Volume 23, Issue 1: KUS: 1419: 115-123: 23052025: 2026

KHULNA UNIVERSITY
STUDIES

journal homepage: www.kus.ku.ac.bd

Research article

# A Dynamic Study of Depression Transmission and Recovery Using Mathematical Modeling

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## ABSTRACT

Depression has become a major global mental health issue and a significant factor contributing to suicide attempts. In this study, a nonlinear mathematical model is developed to investigate the dynamics of depression using four compartments: susceptible individuals, minor depressive disorder, major depressive disorder, and recovered individuals. The model has been analyzed qualitatively. The positivity and boundedness have been shown. The model has three equilibria: one depression-free equilibrium and two endemic equilibria for depression. Basic reproduction number is obtained, and stability analysis is performed. Sensitivity analysis to done to identify the parameters that most strongly influence the spread of depression. Numerical simulations of the model are also carried out to support the qualitative findings and demonstrate the behavior of the system. Our results reveal that the spread of depression largely depends on the progression onsets and the recovery levels. Our findings also highlight that early intervention and effective treatment strategies might be effective in reducing the prevalence of depression and its related risks.

## ARTICLE INFO

***Article timeline:***
Date of Submission:
23 May, 2025
Date of Acceptance:
18 May, 2026
Article available online:
28 June, 2026

***Keywords*:**
Mathematical model
Depression
Numerical simulations

## Introduction

Depression is a leading cause of suicide worldwide and affects more than 280 million people across all age groups (World Health Organization [WHO], 2021). It progresses to major depressive disorder (MDD), characterized by severe affective symptoms (American Psychiatric Association [APA], 2013; Cuijpers et al., 2007). Major depression frequently occurs and impairs social, occupational, and daily functioning (Kessler et al., 2003; Marcus et al., 2012). While minor depression presents with fewer symptoms, it often progresses to major depression if left untreated (Judd et al., 2013; Cuijpers & Smit, 2004).

Epidemiological studies have shown that approximately 15–20% of individuals experience a depressive episode at some point in their lives. On the other hand, about 5% of people suffer from major depressive disorder (MDD) annually (Ferrari et al., 2013; Bromet et al., 2011). The economic burden of depression is immense, including healthcare costs and indirect losses due to reduced productivity and decreased workplace performance (Greenberg et al., 2015). Moreover, depression is a major contributor to global suicide mortality, especially among individuals with untreated or poorly managed depression symptoms (Nock et al., 2008; World Health Organization [WHO], 2021). The progression between minor and major depression, as well as the recovery rate of individuals, is influenced by complex social and environmental factors (Kendler et al., 2002). Traditional approaches to studying depression, such as clinical trials and population surveys, are valuable but are often limited by cost, time, and available facilities. In this context, mathematical modeling serves as a powerful tool for analyzing the transmission and treatment of mental health disorders (Bauch & Earn, 2003). Mathematical models of depression can simulate the dynamics among susceptible (healthy), minorly depressed, majorly depressed, and recovered individuals, similar to the compartmental models used in epidemiology (Nursuprianah et al., 2022). Psychotherapy and cognitive-behavioral therapy (CBT) can be applied as control parameters to reduce transmission and increase the recovery rate (Rush et al., 2006; Cuijpers et al., 2014). Modeling also enables the exploration of how different subgroups, such as gender, age, and socioeconomic status, experience depression differently (Piccinelli & Wilkinson, 2000; Kuehner, 2017). For example, women are about twice as likely to experience depression as men, and this disparity can be captured through model parameters (Kessler, 2003). Furthermore, social isolation, stress, and poor mental health conditions can be incorporated into models to assess their impact on disease burden and recovery dynamics (Corrigan et al., 2014).

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DOI: https://doi.org/10.53808/KUS.2026.23.01.1419-se

Recent inspections have emphasized the importance of analyzing and understanding depressive disorders and their associated behavioral mechanisms. For instance, recent studies have examined that major depressive disorder (MDD) significantly affects the emotional function during risky decision-making processes. Computational behavioral modeling can be applied to investigate the latent decision-making factors in patients with MDD (Zhou et al., 2025). In addition, the study investigated the excess use of social media, which replaced person to person interactions among undergraduate students during the COVID-19 pandemic. It also found that loneliness can lead to depressive symptoms among individuals (Rosen S., 2023). Recent studies have also focused on the application of nonlinear mathematical models to investigate the complex progression of depressive disorders and their associated behavioral transitions. Although several compartmental models have been proposed over time, many of these studies are primarily based on statistical observations or clinical interpretations, rather than dynamical analysis. In addition, there are very few models which distinguish between minor depressive disorder and major depressive disorder progressions and suicide risk. Furthermore, limited attention has been given to sensitivity analysis and stability behaviors, which are important for identifying the most influential parameters affecting the spread and recovery of depression.

Public health policies of various mental health campaigns and structured strategies are also analyzed via mathematical modeling (Fineberg et al., 2021). Models have been proven useful during the COVID-19 pandemic as the appearance of depression was visible due to stress, isolation, and economic distress (Pfefferbaum & North, 2020). Therefore, there remains a strong need for a generalized mathematical framework that can simultaneously analyze susceptible individuals, minor depressive disorder, major depressive disorder, recovery dynamics, and suicide-related transitions within a combined system. Motivated by these research gaps, the present study develops a nonlinear compartmental mathematical model to investigate the transmission dynamics of depression and to evaluate the qualitative behavior of the system through equilibria, stability, sensitivity analysis, and numerical simulations. This model might serve as a tool to health planners and social workers to understand the dynamics of depression.

## Materials and Methods

### *Formulation of the mathematical model*

A four-compartmental mathematical model is formulated to describe the transmission dynamics of depression. The model is a classical epidemiological-type model (Kermack & McKendrick, 1927). The total population is divided into four distinct epidemiological classes: susceptible individuals $S(t)$, individuals with minor depressive disorder $A(t)$, individuals with major depressive disorder $B(t)$, and recovered individuals $C(t)$. Individuals enter the susceptible compartment with a constant recruitment rate $\Delta$, while all compartments are subjected to a natural mortality rate μ. Susceptible individuals may develop minor depressive disorder through effective psychological or social interaction with individuals suffering from minor depressive disorder at a rate $\alpha$. This interaction captures the progression to early-stage depression. Individuals in the minor depressive disorder class $A(t)$ may progress to major depressive disorder $B(t)$ through a deterioration process at rate $\beta$ (worsening mental health conditions). Alternatively, individuals in this class may recover and move to the recovered class $C(t)$ at rate $\sigma$. Individuals in the major depressive disorder class $B(t)$ may recover at rate $\gamma$, or may experience suicide-related mortality at rate $\delta$, in addition to natural death. Recovered individuals are still subjected to natural mortality. The variables and parameters used in the model are summarized in Table 1. Based on these assumptions, the model is governed by the following system of nonlinear ordinary differential equations:

$$\begin{aligned}
\frac{dS}{dt} &= \Delta - \alpha SA - \mu S,\\
\frac{dA}{dt} &= \alpha SA - \beta AB - \sigma A - \mu A,\\
\frac{dB}{dt} &= \beta AB - \gamma B - \delta B - \mu B,\\
\frac{dC}{dt} &= \gamma B + \sigma A - \mu C.
\end{aligned} \tag{2.1}$$

Note that, here the total population
$N = S + A + B + C,$
and
$S(0) \geq 0, A(0) > 0, B(0) > 0, C(0) > 0.$

**Table 1:** Description of the state variables and parameters used in model (2.1).

| Symbol | Description |
|---|---|
| $S(t)$ | Susceptible individuals |
| $A(t)$ | Individuals with minor depressive disorder |
| $B(t)$ | Individuals with major depressive disorder |
| $C(t)$ | Recovered individuals |
| $\Delta$ | Recruitment rate into susceptible population |
| $\alpha$ | Progression rate at which susceptible individuals develop minor depression |
| $\beta$ | Progression rate from minor to major depression |
| $\sigma$ | Recovery rate from minor depression |
| $\gamma$ | Recovery rate from major depression |
| $\delta$ | Suicide-related mortality rate |
| $\mu$ | Natural mortality rate |

### *Mathematical analysis of the model*

In this section, the proposed depression model (2.1) is analyzed mathematically to investigate its qualitative behavior. We show the positivity and boundedness of the model, and derive the depression-free and endemic equilibrium points. We also determine the basic reproduction number $R_0$. Sensitivity analysis of $R_0$ is conducted to identify the most influential parameters driving the spread of depression.

### *Positivity of solutions*

Since all state variables represent populations, therefore we have

$$S(t), A(t), B(t), C(t) \geq 0.$$

Here, we show the positivity using the first equation

$$\frac{dS}{dt} = \Delta - \alpha SA - \mu S$$

Ignoring the positive recruitment term,

$$\frac{dS}{dt} \geq -(\alpha A + \mu)S.$$

Thus,

$$S(t) \geq S(0)e^{-(\alpha A+\mu)t} > 0.$$

Similarly, one can show that $A(t), B(t)$, and $C(t)$ remain nonnegative for all $t > 0$.

***Invariant region***

As the total population:

$$N(t) = S(t) + A(t) + B(t) + C(t).$$

Differentiating gives:

$$\frac{dN}{dt} = \Delta - \mu N - \delta B.$$

Since $\delta B \geq 0$,

$$\frac{dN}{dt} \leq \Delta - \mu N.$$

Hence,

$$N(t) \leq \frac{\Delta}{\mu}.$$

Therefore, the feasible region is:

$$\Omega = \left\{(S, A, B, C) \in \mathbb{R}_+^4 : N(t) \leq \frac{\Delta}{\mu}\right\}.$$

Thus, the region $\Omega$ is positively invariant.

***Equilibrium points***

The equilibrium points of the system (2.1) are obtained by setting

$$\frac{dS}{dt} = \frac{dA}{dt} = \frac{dB}{dt} = \frac{dC}{dt} = 0.$$

Thus, the system

$$\Delta - \alpha SA - \mu S = 0$$

$$\alpha SA - \beta AB - \sigma A - \mu A = 0$$

$$\beta AB - \gamma B - \delta B - \mu B = 0$$

$$\gamma B + \sigma A - \mu C = 0$$

is solved simultaneously. The model admits three equilibrium points: depression-free equilibrium point $\xi^0$ and two endemic equilibria (partial $\xi^\circ$ and full $\xi^*$). These three equilibria are given in the following subsections.

***Depression-free equilibrium point***

The depression-free equilibrium $\xi^0$ corresponds to the absence of depression in the population (that is, $A = 0, B = 0$ and $C = 0$).

So, we have

$$S = \frac{\Delta}{\mu}$$

Therefore, the depression-free equilibrium point of the model (2.1) is,

$$\xi^0(S^0, A^0, B^0, C^0) = \left(\frac{\Delta}{\mu}, 0,0,0\right).$$

At this equilibrium point, the population consists entirely of susceptible individuals, while the other compartments are all absent.

***Partial endemic equilibrium point***

The model admits a partial endemic equilibrium point $\xi^\circ(S^\circ, A^\circ, B^\circ, C^\circ)$, which is given by

$$S^\circ = \frac{\mu + \sigma}{\alpha},$$

$$A^\circ = \frac{\Delta\alpha - \mu(\mu + \sigma)}{\alpha(\mu + \sigma)},$$

$$B^\circ = 0,$$

$$C^\circ = \frac{\sigma\left(\Delta\alpha - \mu(\mu + \sigma)\right)}{\alpha\mu(\mu + \sigma)}.$$

Note that here $A^\circ \neq 0$, but $B^\circ = 0$. At this equilibrium, the compartment representing major depressive disorder disappears, while individuals with minor depressive disorder and recovered individuals remain present in the population.

***Full endemic equilibrium point***

We denote the full endemic equilibrium point by $\xi^*(S^*, A^*, B^*, C^*)$. At this equilibrium point, depression persists in the population. Thus, it has

$A^* \neq 0$ and $B^* \neq 0$,

which means, at this point, the susceptible individuals coexist with the individuals experiencing minor depressive disorder, individuals with major depressive disorder, and recovered individuals. By calculation, we can directly write $\xi^*(S^*, A^*, B^*, C^*)$,

where

$$S^* = \frac{\Delta\beta}{\chi_3}, \qquad A^* = \frac{\chi_1}{\beta},$$

$$B^* = -\frac{\eta_1}{\beta\chi_3}, \qquad C^* = \frac{\eta_2}{\beta\mu\chi_3},$$

with

$$\chi_1 = \delta + \gamma + \mu,$$
$$\chi_2 = \mu + \sigma,$$
$$\chi_3 = \alpha\chi_1 + \beta\mu$$

and

$$\eta_1 = \alpha\chi_1(\mu + \sigma) + \beta\mu\chi_2 - \Delta\alpha\beta,$$
$$\eta_2 = \alpha\mu^2\psi_1 + \beta\mu^2\psi_1 + \alpha\sigma\psi_2 + \beta\delta\mu\sigma + \Delta\alpha\beta\gamma - \alpha\gamma\mu\psi_3,$$

where

$$\psi_1 = \sigma - \gamma,$$

$$\psi_2 = \delta^2 + \delta\gamma + 2\delta\mu + \gamma\mu\,,$$

and

$$\psi_3 = \gamma + \delta.$$

***Basic reproduction number***

In the context of epidemiology, the basic reproduction number $R_0$ of a model can be interpreted as the number of secondary infectious resulting from a single primary infection (Dietz, K. 1993). When $R_0 > 1$, it is an indication that the infectious disease is going to sustain in the community. On the other hand, the disease will die out when $R_0 < 1$. Same approach is applicable in our depression case. Below we find the basic reproduction number of our model using the Next Generation method (Diekmann et al., 1990, Van den Driessche & Watmough, 2002).

We have the infected compartments: $A(t)$ and $B(t)$.
Let $X = [A, B]^T$.
We write the system as:

$$\frac{dX}{dt} = F(X) - V(X),$$

where
$F(X)$ represents new infection terms,
$V(X)$ represents transition/removal terms.
The new infection matrix is:

$$F(X) = \begin{pmatrix} \alpha SA \\ 0 \end{pmatrix},$$

and the transition matrix is:

$$V(X) = \begin{pmatrix} \beta AB + \sigma A + \mu A \\ -\beta AB + \gamma B + \delta B + \mu B \end{pmatrix}.$$

At the depression-free equilibrium point $\xi^0(S^0, A^0, B^0, C^0) = \left(\frac{\Delta}{\mu}, 0,0,0\right)$, we have $A = 0$, and $B = 0$.

Thus

$$F = \begin{pmatrix} \alpha S^0 & 0 \\ 0 & 0 \end{pmatrix};\ V = \begin{pmatrix} \sigma + \mu & 0 \\ 0 & \gamma + \delta + \mu \end{pmatrix},$$

Since $V$ is diagonal:

$$V^{-1} = \begin{pmatrix} \frac{1}{\sigma + \mu} & 0 \\ 0 & \frac{1}{\gamma + \delta + \mu} \end{pmatrix}.$$

Now the Next-Generation matrix is

$$FV^{-1} = \begin{pmatrix} \frac{\alpha\Delta}{\mu(\sigma+\mu)} & 0 \\ 0 & 0 \end{pmatrix}.$$

The basic reproduction number $R_0$ is the spectral radius of $FV^{-1}$, which means
$R_0 = \rho(FV^{-1})$.

Hence,

$$R_0 = \frac{\alpha\Delta}{\mu(\sigma+\mu)}.$$

***Stability analysis***

**Theorem 1.** The depression-free equilibrium $\xi^0$ is asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof:
The Jacobian matrix of our system (2.1) is

$$J(S, A, B, C) = \begin{pmatrix} -\theta_1 & -\theta_4 & 0 & 0 \\ \theta_7 & \theta_2 & -\theta_5 & 0 \\ 0 & \theta_6 & \theta_3 & 0 \\ 0 & \sigma & \gamma & -\mu \end{pmatrix}, \quad (2.2)$$

where

$$\theta_1 = \alpha A + \mu,$$
$$\theta_2 = \alpha S - \beta B - \sigma - \mu,$$
$$\theta_3 = \beta A - \gamma - \delta - \mu,$$
$$\theta_4 = \alpha S,$$
$$\theta_5 = \beta A,$$
$$\theta_6 = \beta B,$$

and

$$\theta_7 = \alpha A.$$

At the depression-free equilibrium $\xi^0(S^0, A^0, B^0, C^0) = \left(\frac{\Delta}{\mu}, 0,0,0\right)$, this Jacobian matrix is triangular. So, the eigenvalues are the diagonal entries:

$$\lambda_1 = -\mu$$

$$\lambda^2 = \frac{\alpha\Delta}{\mu} - \sigma - \mu$$

$$\lambda_3 = -(\gamma + \delta + \mu)$$

$$\lambda_4 = -\mu$$

Clearly, $\lambda_1 < 0, \lambda_3 < 0$, and $\lambda_4 < 0$. Now, the stability depends only on $\lambda_2$.
We write

$$\lambda^2 = \frac{\alpha\Delta}{\mu} - (\sigma + \mu).$$

Recall that

$$R_0 = \frac{\alpha\Delta}{\mu(\sigma + \mu)}.$$

Thus,

$$\alpha\Delta = R_0\mu(\sigma + \mu).$$

Substituting gives:

$$\lambda^2 = \frac{R_0\,\mu(\sigma + \mu) - \mu(\sigma + \mu)}{\mu}.$$

Factoring,

$$\lambda_2 = (\sigma + \mu)(R_0 - 1).$$

If $R_0 < 1$, then $\lambda_2 < 0$.
Hence all eigenvalues are negative, and the depression-free equilibrium is locally asymptotically stable.

If $R_0 > 1$, then $\lambda_2 > 0$.

Thus, at least one eigenvalue is positive and the depression-free equilibrium becomes unstable. Hence the proof is complete.

**Theorem 2.** If $R_0 > 1$, the partial endemic equilibrium point $\xi^\circ(S^\circ, A^\circ, B^\circ, C^\circ)$ exists and is locally asymptotically stable.

Proof:
We use (2.2) to evaluate the Jacobian matrix at $\xi^\circ$, which is denoted by $J(\xi^\circ)$.
One important eigenvalue of $J(\xi^\circ)$ is

$$\lambda_1 = \beta A^\circ - (\gamma + \delta + \mu).$$

Substituting $A^\circ$,

$$\lambda_1 = \frac{\beta(\Delta\alpha - \mu(\mu+\sigma))}{\alpha(\mu+\sigma)} - (\gamma + \delta + \mu).$$

Thus, the partial endemic equilibrium is locally asymptotically stable provided
$R_0 > 1$ and $\beta A^\circ < \gamma + \delta + \mu$.
Equivalently,

$$\frac{\beta(\Delta\alpha - \mu(\mu+\sigma))}{\alpha(\mu+\sigma)} < \gamma + \delta + \mu.$$

This completes the proof.

**Theorem 3.** If $R_0 > 1$, the full endemic equilibrium point $\xi^*(S^*, A^*, B^*, C^*)$ exists and is locally asymptotically stable.

Proof:
From (2.2), the Jacobian matrix of the system at $\xi^*$ is $J(\xi^*)$.

The characteristic equation of this $J(\xi^*)$ has a fourth-degree polynomial with four coefficients, say $a_1, a_2, a_3$, and $a_4$. With the positive parameters of the model (2.1) and with $R_0 > 1$, and using the Routh-Hurwitz criterion, $\xi^*$ will be locally asymptotically stable if all these coefficients are positive and $a_1a_2a_3 > {a_3}^2 + {a_1}^2a_4$. This completes the proof.

***Sensitivity analysis***

Sensitivity analysis identifies the key parameters influencing a mathematical model and helps to understand how the system behavior changes under varying conditions [Chitnis et al., 2008]. In dynamic models, it is used to determine which parameters most strongly affect the basic reproduction number ($R_0$), and the dynamics.

The normalized forward sensitivity index $\Upsilon_p^{R_0}$ for each parameter $p$ is:

$$\Upsilon_p^{R_0} = \left(\frac{\partial R_0}{\partial p}\right)\left(\frac{p}{R_0}\right).$$

Table 2 and Figure 1 present the sensitivity indices of the basic reproduction number ($R_0$) with respect to the model parameters. The results show that the recruitment rate ($\Delta$) and the transmission parameter $\alpha$ have positive sensitivity indices of +1. This indicates that $R_0$ increases proportionally with increases in these parameters. In particular, a 1% increase in either $\Delta$ or $\alpha$ leads to a 1% increase in the basic reproduction number. Biologically, this suggests that higher recruitment into the susceptible population and stronger social or psychological interaction contributing to depression transmission significantly enhance the spread of depression within the population. Conversely, the recovery rate from minor depressive disorder $\sigma$ and the natural mortality rate $\mu$ exhibit negative sensitivity indices. This implies that increasing either parameter reduces the value of $R_0$. The negative sensitivity of $\sigma$ demonstrates that improving recovery from minor depressive disorder can substantially reduce depression transmission. So, this analysis reveals that the transmission parameter $\alpha$ and the recovery parameter $\sigma$ are the most influential parameters.

**Table 2:** Sensitivity indices of $R_0$ with respect to model parameters

| Parameters | Sensitivity indices |
|---|---|
| $\Delta$ | +1.000 |
| $\alpha$ | +1.000 |
| $\sigma$ | -0.9895 |
| $\mu$ | -1.0105 |

**Results and Discussion**

In this section we discuss the numerical results of our model (2.1). The model is solved using the 'ode45' solver of MATLAB. The initial values of the variables and all other parameters are assumed for simulation purposes. The parameter values are $\Delta = 0.013, \alpha = 0.011, \beta = 0.032, \delta = 0.07, \gamma = 0.521, \sigma = 0.8729,$ and $\mu = 0.0093$ with initial conditions $S(0) = 209.13\ million, A(0) = 12.2\ million, B(0) = 3.483\ million$ and $C(0) = 12.42\ million$

Using these parameter values, we have $R_0 = 0.0174 < 1$.

Figure 2 shows that the susceptible class $S(t)$ decreases rapidly during the early stage of the simulation, declining from its initial level to a significantly lower value as time progresses. The minor depressive class $A(t)$ initially increases and reaches a maximum value around the second year before gradually decreasing. This decline is associated with both recovery and progression into the major depressive class. The major depressive class $B(t)$ shows a delayed increase compared with $A(t)$, attaining its peak approximately around the third year. Afterward, the population in this compartment decreases steadily due to recovery, suicide-related mortality, and natural mortality effects. In contrast, the recovered class $C(t)$ increases continuously throughout the simulation and eventually becomes the dominant compartment after approximately 2.5 years. The growth of this class indicates the significant influence of the recovery mechanisms represented by the parameters $\sigma$ and $\gamma$.

Figure 3 illustrates the influence of the progression parameter $\alpha$ on the dynamics of the system for three different values, namely $\alpha = 0.0080$, $\alpha = 0.0110$, and $\alpha = 0.0165$. The simulations show that increasing $\alpha$ leads to a more rapid decline in the susceptible class $S(t)$. For larger values of $\alpha$, susceptible members transition more quickly into the minor depressive compartment, resulting in a sharper reduction of $S(t)$ during the early years of the simulation. The minor depressive class $A(t)$ is strongly affected by variations in $\alpha$. As $\alpha$ increases, the peak value

of $A(t)$ becomes significantly higher and occurs earlier in time. In contrast, smaller values of $\alpha$ produce a slower increase and a lower peak in the minor depressive compartment. A similar trend is observed in the major depressive class $B(t)$. Higher values of $\alpha$ lead to a

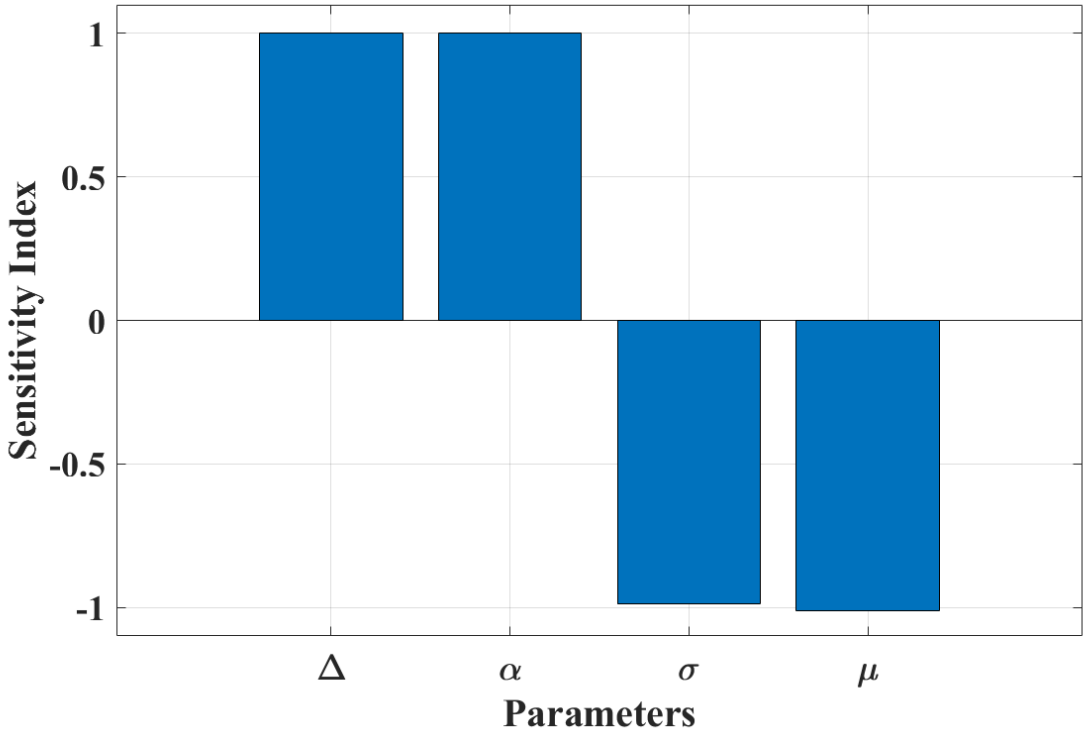


**Figure 1:** Sensitivity analysis of $R_0$

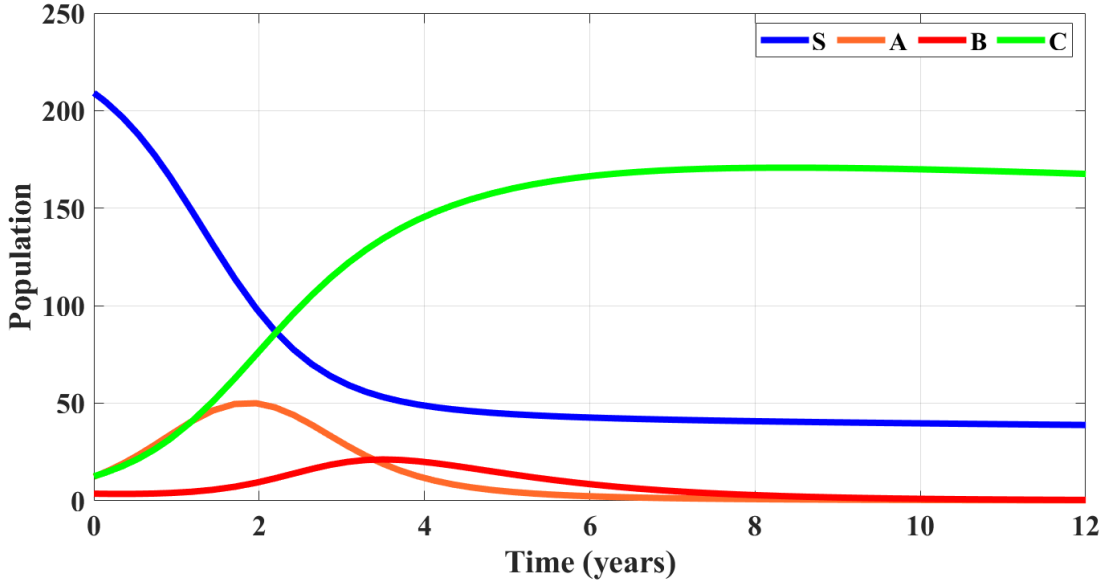


**Figure 2:** Dynamics of model (2.1), showing the evolution of susceptible $S(t)$, minor depressive $A(t)$, major depressive $B(t)$, and recovered $C(t)$ populations over 12 years

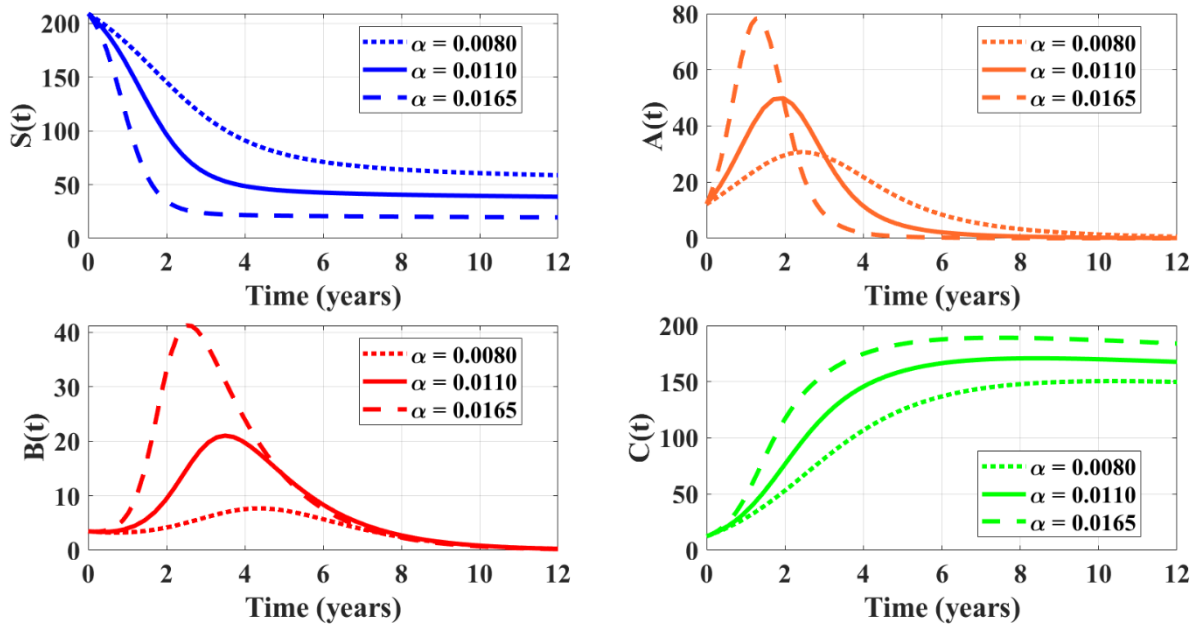


**Figure 3:** Effect of varying the progression parameter $\alpha$ on the dynamics of the susceptible $S(t)$, minor depressive $A(t)$, major depressive $B(t)$, and recovered $C(t)$ compartments over a 12-year period. The results are presented for $\alpha = 0.0080$, $\alpha = 0.0110$, and $\alpha = 0.0165$, while all other parameters are kept fixed.

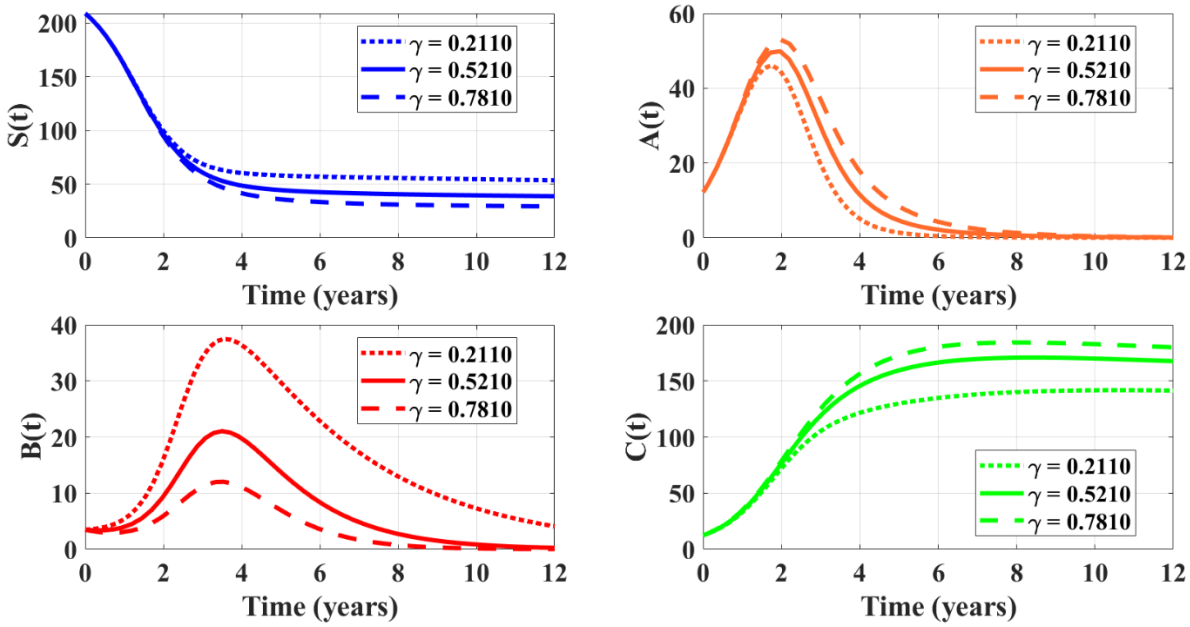


**Figure 4:** Effect of varying the recovery rate parameter $\gamma$ on the dynamics of the susceptible $S(t)$, minor depressive $A(t)$, major depressive $B(t)$, and recovered $C(t)$ compartments over a 12-year period. The results are presented for $\gamma = 0.2110$, $\gamma = 0.5210$, and $\gamma = 0.7810$, while all other parameters are kept fixed.

substantial increase in the magnitude of $B(t)$, indicating that rapid movement into the minor depressive class subsequently accelerates progression into the major depressive compartment. Moreover, the peak of $B(t)$ occurs earlier and reaches considerably larger values for higher $\alpha$. The recovered class $C(t)$ increases for all parameter choices, but larger values of $\alpha$ produce faster growth and higher recovery levels over time. This behavior occurs because increased progression through the depressive compartments also increases the number transitioning into recovery.

Figure 4 illustrates the influence of the recovery rate from major depressive disorder, $\gamma$, on the dynamics of the system for three different values, namely $\gamma = 0.2110$, $\gamma = 0.5210$, and $\gamma = 0.7810$. The simulations indicate that increasing $\gamma$ slightly accelerates the decline of the susceptible class $S(t)$, leading to lower equilibrium levels over time. Although the effect on $S(t)$ is less pronounced compared to other compartments, variations in $\gamma$ still influence the long-term population distribution. The minor depressive class $A(t)$ exhibits moderate sensitivity to changes in $\gamma$. Larger values of $\gamma$ result in slightly higher

peak levels of $A(t)$ and a slower decline afterward, whereas smaller values of $\gamma$ produce lower peaks and faster reduction of the compartment. A significant impact of $\gamma$ is observed in the major depressive compartment $B(t)$. Increasing $\gamma$ substantially reduces both the peak magnitude and persistence of $B(t)$. In particular, higher recovery rates from major depressive disorder lead to a faster decrease in severe depressive cases, while lower values of $\gamma$ allow the major depressive population to remain elevated for a longer period. The recovered compartment $C(t)$ increases for all considered values of $\gamma$, with larger values producing more rapid growth and higher long-term recovery levels. This behavior reflects the direct transfer of members from the major depressive class into the recovered compartment through the recovery term $\gamma B$.

***Implications and Limitations***

This model reveals that early interaction, progression and recovery system can play important roles in depression dynamics. It suggests that early detection, early treatment and mental health support can reduce the spread of depressive disorders. However, the model has limitations. It assumes homogeneous mixing and constant parameters. Despite this, the model provides a useful framework for understanding the dynamics of depression and guiding future research.

**Conclusions**

In this study, a nonlinear compartmental mathematical model has been proposed to investigate the transmission dynamics of depressive disorders within a population. The model divides the population into susceptible, minor depressive, major depressive, and recovered classes in order to analyze the progression and control of depression. The qualitative behavior of the model was examined through positivity, boundedness, equilibrium analysis and stability analysis. The basic reproduction number $R_0$ was derived to discuss the asymptotic stability. Sensitivity analysis was carried out to identify the most influential parameters affecting the spread and progression of depression. The numerical simulations demonstrated that increasing social awareness, psychiatric treatment, and recovery-related parameters significantly reduce both the minor and major depressed classes and increase the recovered class. On the other hand, higher transmission parameters accelerate the progression of severe depression. Overall, the proposed model provides important insights of the depression dynamics, and highlights the significance of early intervention, counseling and mental health support-programs to control the spread of depression. The findings of this study might help the policymakers and healthcare professionals to develop the prevention and treatment strategies. Future works may include extending this model by incorporating fractional-order derivatives, time delays, stochastic effects, or real epidemiological data for more realistic analysis.

**Data Statement:** No real-world data was used. The data of the figures are based on the model simulations.

**Acknowledgments:** We thank the anonymous reviewers and the editor for their constructive suggestions.

**Funding:** There was no funding associated with this study.

**Conflict of Interest**

The authors declare that they have no known competing interests.

**Ethical approval**: Not applicable.

**Authorship contribution statement:**

**MMI, SA,** and **SRM:** Equal contribution in conceptualization, methodology, investigation, formal analysis, and writing the original draft;

**SSS:** Supervision, validation, writing proofs in the final revised version with editing.

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