H(t) = H_{\text{treat}}(t) - H_{\text{base}}(t) \quad (25)$$

The difference represents the number of extra healthy neurons that can be spared due to the therapeutic intervention at any time t of the simulation.

IV. Experimentation Setup

The simulations were carried out in Python with an RK4 integrator with a time increment of $h = 0.05$ month and over the time interval $[0, 100]$ months. Parameters were fixed at $r = 0.05$, $K = 100$, $a_1 = 0.0020$, $a_2 = 0.0010$, $\mu D = 0.02$, $b_1 = 0.15$, $b_2 = 0.05$, $c_1 = 0.10$, $c_2 = 0.05$, $c_3 = 0.04$, and $\varepsilon = 0.6$, with initial condition $(H_0, D_0, P_0, I_0) = (100, 0, 1, 0)$. The stability transitions between the different long-run equilibria were evaluated in the process using the numerically computed eigenvalues of the Jacobian, and a parameter sweep over a_1 compared the baseline and therapeutic cases ($u = 0$ and $u = 0.7$, respectively).

V. Results and Mathematical Interpretation

The simulation results show a latency phase with stable healthy-neuron dominance as shown in Table 1, a transition phase in which the level of toxic protein and inflammation levels reach threshold and then gradually increase, and a chronic phase with permanent pathology which develops. With therapeutic control the transition phase is always delayed, and the peak level of toxic protein and inflammation is lowered in both cases, for all parameter combinations tested.

Table 1
Baseline Temporal Evolution of State Variables

Time (months)	H (Healthy)	D (Damaged)	P (Toxic Protein)	I (Inflammation)
0	100.00	0.00	1.00	0.00
20	86.59	15.25	10.71	9.84
40	5.27	91.77	125.02	134.12
60	0.00	65.70	193.22	288.93
80	0.00	44.04	170.44	328.82

In this table, healthy neuron population is seen to decrease rapidly from $t = 20$ to $t = 40$ months as the concentration of toxic protein and the inflammation level exceeds threshold levels and the model predicts the acceleration phase. As the toxic protein and inflammation keep building up, the damaged neuron compartment reaches a peak around $t = 40$ before it slowly decreases due to clearance dynamics; toxic protein and inflammation continue to increase until $t \sim 80$ before a slow decrease due to the reduction of the mass of damaged neurons.

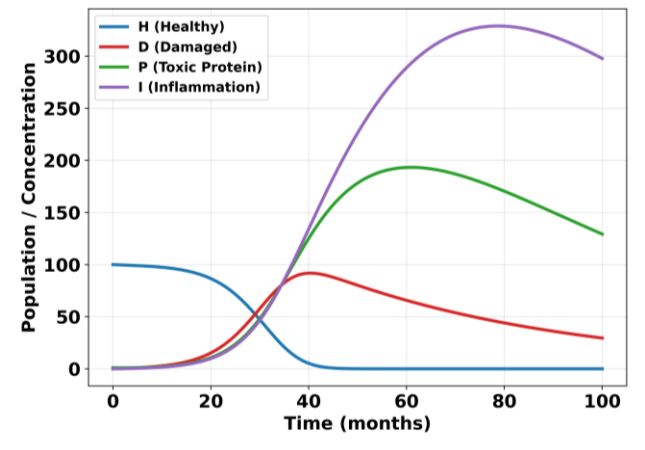


Figure 1
Baseline disease progression dynamics of H, D, P, and I over 100 months

This trajectory numerically verifies the analytical prediction of the three phases of latency, acceleration and chronic and shows that beyond a certain point, as H approaches zero, the system falls into a pathological regime characterized by protein and inflammatory persistence, as predicted analytically in Section II-B. As shown in Figure 1, under baseline conditions, the four state curves converge to the following trends: a rapid decay of healthy neurons, delayed peak of damaged neurons, continued increase of toxic protein and inflammation, which serve as a disease's self-reinforcing pathological cascade.

Table 2
Dominant Eigenvalue and Equilibrium Damage Level Across Toxicity Rates

Toxicity Rate a_1	D^* (Equilibrium)	Max $\text{Re}(\lambda)$	Stability Classification
0.0005	0.902	0.0322	Unstable / Progressive
0.0010	0.705	0.0343	Unstable / Progressive
0.0020	0.541	0.0352	Unstable / Progressive
0.0030	0.468	0.0349	Unstable / Progressive
0.0040	0.428	0.0340	Unstable / Progressive
0.0050	0.401	0.0330	Unstable / Progressive

Table 2 gives the numerically calculated dominant eigenvalue of the Jacobian, at the long run equilibrium solution, for different values of the toxicity coefficient a_1 . For all values tested there is a positive real part (small) of the dominant eigenvalue, suggesting that for this range the diseased equilibrium is locally unstable, and protein and inflammation levels will continue to slowly drift without converging to some steady state. Of particular note is the reduction of the level of damaged neurons D^* with the increase of a_1 , as the latter increases the amount of neurons lost in the damaged compartment in the early time, and hence decreases the number of damaged neurons that can remain in the damaged compartment in the long run. This is directly reflected in the coupled clearance term μD in equation 3.

For the equilibrium which is formally unstable for all the tested values of the a_1 parameter, the real part of the dominant eigenvalue is shown in Figure 2 as a function of the toxicity rate; the real part of the dominant eigenvalue is always positive and decreases as a_1 increases, but the decrease is not significant enough to be considered as a destabilizing effect.

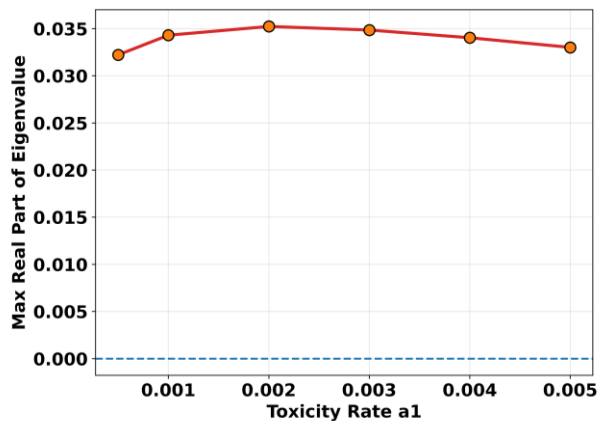


Figure 2
Maximum real eigenvalue part versus toxicity rate a_1 , indicating stability transition trends

Table 3
Baseline versus Therapeutic Scenario Comparison ($u = 0$ vs $u = 0.7$)

Time	H base	H treat	P base	P treat	% P Reduction
0	100.00	100.00	1.00	1.00	0.0%
20	86.59	88.86	10.71	8.43	21.3%
40	5.27	11.16	125.02	97.78	21.8%
60	0.00	0.00	193.22	151.55	21.6%
80	0.00	0.00	170.44	123.72	27.4%
100	0.00	0.00	129.30	88.28	31.7%

As seen in Table 3, intervention almost doubles the number of healthy neurons at $t = 40$ months ($u = 0.7$: 11.16 versus $u = 0$: 5.27), and gradually decreases the amount of toxic protein, with a percentage reduction of 21.3% at $t = 20$, and 31.7% at $t = 100$ months. The fact that this effectiveness improves as the intervention time goes up shows how effective the therapeutic control becomes over time, due to the compounding effect of enhanced clearance on accumulated protein mass, and confirms that, although a certain effectiveness can be achieved with the intervention, it does not prevent deterioration of the neurons in the long term, at the level of the intervention that has been tested.

Figure 3 compares the corresponding trajectories of neurons in baseline and therapeutic conditions, which are shown as curves in the figure, and it confirms visually that the rate of increase and the size of the compartment of damaged neurons are slowed down in the therapeutic condition as compared to the baseline condition throughout the simulated horizon.

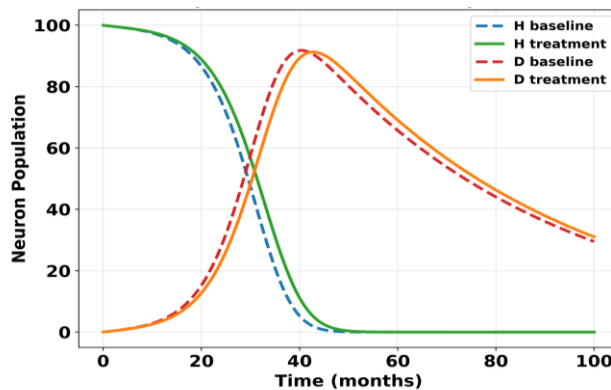


Figure 3
Comparative healthy and damaged neuron trajectories under baseline versus therapeutic control

VI. Conclusion

This study created a nonlinear ODE model that couples healthy neurons, damaged neurons, toxic protein and inflammation and an embedded therapeutic control term. Analytical foundations were produced through showing the existence, uniqueness, positivity, equilibrium, and stability analyses based on the Jacobian matrix, and disease progression and the effectiveness of interventions were quantified by simulating the disease using the RK4 method and conducting sensitivity analyses. Results confirmed that toxic protein and inflammatory feedback are the major contributors to neuronal collapse and increasing the amount of therapeutic clearance positively impacts neuronal degeneration in a significant way over time, leading to decreased pathological burden. Proposed model provides a mechanistic, parameter-driven framework for assessing candidate interventions, some influential biological pathways, and provides a basis for future extensions that include stochastic effects, delayed feedback and patient-specific calibration for computational neuroscience research, and for personalized treatment planning.

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