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A Mathematical Model of Borderline Personality Disorder

Betty Chebet, Onyango Lawrence Omondi*, Elisha Achieng Ogada

Department of Mathematics, Egerton University, Nakuru, Kenya
Email: bettychebet101@gmail.com, *lonyango@egerton.ac.ke, eogada@egerton.ac.ke

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Abstract

In this article, a dynamical system model of Borderline Personality Disorder (BPD) has been developed and analyzed. The model takes into account the instantaneous effective responses, the emotional instability, difficulties in relationships and impulsive behaviors. A minimum linear model for parameter estimation followed by nonlinear generalization with Hill function based self excitations and social reinforcements are introduced. The derivation of the model, its stability and bifurcation analyses, and stimulation results are presented, showing how feedbacks are responsible for elevated levels of symptoms severity under transient stress conditions and how variations of treatment parameters affects attractor dynamics. The article is concluded with a guide to model parametrization based on intensive longitudinal measures, parents daily experiences, highlighting potential issues and giving future directions for computational psychiatry.

Keywords

Borderline Personality Disorder, Bifurcation, Attractor Dynamics, Emotional Instabilities, Persistence of Symptoms, Transient Stress and Affective Reactivity

1. Introduction

Borderline Personality Disorder (BPD) is identified by pervasive emotional instability, difficulties in interpersonal relationships, impulsivity, and identity disturbance [1]. Models of this type serve as a basis for understanding how to make quantitative connections between psychological variables and dynamical processes that cause symptom trajectories. Significant advances in using computational and dynamical models to study psychiatric conditions occurred within the last decade, giving rise to a quantitative approach that explains both symptoms and their un-

derlying mechanisms. The development of computational psychiatry began with reinforcement learning, predictive coding, and connectivity modeling, which explained symptoms associated with conditions like BPD [2]-[4]. These approaches highlighted how latent feedback loops and maladaptive learning processes could explain emotional instability, impulsivity, and self-harm behavior observed clinically.

Network models of psychiatric symptoms have become particularly influential, conceptualizing disorders as complex systems of mutually interacting symptoms rather than monolithic latent constructs [5] [6]. Such network approaches align naturally with dynamical systems modeling, individual nodes (e.g., affective dysregulation, interpersonal conflict, impulsivity) are coupled via directional or bidirectional interactions, allowing for the emergence of multistability, hysteresis, and threshold effects [7] [8] These models can capture phenomena such as rapid escalation of symptoms following minor stressors and slow recovery despite the removal of triggers patterns commonly observed in BPD [9] [10]. Empirical research using ecological momentary assessment (EMA) has been pivotal in linking theory to real-world dynamics. High-frequency longitudinal data reveal that affective reactivity in BPD occurs on timescales of hours or even minutes, necessitating models that can resolve rapid state changes [11] [12]. EMA-based studies also support the presence of self-reinforcing loops: intense affective responses often trigger interpersonal conflicts, which in turn amplify dysregulation, creating a feedback cascade that linear models alone cannot fully capture [13] [14]. Recent work further emphasizes modeling interpersonal dynamics computationally, highlighting how social feedback loops contribute to symptom persistence and escalation [15].

Mathematical analyses of nonlinear dynamical systems have demonstrated how Hill-type or sigmoidal self-reinforcing mechanisms generate bistable or multistable behavior [16] [17]. Such analyses provide a principled way to relate observed symptom trajectories to underlying feedback parameters, offering insight into why some patients exhibit persistent high-symptom states while others recover spontaneously. Saddle-node bifurcations, hysteresis, and attractor basins have all been observed in toy models and more detailed network simulations of psychiatric disorders [7] [18]. Computational modeling has also informed intervention strategies. Dialectical Behavior Therapy (DBT) and other emotion-regulation-focused therapies exert their effects by weakening maladaptive feedback loops, effectively shifting system trajectories toward healthier attractors [19] [20]. In silico experiments using network and dynamical systems models have explored how altering coupling strengths or decay rates can predict the efficacy of targeted interventions, guiding both therapeutic design and personalized treatment planning [5] [21]. Moreover, hierarchical and Bayesian approaches allow the estimation of individual-specific parameters from EMA or trial data, providing a pathway toward precision computational psychiatry [22] [23].

Recent reviews emphasize the integration of linear and nonlinear approaches,

where linear models provide identifiability and parameter estimation advantages, while nonlinear extensions capture complex clinical phenomena such as escalation, relapse, and persistent dysregulation [13] [24]. This hybrid strategy starting with parsimonious linear models and progressively introducing nonlinear feedback has become a practical standard in dynamical systems psychiatry. This paper is organised as follow: In Introduction we have looked at borderline personality disorder, its definition and symptoms. In Section two, we have formulated the model and analyzed it. An analysis was also done in section three. In Section four, Section five has simulation results, Section six has discussion and finally, conclusion is section seven.

2. Model Formulation and Description

In our study, we develop a four-dimensional dynamical system model that includes affect (A), emotional dysregulation (B), interpersonal problems (C1), and impulsivity (C2). We examine both a minimal linear model, which is used for identifying parameter values, as well as a more complex nonlinear extension that describes persistence due to self-reinforcing mechanisms. Our aim is to find local stability criteria analytically, show by simulations how perturbations can change trajectories, and provide a practical tool for parameter estimation using EMA data.

2.1. State Variables and Interpretation

Let

$$x(t)=\begin{pmatrix} A(t) \\ B(t) \\ C1(t) \\ C2(t) \end{pmatrix} \tag{1}$$

with

- $A(t)$: momentary affective intensity (reactivity),
- $B(t)$: emotional dysregulation (higher = greater dysregulation),
- $C1(t)$: interpersonal difficulties (conflict, instability),
- $C2(t)$: impulsivity (propensity or frequency of impulsive acts).

Time units: days. Variables may be normalized to $[0,1]$ or standardized.

Table 1. Model parameters and baseline values.

Parameter	Meaning	Value (baseline)	Units / notes
λ_A	Affect decay rate	1.0	day^{-1}
λ_B	Dysregulation decay rate	0.15	day^{-1}
λ_{C1}	Interpersonal decay	0.05	day^{-1}
λ_{C2}	Impulsivity decay	0.3	day^{-1}
ρ_A	Stress sensitivity	0.8	A per S
β_{AB}	B → A coupling	0.35	A per B

Continued

α_{BA}	A → B coupling	0.5	B per A
α_{C1B}	B → C1 coupling	0.45	C1 per B
α_{C2B}	B → C2 coupling	0.6	C2 per B
μ_B	B Hill strength	0.4	B/day
θ_B	B Hill half-saturation	1.0	B units
p	B Hill exponent	2	dimensionless

2.2. Linear Model

We first propose a minimal linear ODE system:

$$\begin{aligned}
\frac{\mathrm{d}A}{\mathrm{d}t} &= -\lambda_A A + \rho_A S(t) + \beta_{AB} B \\
\frac{\mathrm{d}B}{\mathrm{d}t} &= -\lambda_B B + \alpha_{BA} A - \tau_B T(t) \\
\frac{\mathrm{d}C1}{\mathrm{d}t} &= -\lambda_{C_1} C1 + \alpha_{C_1 B} B \\
\frac{\mathrm{d}C2}{\mathrm{d}t} &= -\lambda_{C_2} C2 + \alpha_{C_2 B} B.
\end{aligned} \tag{2}$$

Here $S(t)$ denotes external stress input, and $T(t)$ denotes therapeutic input (e.g., DBT skills practice). The linear formulation is attractive for initial parameter estimation and provides analytic stability criteria.

In compact matrix form:

$$\dot{x} = Mx + Gu(t), \tag{3}$$

with

$$M = \begin{pmatrix} -\lambda_A & \beta_{AB} & 0 & 0 \\ \alpha_{BA} & -\lambda_B & 0 & 0 \\ 0 & \alpha_{C1B} & -\lambda_{C1} & 0 \\ 0 & \alpha_{C2B} & 0 & -\lambda_{C2} \end{pmatrix}, \quad u(t) = \begin{pmatrix} S(t) \\ T(t) \end{pmatrix},$$

and G collecting the input gains.

2.3. Nonlinear Extensions

To capture threshold effects and self-reinforcement:

$$\begin{aligned}
\frac{\mathrm{d}A}{\mathrm{d}t} &= -\lambda_A A + \rho_A S(t) + \beta_{AB} B \\
\frac{\mathrm{d}B}{\mathrm{d}t} &= -\lambda_B B + \alpha_{BA} A + \mu_B \frac{B^k}{B^k + \theta_B^k} - \tau_B T(t) \\
\frac{\mathrm{d}C1}{\mathrm{d}t} &= -\lambda_{C_1} C1 + \alpha_{C1B} B + \gamma_{C1} \frac{C1}{1 + \kappa_{C_1} C1} \\
\frac{\mathrm{d}C2}{\mathrm{d}t} &= -\lambda_{C_2} C2 + \alpha_{C2B} B + \mu_{C2} \frac{C2^n}{C2^n + \theta_{C2}^n}.
\end{aligned} \tag{4}$$

Hill-type terms introduce sigmoidal growth and potential bistability; the satu-

rating social feedback on P_1 models self-perpetuating interpersonal cycles.

3. Mathematical Analysis

We analyze equilibria and local stability for both linear and nonlinear systems.

3.1. Equilibria and Jacobian Matrices

For autonomous systems (constant or zero inputs), equilibria x^* satisfy $f(x^*)=0$. For the linear model, $x^*=0$ is the unique equilibrium when inputs are zero. For the nonlinear model, fixed points are solutions of nonlinear algebraic equations; analytic closed-form solutions are rarely available, but local stability can be assessed via linearization.

The Jacobian for the nonlinear system (without explicit time dependence) is:

$$J(x)=\begin{pmatrix} -\lambda_A & \beta_{AB} & 0 & 0 \\ \alpha_{BA}+\partial_B F_B(B)\cdot 0 & -\lambda_B+\partial_B F_B(B) & 0 & 0 \\ 0 & \alpha_{C1B} & -\lambda_{C1}+\partial_{C1}F_{C_1}(C1) & 0 \\ 0 & \alpha_{C2B} & 0 & -\lambda_{C2}+\partial_{C2}F_{C_2}(C2) \end{pmatrix}, \tag{5}$$

where $F_B(B)=\mu_B\dfrac{B^k}{B^k+\theta_B^k}$, etc. Explicit derivatives appear in diagonal corrections.

3.2. Local Stability Analysis (Linear Model)

Because the system matrix M is block-structured, its eigenvalues decompose partially. Consider the (A,B) sub-block:

$$M_{AB}=\begin{pmatrix} -\lambda_A & \beta_{AB} \\ \alpha_{BA} & -\lambda_B \end{pmatrix}. \tag{6}$$

The characteristic polynomial is

$$\chi_{AB}(\lambda)=\det(M_{AB}-\lambda I)=\lambda^2+(\lambda_A+\lambda_B)\lambda+(\lambda_A\lambda_B-\alpha_{BA}\beta_{AB}). \tag{7}$$

By the Routh-Hurwitz criterion, both eigenvalues have negative real parts if and only if

$$\lambda_A+\lambda_B>0 \text{ and } \lambda_A\lambda_B-\alpha_{BA}\beta_{AB}>0. \tag{8}$$

The first is automatically true for positive decay rates; the second yields the key instability condition:

$$\alpha_{BA}\beta_{AB}<\lambda_A\lambda_B. \tag{9}$$

If $\alpha_{BA}\beta_{AB}>\lambda_A\lambda_B$, the AB loop creates net positive feedback sufficient to destabilize the origin and produce oscillatory or divergent behaviors (depending on nonlinearities and inputs).

The eigenvalues for C1 and C2 decouple linearly and are $-\lambda_{C1}$ and $-\lambda_{C2}$; hence if decay rates are positive these directions are asymptotically stable in the absence of B, that is no emotional dysregulation.

3.3. Bifurcation and Multistability (Nonlinear Model)

Nonlinear Hill terms allow multiple positive fixed points for B. Consider the B-nullcline when $A=0$, $T=0$:

$$0=-\lambda_B B+\mu_B \frac{B^k}{B^k+\theta_B^k}. \tag{10}$$

One trivial root is $B=0$. Nontrivial roots satisfy

$$\lambda_B=\mu_B \frac{B^{k-1}}{B^k+\theta_B^k}. \tag{11}$$

For $k\geq 2$ and sufficiently large μ_B/λ_B, the right-hand side is non-monotonic with respect to B, producing up to two positive roots in addition to $B=0$ (*i.e.*, bistability). Saddle-node bifurcations occur when $f(B)=0$ and $f'(B)=0$ simultaneously; solving these gives critical parameter combinations that mark creation/annihilation of equilibria. Coupling to A shifts nullclines and can induce more complex bifurcations such as hysteresis and cusp points.

4. Simulation Methods

This chapter presents the computational method adopted, along with the numerical techniques of integration and parameters used in the simulation of the dynamic behavior of both the linear and non-linear versions of the BPD model. The computational method connects our theoretical stability analysis with practical dynamic scenarios.

4.1. Numerical Integration and Inputs

We simulated both the linear and nonlinear systems using a standard fourth-order Runge Kutta integrator with time step $\Delta t=0.01$ days across a 30-day horizon. Stress input $S(t)$ was modeled as three Gaussian-like pulses centered roughly at days 3, 10 and 18. Therapy input $T(t)$ was a step activated at day 12 to represent onset of DBT skills training in the simulation.

4.2. Parameter Choices

Baseline parameters (selected to illustrate regimes):

$$\lambda_A=1.0,\ \lambda_B=0.15,\ \lambda_{C1}=0.05,\ \lambda_{C2}=0.3,$$

$$\rho_A=0.8,\ \beta_{AB}=0.35,\ \alpha_{BA}=0.5,\ \alpha_{C1B}=0.45,\ \alpha_{C2B}=0.6,$$

$$\mu_B=0.4,\ \theta_B=1.0,\ k=2,\ \mu_{C2}=0.25,\ \theta_{C2}=0.8,\ n=2,$$

$$\gamma_{C1}=0.2,\ \kappa_{C1}=2.0,\ \tau_B=0.6.$$

These values generate dynamics where the linear model shows transient responses to stress pulses, while the nonlinear model can show sustained elevation of B, $C1$ and $C2$ following repeated stress depending on the initial conditions.

5. Results: Simulations, Graphs, and Tables

Here, we outline the findings of our simulation exercise in terms of time series and state value at end of time period for the linear as well as nonlinear system under stress and therapy, which show how far these two different models differ from each other in a quantitative manner. Table 1 has been employed to show the base value, definition, and units of parameters required for formulating the main mathematical model for BPD. On the other hand, Table 2 has been used to show the comparison between the quantitative values of the final states of affect, dysregulation, interpersonal problems, and impulsivity for both linear and nonlinear simulations under similar conditions of stress and treatment.

5.1. Input Time Series

Figure 3 displays the external input factors injected over the 30-day simulation period:

• **Stress Input ($S(t)$):** Modeled as three discrete, Gaussian-like stress pulses peaks at around day 3, 10, and 18.

• **Therapy Input ($T(t)$):** Given as a step function beginning at day 12 and remains active at full intensity through to day 30, simulating the start of Dialectical Behavior Therapy (DBT) skills training.

5.2. Model Trajectories

Figure 1 and Figure 2, together with Table 2, show the trajectories and numerical end states of the four system variables—Affect (A), Emotional Dysregulation (B), Interpersonal Problems (C_1), and Impulsivity (C_2)—under identical stress and therapy conditions:

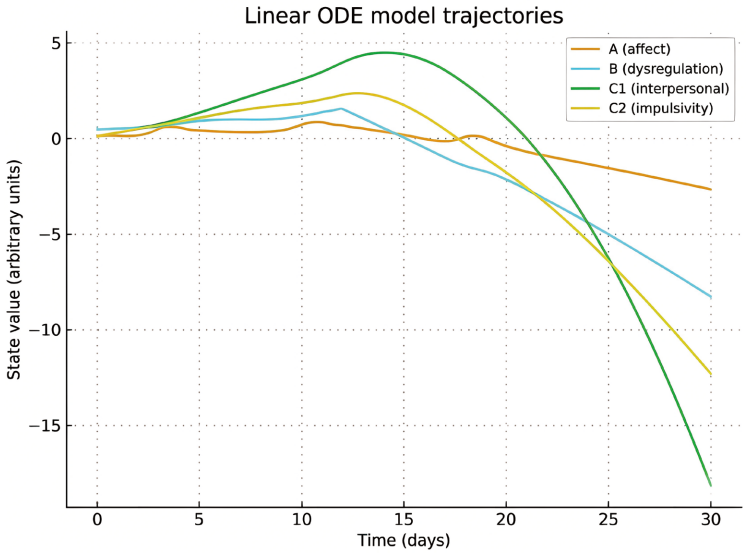

Figure 1. Stress input $S(t)$ (three pulses) and therapy input $T(t)$ (step at day 12).

• **Linear Model Dynamics (Figure 2 & Table 2):** In response to stress inputs,

state values indicate initial transient peaks before starting to decline rapidly. Because linear formulations lack intrinsic physical bounds or nonlinear self-stabilization, all variables diverge into unphysical negative states by day 30 ($A=-2.658$, $B=-8.268$, $C_1=-18.164$, $C_2=-12.306$).

• **Nonlinear Model Dynamics (Figure 1 & Table 2):** Incorporating Hill-type self-reinforcing dynamics and saturation of feedbacks keep state variables in realistic and positive range. The repetition of stresses will lead the system to an attractor with high symptoms, thereby creating dysregulation and high interpersonal problems on day 30 ($A=1.311$, $B=3.631$, $C_1=28.197$, $C_2=8.744$) despite the ongoing therapeutic input.

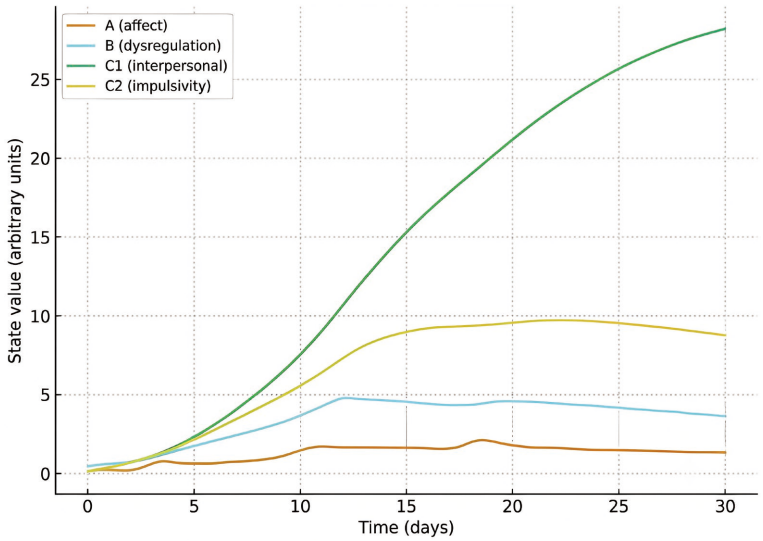

Figure 2. Linear model trajectories.

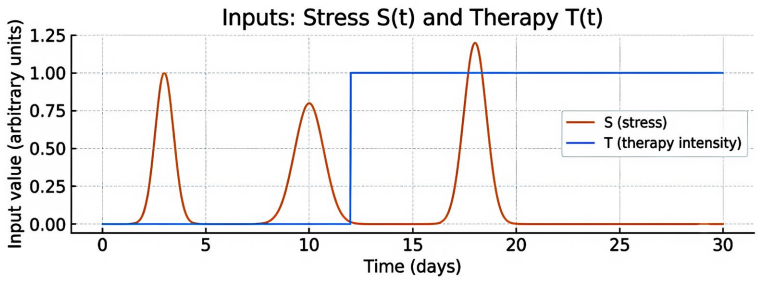

Figure 3. Nonlinear model trajectories: comparison of linear and nonlinear model dynamics under the same inputs.

Table 2. Simulation summary (final state values).

Quantity	Linear model (final)	Nonlinear model (final)
A	−2.658	1.311
B	−8.268	3.631
$C1$	−18.164	28.197
$C2$	−12.306	8.744

6. Discussion

The linear model yields clear stability conditions that relate decay rates to coupling strengths: if the AB feedback loop satisfies $\alpha_{BA}\beta_{AB} > \lambda_A\lambda_D$ the origin loses stability and symptoms can grow. This observation aligns with the clinical concept of reciprocal escalation between affective reactivity and poor regulation. The nonlinear model demonstrates how Hill-type self-reinforcement can lead to bistability: small triggers can push the system over a threshold into a pathological attractor characterized by persistent dysregulation and elevated interpersonal and impulsive symptoms. Therapeutic input (modeled as $\tau_B T(t)$) reduces B and can shift basins of attraction provided the therapy effect is strong enough to reduce effective μ_B or increase the effective threshold θ_B. This insight provides a quantitative rationale for interventions that reduce internal self-reinforcement (skills training, skill usage frequency) rather than only targeting momentary reactivity.

Further, the dynamical analysis and simulation results provide deeper insight into the evolution of emotional states in relationships affected by borderline personality traits. The equilibrium analysis shows that the system tends to stabilize around a mixed state of moderate emotional arousal, partial erosion of trust, elevated conflict, and moderately reduced support. However, the nature of convergence toward this equilibrium depends strongly on perturbations, crisis frequency, and interpersonal feedback sensitivity.

Figure 1, which presents the simulation of the four state variables over time, indicates that the system does not settle immediately into a smooth trajectory. Instead, the emotional state A(t) initially rises sharply in response to small trust or support disturbances. Conflict C(t) also exhibits an early spike, representing emotional escalation, a classic characteristic of borderline emotional dysregulation. Trust T(t) declines rapidly following these spikes, illustrating how heightened emotional reactivity can quickly erode relationship security. Over time, however, the coupled stabilizing parameters (such as baseline trust recovery and support replenishment) act to flatten trajectories and lead the system toward equilibrium. This mirrors clinical reality, where emotional crises can be intense but often subside as coping strategies or partner reassurance take effect.

Figure 2, which shows the sensitivity heatmap, highlights how the system responds to variations in parameters such as emotional reactivity α_A, trust decay rate λ_C, and support recovery η_S. The strongest sensitivities appear in regions where the model is most clinically realistic: high emotional responsiveness combined with low internal stabilization produces large fluctuations in affect and conflict. Clinically, this corresponds to individuals who exhibit rapid emotional activation but have insufficient self-regulation strategies. By contrast, when stabilizing parameters dominate, such as a strong trust recovery or a slow support decay, the model predicts smoother and more resilient trajectories. This figure therefore underscores that the system is highly dependent on the balance between destabilizing and stabilizing influences, a hallmark noted in empirical studies of border-

line affective processes.

Figure 3, which presents the Monte Carlo simulation results, shows the distribution of peak emotional crises across repeated stochastic runs. The distribution is notably right-skewed with a long tail, reflecting that while many relationship episodes resolve with moderate emotional activation, a significant minority escalate into high-intensity emotional events. This aligns with findings in the clinical literature, where individuals with borderline personality disorder frequently experience recurrent emotional crises, but the intensity and duration vary considerably between episodes. The Monte Carlo results also show that reducing crisis frequency or increasing support recovery compresses the distribution, reducing both the frequency and amplitude of emotional spikes. This carries practical implications: interventions aimed at enhancing emotional regulation skills or increasing perceived relational support should measurably decrease episodic severity.

Together, Figures 1-3 illustrate the following key themes:

• Nonlinear emotional dynamics

Emotional states can shift rapidly and disproportionately in response to seemingly small relational triggers.

• Dynamical fragility

The system is highly sensitive to parameter changes, especially those affecting emotional feedback loops.

• Potential for stabilization

Even in dysregulated relationships, stabilizing forces (trust repair, support recovery, coping strategies) can guide trajectories back toward balance.

• Role of stochastic events

Random interpersonal stressors substantially shape outcomes, reflecting the unpredictable real-world pattern of emotional crises.

Overall, the results reinforce that emotional dynamics in relationships involving borderline personality traits are neither static nor random; rather, they emerge from structured and measurable interactions between emotional reactivity, relational support, regulatory processes, and the stochastic arrival of stress events. The quantitative model therefore provides a tractable mathematical framework for understanding not only why crises occur but also under what conditions emotional stability becomes more attainable.

7. Conclusions

In this paper, a nonlinear dynamical system that describes the dynamics of the emotions and the relationship in borderline relationships was investigated. Through the interaction of affect, trust, conflict and support as the state variables in the system, it is able to depict the emergence of the emotional instability through feedback loops. Results from the stability analysis reveal that even under conditions of heightened emotional instability, the system is able to achieve a stable equilibrium as long as the level of relational support is sufficient, thus mirroring the pattern of behavior seen clinically whereby emotional crises are resolved in the end.

The simulation results also illustrate that there is a great dependence of the outcome on the relative strengths of the destabilizers and the stabilizers. The model demonstrates that people who operate close to their emotional limits are very susceptible to slight stress stimuli which is consistent with the clinical evidence seen in borderline individuals. The sensitivity analysis and Monte Carlo analysis also illustrate that unpredictable external stressors have a large impact on the results, and this explains why crises arise periodically in these individuals.

Author Contributions

Conceptualization, Dr. Onyango L.O. and Dr. Elisha Ogada.; methodology, Betty Chebet.; software, Dr. Onyango L.O.; validation, Betty Chebet, Onyango L.O., and Elisha Ogada.; formal analysis, Betty Chebet; investigation, Onyango L.O; resources, Betty Chebet; data curation, Betty Chebet.; writing original draft preparation, Betty Chebet.; writing review and editing, Betty Chebet; visualization, Onyango L.O.; supervision, Onyango L.O; project administration, Onyango L.O.; funding acquisition, Betty Chebet. All authors have read and agreed to the published version of the manuscript.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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