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“The beauty of mathematics only shows itself to more patient followers.”

Maryam Mirzakhani

UNIVERSITY OF FERRARA

Abstract

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MATHEMATICAL APPROACHES TO SOCIO-EPIDEMIOLOGICAL MODELLING AND SIMULATION

by ABU SAFYAN ALI

Several complex phenomena in modern science, such as epidemics, social interactions, opinion formation, and collective behavior, pose significant challenges due to their nonlinear dynamics, multiscale features, stochastic fluctuations, and limited data availability. In this PhD thesis, we explore a unified framework that integrates kinetic theory, mathematical epidemiology, and modern machine learning techniques to address some of these issues.

In contemporary epidemic contexts, effective prevention strategies require understanding the interplay between individuals' social behavior and their physical health states. We first investigate consensus formation through kinetic models, considering a large system of interacting agents embedded in a fixed social structure. To capture the heterogeneity of large-scale social networks, we employ graphons built from real-world network data, enabling a continuum representation of individual connectivity. We then develop an integrated Fokker–Planck compartmental opinion model on networks, derived via a mean-field limit using kinetic descriptions.

To incorporate more realistic interactions, we successively introduce multiplex networks that explicitly represent both social and physical contacts, highlighting their mutual influence through an overlapping factor. A kinetic compartmental model on multiplex networks is then formulated to characterize phase transitions in epidemic outbreaks, as the overlap between layers varies.

To address data scarcity, we propose a data augmentation strategy that integrates compartmental epidemic models with social structure and machine learning tools to improve predictive accuracy. We develop hybrid data-driven schemes that combine mechanistic models with neural networks to generate synthetic data that compensates for missing or incomplete observations. This approach enhances the reliability of neural predictors and provides an alternative to physics-informed neural networks. Parameter identification using physics-informed neural networks is a beneficial approach for validating the optimal control model.

Finally, we extend compartmental modeling to examine smoking dynamics under treatment interventions and media campaigns. Behavioral dynamics are also explored to reveal links between social media addiction and depression, emphasizing their relevance during disease outbreaks and in systems with memory effects. Mathematical features such as positivity, boundedness, and local and global stability are established to validate the proposed models. Threshold parameters and sensitivity analyses are used to predict disease persistence and identify key determinants.

Overall, this thesis advances multiscale modeling by integrating kinetic equations, network topologies, and data-driven methodologies, with particular emphasis on improving epidemic forecasting.

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Contents

Abstract	v
Acknowledgements	vii
1 Introduction	1
1.1 Disease dynamics	3
1.2 Kinetic theory and Boltzmann equation	7
1.2.1 Kinetic equation: Mathematical perspective	8
Microscopic level	8
Mesoscopic level (Kinetic formulation)	9
Macroscopic level	10
1.2.2 Non-Interacting particles system	10
1.2.3 Mean field particles system	11
1.2.4 The Boltzmann equation	12
Collision operator	14
H Theorem	15
Maxwellian distribution	16
Hydrodynamics approximation	16
1.2.5 Some kinetic models of interest	17
1.3 Role of network theory	19
1.3.1 Network models	19
1.3.2 Mean field limit of large networks	20
1.4 Data augmentation with neural networks	21
I Compartment Modeling of Disease Dynamics	23
2 Smoking dynamics in the context of nicotine gum and media campaigns	25
2.1 Introduction	25
2.2 Mathematical model to control smoking dynamics with treatment	28
2.3 Mathematical analysis of model	31
2.3.1 Positively invariant region	31
2.3.2 Positivity of solutions	33
2.3.3 Boundedness of solutions	34
2.3.4 Existence and Uniqueness of solution	34
2.3.5 Steady states of model	36
2.3.6 Threshold parameter $\mathcal{R}_0$	37
2.3.7 Stability analysis of model	38
Local stability analysis	38
Global stability analysis	40
2.3.8 Sensitivity analysis	43
2.3.9 Numerical simulation of model without treatment	44
2.3.10 Numerical simulation of model with treatment	45

	Effect of treatment to overcome smoking dynamics	47
	Effect of treatment on smoking-related disease mortality	48
2.4	Mathematical model to advertise daily news of addicted smokers through media and treatment to control smoking dynamics	51
2.4.1	Steady states of model	52
2.4.2	Threshold parameter R_0	52
2.4.3	Sensitivity analysis	53
2.4.4	Numerical simulation of model with daily media advertisement of smokers and treatment	53
	Impact of daily media advertisement of smokers and treatment	55
	Impact of daily news on smoking-related diseases mortality	57
2.5	Mathematical model of combined impact of TV, Radio, Social media advertisement, and treatment to control smoking dynamics	60
2.5.1	Computation of steady states	62
2.5.2	Threshold parameter R_0	63
2.5.3	Sensitivity analysis	63
2.5.4	Numerical simulation of model of combined impact of TV, Radio, Social media advertisement and treatment to control smoking dynamics	64
	Efficiency of TV, Radio, and Social media advertisement with treatment on the model	66
	Efficiency of TV, Radio, and Social media advertisement with treatment on smoking-related deaths	70
2.6	Comparison of models	71
2.6.1	Future work	76
2.7	Conclusion	76
3	Behavioral dynamics focusing on the interplay of social media addiction and depression with memory effect	79
3.1	Introduction	79
3.2	Social media addiction and depression model formulation	81
3.3	Mathematical analysis	83
3.3.1	Threshold parameter	85
3.3.2	Forward bifurcation analysis of model	87
3.4	Sensitivity analysis of model	92
3.4.1	Global sensitivity analysis by Partial Rank Correlation Coefficient	94
3.5	Dynamic analysis of model	95
3.5.1	Case 1: for $R_0 < 1$	97
3.5.2	Case 2: for $R_0 > 1$	97
3.6	Fractional derivatives	97
3.7	Model with Caputo derivative	101
3.7.1	Equilibrium points	103
3.7.2	Stability Analysis	104
3.8	Existence and uniqueness of model	107
3.8.1	Existence of the solution	109
3.8.2	Uniqueness of the solution	113
3.9	Ulam's stability analysis of model	115
3.10	Numerical simulation of model	118
3.10.1	Adams–Bashforth–Moulton PECE scheme	118
3.10.2	Fractional Forward Euler Scheme (FFE)	120

3.10.3	Adams-Bashforth Molten Scheme (ABM)	120
3.11	Effect of Parameters	123
3.11.1	Case 1: The rate at which addiction spreads to susceptible people (a_{12}).	123
3.11.2	Case 2: Natural death rate (a_4).	124
3.11.3	Case 3: The rate at which media effects become a cause of depression (a_5).	124
3.11.4	Case 4: Depressed individuals who join the recovered class after the treatment (a_8).	125
3.12	Discussion	126
3.13	Conclusion	130
II	Forecasting, Data Augmentation and Control Problem	133
4	Data augmentation in epidemic model with Feed Forward Neural Network and Nonlinear Autoregressive Network	135
4.1	Introduction	135
4.2	Problem setting	137
4.3	On the the construction of a data driven social-SIR model	139
4.3.1	Parameters estimation	139
4.4	Estimation and prediction using real COVID-19 data and Neural Networks design for epidemic prediction	142
4.4.1	Neural network for epidemic prediction	145
	Feed forward neural network	145
	Nonlinear autoregressive neural network	145
4.5	Conclusion	149
5	Parameters identification and optimal control of Behavioral dynamics using Physics Informed Neural Network	151
5.1	Introduction	151
5.2	Model formulation	154
5.3	Mathematical analysis	157
5.4	Construction of data driven model	172
5.4.1	Parameter estimation	172
5.4.2	Feed forward neural network	176
5.4.3	Physics informed neural network	178
5.5	Global sensitivity analysis	181
5.6	Formulation of optimal control model	185
5.7	Numerical analysis	190
5.7.1	Evaluation of data driven model	191
5.7.2	Transition flow and media influence	194
5.7.3	Simulation of control model	200
5.8	Conclusion	204
III	Boltzmann Type and Mean Field Modeling of Social & Disease Dynamics	207
6	Coupled opinion-epidemic dynamics on graphon-based networks	209
6.1	Introduction	209

6.2	A new model of opinion-driven epidemic dynamics with graphon based connectivity structure	211
6.2.1	Graphon based social connectivity structure	211
6.2.2	A new model of opinion-driven epidemic dynamics model . . .	212
6.2.3	Opinion formation under graphon structures	213
6.2.4	Macroscopic contacts and opinion-based epidemic dynamics .	218
6.3	Graphon Estimation from observed network data	220
6.4	Numerical Tests	222
6.4.1	Numerical experiments in homogeneous and graphon-based settings	224
6.4.2	Opinion formation model with real network data	226
6.4.3	Epidemic model simulation with networks	228
6.5	Conclusion	232
7	Phase transition in interacting opinion and disease dynamics over multiplex networks	233
7.1	Introduction	233
7.2	Physical and social interactions on multiplex networks: the discrete case.	235
7.2.1	The microscopic process of opinion formation	237
7.3	A mesoscopic model of opinion formation and disease spread	239
7.3.1	A Fokker–Planck limit model for the opinion formation process with physical and social connections	241
7.4	Numerical simulations	242
7.4.1	Construction of the adjacency matrices	242
7.4.2	Numerical simulations for the microscopic model	243
7.5	Conclusion	247
A	Derivation of fractional differential system schemes	249
B	Steady states and polynomial coefficients	261
	Bibliography	263

List of Figures

1.1	A schematics diagram of the SIR compartmental model.	4
1.2	Visualization of some real-world phenomena of complex interacting particle dynamics.	7
1.3	Geometry of Boltzmann dynamics. The first shows the elastic collisions, while the second describes the case of a hard sphere.	13
1.4	Display of various networks.	19
1.5	A schematic diagram of data augmentation with neural network surrogate models.	22
2.1	Schematic diagram of smoking dynamics in model (5.1).	31
2.2	Sensitivity analysis of $\mathcal{R}_0$ shows the significant parameters of model (5.1).	44
2.3	Impact of different parameters on $\mathcal{R}_0$ of model (5.1).	45
2.4	Numerical simulation of model (5.1) without treatment of smokers (ρ) with parameters in Table 3.2, step size $h = 0.01$ and $T = [0 \ 70]$	45
2.5	Numerical simulation of model (5.1) without treatment of smokers (ρ) separately with parameters in Table 3.2, step size $h = 0.01$ and $T = [0 \ 70]$	46
2.6	Numerical simulation of model (5.1) with treatment of smokers (ρ) with parameters in Table 3.2, step size $h = 0.01$ and $T = [0 \ 70]$	46
2.7	Numerical simulation of model (5.1) with treatment of smokers (ρ) separately with parameters in Table 3.2, step size $h = 0.01$ and $T = [0 \ 70]$	47
2.8	Numerical simulation of model (5.1) with variation of treatment of smokers (ρ): (a) shows the variation of $\rho = \{0, 0.02, 0.05, 0.1, 0.17\}$ on smoker S , (b) presents the 3D views by variation of $\rho = 0 : 0.001 : 0.17$	48
2.9	Simulation of $\mathcal{R}_0$ of model (5.1) w.r.t β_1 and μ with variation of treatment of smokers (ρ): Variation of $\rho = \{0, 0.02, 0.05, 0.1, 0.17\}$ on $\mathcal{R}_0$ w.r.t β_1 and μ are shown.	48
2.10	Numerical simulation of model (5.1) with variation of treatment of smokers (ρ): (a) shows the variation of $\rho = \{0, 0.02, 0.05, 0.1, 0.17\}$ on death smoker D , (b) presents the 3D views by variation of $\rho = 0 : 0.001 : 0.17$	50
2.11	Simulation of $\mathcal{R}_0$ of model (5.1) w.r.t γ and μ with variation of treatment of smokers (ρ): Variation of $\rho = \{0, 0.1, 0.17\}$ on $\mathcal{R}_0$ w.r.t γ and μ are shown.	50
2.12	Simulation of $\mathcal{R}_0$ of model (5.1) w.r.t β_1 and γ with variation of treatment of smokers (ρ): Variation of $\rho = \{0, 0.1, 0.17\}$ on $\mathcal{R}_0$ w.r.t β_1 and γ are shown	50
2.13	Sensitivity analysis of $\mathcal{R}_0$ of model (2.33)	54
2.14	Simulation of $\mathcal{R}_0$ of model (2.33) w.r.t different parameters	54

2.15	Numerical simulation of model (2.33) with daily media advertisement (m) and treatment of smokers (ρ) with parameters in Table 3.2, step size $h = 0.01$ and $T = [0 \ 70]$	55
2.16	Numerical simulation of model (2.33) with daily media advertisement of smokers (m) and treatment of smokers (ρ) separately with parameters in Table 3.2, step size $h = 0.01$ and $T = [0 \ 70]$	56
2.17	Numerical simulation of model (2.33) with variation of treatment of smokers (ρ) and daily media advertisement (m): (a) shows the variation of $\rho = \{0, 0.02, 0.05, 0.1, 0.17\}$ on smoker S while taking daily media advertisement of smokers (m) fixed, (c) shows the variation of $m = \{0, 0.5, 1, 1.5, 2\}$ on smoker S while taking treatment (ρ) fixed, (b) and (d) presents the 3D views by variation of $\rho = 0 : 0.001 : 0.17$ and $m = 0 : 0.01 : 2$ respectively.	57
2.18	Simulation of $\mathcal{R}_0$ of model (2.33) w.r.t β_3 and μ with variation of treatment of smokers $\rho = \{0, 0.02, 0.05, 0.1, 0.17\}$	58
2.19	Numerical simulation of model (2.33) with variation of treatment of smokers (ρ) and daily media advertisement (m): (a) shows the variation of $\rho = \{0, 0.02, 0.05, 0.1, 0.17\}$ on death smoker D while taking daily media advertisement of smokers (m) fixed, (c) shows the variation of $m = \{0, 0.5, 1, 1.5, 2\}$ on death smoker D while taking treatment (ρ) fixed, (b) and (d) presents the 3D views by variation of $\rho = 0 : 0.001 : 0.17$ and $\rho = 0 : 0.01 : 2$ respectively. (e) is death compartments with variation of treatment of smokers $\rho = \{0, 0.02, 0.05, 0.1, 0.17\}$ and daily media advertisement of smokers $m = \{0, 0.5, 1, 1.5, 2\}$	59
2.20	Numerical simulation of model (2.33) with variation of treatment of smokers (ρ) and daily media advertisement (m) on $\mathcal{R}_0(\gamma, \mu)$	59
2.21	Numerical simulation of model (2.33) with variation of treatment of smokers (ρ) and daily media advertisement (m) on $\mathcal{R}_0(\beta_3, \gamma)$	60
2.22	Sensitivity analysis of $\mathcal{R}_S$, $\mathcal{R}_R$, $\mathcal{R}_Q$ and $\mathcal{R}_0$ of model (2.37).	64
2.23	Variation of parameters on $\mathcal{R}_0$ of model (2.37).	65
2.24	Numerical simulation of model (2.37) with media awareness program (M) and treatment of smokers (ρ) with parameters in Table 3.2, step size $h = 0.01$ and $T = [0 \ 70]$	66
2.25	Numerical simulation of model (2.37) with media awareness program (M) and treatment of smokers (ρ) separately with parameters in Table 3.2, step size $h = 0.01$ and $T = [0 \ 70]$	66
2.26	Numerical simulation of model (2.37) with variation of treatment of smokers (ρ) and TV, radio, social media advertisement (M): (a) shows the variation of $\rho = \{0, 0.02, 0.05, 0.1, 0.17\}$ on smoker S while no TV, radio, social media advertisement (M), (c) shows the variation of $M = \{0, 0.25, 0.5, 0.75, 1\}$ on smoker S while no treatment (ρ) of smokers, (b) and (d) presents the 3D views by variation of $\rho = 0 : 0.001 : 0.17$ and $M = 0 : 0.001 : 1$ respectively. (e) smoker compartments with variation of treatment of smokers (ρ) and TV, radio, social media advertisement (M) at $M = \{0, 0.2, 0.4, 0.6, 0.8, 1\}$ and $\rho = \{0, 0.02, 0.05, 0.1, 0.14, 0.17\}$	68
2.27	Simulation of $\mathcal{R}_0$ of model (2.37) w.r.t β and μ with variation of treatment of smokers $\rho = \{0, 0.05, 0.1, 0.17\}$ and TV, radio, social media advertisement $M = \{0, 0.3, 0.7, 1\}$	69

2.28	Simulation of $\mathcal{R}_S$, of model (2.37) w.r.t β and μ with variation of treatment of smokers $\rho = \{0, 0.05, 0.1, 0.17\}$ and TV, radio, social media advertisement $M = \{0, 0.3, 0.7, 1\}$	69
2.29	Simulation of $\mathcal{R}_R$, of model (2.37) w.r.t β and μ with variation of treatment of smokers $\rho = \{0, 0.05, 0.1, 0.17\}$ and TV, radio, social media advertisement $M = \{0, 0.3, 0.7, 1\}$	69
2.30	Simulation of $\mathcal{R}_Q$, of model (2.37) w.r.t β and μ with variation of treatment of smokers $\rho = \{0, 0.05, 0.1, 0.17\}$ and TV, radio, social media advertisement $M = \{0, 0.3, 0.7, 1\}$	70
2.31	Numerical simulation of model (2.37) with variation of treatment of smokers (ρ) and TV, radio, social media advertisement (M): (a) shows the variation of $\rho = \{0, 0.02, 0.05, 0.1, 0.17\}$ on smoker S while no TV, radio, social media advertisement (M) , (c) shows the variation of $M = \{0, 0.25, 0.5, 0.75, 1\}$ on smoker S while no treatment (ρ) of smokers, (b) and (d) presents the 3D views by variation of $\rho = 0 : 0.001 : 0.17$ and $M = 0 : 0.001 : 1$ respectively. (e) smoker compartments with variation of treatment of smokers (ρ) and TV, radio, social media advertisement (M) at $M = \{0, 0.2, 0.4, 0.6, 0.8, 1\}$ and $\rho = \{0, 0.02, 0.05, 0.1, 0.14, 0.17\}$	72
2.32	Simulation of $\mathcal{R}_0$ of model (2.37): $\mathcal{R}_0$ of model w.r.t γ and μ with variation of treatment of smokers $\rho = \{0, 0.05, 0.1, 0.17\}$ and TV, radio, social media advertisement $M = \{0, 0.3, 0.7, 1\}$	72
2.33	Simulation of $\mathcal{R}_0$ of model (2.37): $\mathcal{R}_0$ of model w.r.t β and γ with variation of treatment of smokers $\rho = \{0, 0.05, 0.1, 0.17\}$ and TV, radio, social media advertisement $M = \{0, 0.3, 0.7, 1\}$	72
2.34	Numerical simulation of model (5.1), (2.33), (2.37) smoker compartment respectively with variation of treatment of smokers (ρ), TV, radio, social media advertisement (M) and daily media advertisement of number of smokers (m).	74
2.35	Numerical simulation of model (5.1), (2.33), (2.37) recovered compartment respectively with variation of treatment of smokers (ρ), TV, radio, social media advertisement (M) and daily media advertisement of number of smokers (m).	74
2.36	Numerical simulation of model (5.1), (2.33), (2.37) quitter compartment respectively with variation of treatment of smokers (ρ), TV, radio, social media advertisement (M) and daily media advertisement of number of smokers (m).	75
2.37	Numerical simulation of model (5.1), (2.33), (2.37) death compartment respectively with variation of treatment of smokers (ρ), TV, radio, social media advertisement (M) and daily media advertisement of number of smokers (m).	76
3.1	Variation of different parameters over $\mathbf{R}_0$	94
3.2	Sensitivity Index of Reproduction Number $\mathbf{R}_0$	95
3.3	PRCC values representing the sensitivities of the model outputs.	96
3.4	Surface plots of the solution of all classes of the social media addiction and depression (SMAD) model obtained by the RK-4 scheme for E_{q0} with initial conditions and step size $h = 0.001$. The numerical scheme converges to the true equilibrium point E_{q0} for $\mathbf{R}_0 < 1$	98

3.5	Surface plots of the solution of all classes of the social media addiction and depression (SMAD) model obtained by the RK-4 scheme for E_{q1} with initial conditions and step size $h = 0.001$. The numerical scheme converges to the true equilibrium point E_{q1} for $\mathbf{R}_0 > 1$	99
3.6	Compartmental diagram of transmission of social media addiction and depression dynamics	102
3.7	Simulation of addiction and depression phenomena for model (3.22) with Caputo fractional derivative presents here. first column shows present the case $\mathbf{R}_0 < 1$ when we perturbate the initial condition of both classes with $a_4 = 0.09, a_{12} = 0.2$ while second column describe the case $\mathbf{R}_0 > 1$ with $a_4 = 0.05, a_{12} = 0.8$	117
3.8	All classes simulation for model (3.22) with Caputo fractional derivative shown here. (a) present the case $\mathbf{R}_0 < 1$ with $a_4 = 0.09, a_{12} = 0.2$ while (b) describe the case $\mathbf{R}_0 > 1$ with $a_4 = 0.05, a_{12} = 0.8$ and (c) Compare Addicted and Depressed classes with Caputo derivative $\aleph = 0.995$ for $\mathbf{R}_0 > 1$ and $\mathbf{R}_0 < 1$	118
3.9	Results of model (3.22) with Caputo fractional derivative at order $\aleph = \{0.7, 0.8, 0.85, 0.9, 0.95, 0.995, 1\}$ for $\mathbf{R}_0 < 1$	119
3.10	Results of model (3.22) with Caputo fractional derivative at order $\aleph = \{0.7, 0.8, 0.85, 0.9, 0.95, 0.995, 1\}$ for $\mathbf{R}_0 > 1$	119
3.11	Analysis of Caputo fractional derivative $\aleph$ vs compartment's populations over time.	119
3.12	All classes simulation for model (3.22) with Caputo fractional derivative shown here. (a) present the case $\mathbf{R}_0 < 1$ with $a_4 = 0.09, a_{12} = 0.2$ while (b) describe the case $\mathbf{R}_0 > 1$ with $a_4 = 0.05, a_{12} = 0.8$ and (c) Compare Addicted and Depressed classes with Caputo derivative $\aleph = 0.995$ for $\mathbf{R}_0 > 1$ and $\mathbf{R}_0 < 1$	120
3.13	Results of model (3.22) with Caputo fractional derivative at order $\aleph = \{0.7, 0.8, 0.85, 0.9, 0.95, 0.995, 1\}$ for $\mathbf{R}_0 < 1$	121
3.14	Results of model (3.22) with Caputo fractional derivative at order $\aleph = \{0.7, 0.8, 0.85, 0.9, 0.95, 0.995, 1\}$ for $\mathbf{R}_0 > 1$	121
3.15	Analysis of Caputo fractional derivative $\aleph$ vs compartment's populations over time.	122
3.16	All classes simulation for model (3.22) with Caputo fractional derivative shown here. (a) present the case $\mathbf{R}_0 < 1$ with $a_4 = 0.09, a_{12} = 0.2$ while (b) describe the case $\mathbf{R}_0 > 1$ with $a_4 = 0.05, a_{12} = 0.8$ and (c) Compare Addicted and Depressed classes with Caputo derivative $\aleph = 0.995$ for $\mathbf{R}_0 > 1$ and $\mathbf{R}_0 < 1$	122
3.17	Results of model (3.22) with Caputo fractional derivative at order $\aleph = \{0.7, 0.8, 0.85, 0.9, 0.95, 0.995, 1\}$ for $\mathbf{R}_0 < 1$	122
3.18	Results of model (3.22) with Caputo fractional derivative at order $\aleph = \{0.7, 0.8, 0.85, 0.9, 0.95, 0.995, 1\}$ for $\mathbf{R}_0 > 1$	123
3.19	Analysis of Caputo fractional derivative $\aleph$ vs compartment's populations over time.	123
3.20	Variation of parameters a_{12} on model (3.22)	124
3.21	Variation of parameters a_4 on model (3.22)	125
3.22	Variation of parameters a_5 on model (3.22)	125
3.23	Variation of parameters a_8 on model (3.22)	126
3.24	Comparison of addicted and depressed Class with Caputo and Atangana-Baleanu-Caputo operators	127

4.1	Pre-lockdown phase. Dynamics of the infected population computed by solving the social-SIR model (4.2) with β and γ as in (4.10) (Italy), and as in (4.11) (Spain) and $H(t, I(t)) = 1$, compared with the experimental data. In the embedded plot, the pointwise error between the two quantities is computed as in (4.12). On the left, Italy. On the right, Spain.	141
4.2	Lockdown and post-lockdown phase. On the left the dynamics of the infected population computed by solving the generalized SIR model (4.2) with β and γ as in (4.10) (Italy), and as in (4.11) (Spain), assuming the contact function H to be defined as in (4.18) and, alternatively, with the smoothed version (4.19) setting $k = 1.6$ compared with the experimental data. In the embedded plot the pointwise error between the two infected populations is computed as in (4.12). On the right the contact function H is computed as in (4.18) and its smoothed version is computed introducing an operator as in (4.19). First row, Italy. Second row, Spain.	143
4.3	Feedforward neural network for Italy. On the left the dynamics of the infected population computed by training a FNN on real and synthetic data and tested over the train interval $[t_l, T]$ compared to the available data. On the right the solution computed with the same FNN tested over the time interval $(T, T_{test}]$ compared to the available data. Embedded the error between the available data test and the neural network solution computed as in (4.12).	146
4.4	Feedforward neural network for the Spain case. On the left the dynamics of the infected population computed by training a FNN on real and synthetic data and tested over the train interval $[t_l, T]$ compared to the available data. On the right the solution computed with the same FNN tested over the time interval $(T, T_{test}]$ compared to the available data. Embedded the error between the available data test and the neural network solution computed as in (4.12). First row, Italy. Second row, Spain.	147
4.5	Nonlinear autoregressive network: training data. Solution obtained by training a NAR network on both real and synthetic data, then testing it on the same dataset and comparing it to the available data. On the left, the case for Italy, and on the right, the one for Spain.	147
4.6	Nonlinear autoregressive network: short and long time predictions. Solution obtained by training a NAR network on both real and synthetic data, then testing it to produce daily data from July 2nd to July 8th (first row), and extend to July 15th (second row), compared with the available true data. On the left, the case for Italy, and on the right, the one for Spain.	148
5.1	Graphical representation of the model fits actual depression instances in Spain.	172

- 5.2 A representation of the proposed PINN approach to estimating the model (5.1) solution. An input layer with a single input variable—a time coordinate—makes up the trained network. Each of the three hidden layers has n neurons that are connected to the input and output layers. Since the intended response is expressed by a single neuron, the output layer has model state vectors. Using state vectors and their derivatives, this neural network design is used to evaluate the residual of the ordinary differential equation. A complete loss function is created by merging partial loss functions. After that, it is weighted and reduced as necessary. 179
- 5.3 Evaluation of the global sensitivity of basic reproduction number given in (5.4) of behavioral dynamics proposed in model (5.1). The output shows that $\Lambda, \lambda_1, \xi, \gamma, \nu$ are significant parameters with positive magnitude while $\beta, \phi_1, r_1, \alpha, \mu$ are significant parameters with negative magnitude. Particularly parameters $\lambda_1, \beta, \xi, \nu, \alpha$ are strongly influence the disease state. To decrease the addiction and depression transmission in infected individuals, λ_1, ξ, ν should be decreased in a positive direction while β, α increase in a negative direction. For the simulation of the outputs, we take a sample size $N = 10000$ with $t \in [0, 1000]$ having step size $dt = 1$ and time point of interest 500. . . . 182
- 5.4 Time series evaluation of the global sensitivity description of proposed model (5.1) to represent the nonlinear correlation of all the biological parameters having specific interval with $\pm 50\%$ from their baseline values in Table 5.1 with the compartments $S(t), E(t), I_1(t), I_2(t), R(t), Q(t), D(t)$ row wise from left to right respectively. The simulation was done with sample size $N = 10000$ and time interval $t \in [0, 50]$ of step size $dt = 0.5$. The precise investigation of addiction state $I_1(t)$ indicates that parameter $\nu, \lambda_1, \gamma, \xi, \beta, \alpha$ strongly influence the disease state of this compartment individuals while depression state $I_2(t)$ effectively compromised through $\alpha, \beta, \lambda_1, \gamma, \xi, \mu, q$. This statistical simulation became a strong turning point for the health care centers and policymakers to effectively implement the precaution measures through media awareness, increasing physical therapy centers as first aid. 184
- 5.5 Evaluation of state variables row-wise from left to right of the behavior model (5.1) with estimated parameters and error estimation. The simulation done using the FNN base neural network structure consists of three layers, with each layer having 10 neurons. The hidden activation is performed through linear saturating satlins, while for output activation we use a linear function purelin. The network was trained using Levenberg Marquardt back propagation trainlm with epochs 200 and a data division ratio of train: 80%, val: 10%, test: 10%. The reference solution was generated using the RK-4 method with $t \in [0, 50]$ and a step size of 0.5. The forecasting trajectories of each state variable are biologically meaningful and collectively capture the monotonic relation between social media addiction and depression in individuals. 194
- 5.6 Comparison of the estimated parameters $\nu, \pi, \phi_1, \xi, \gamma, \beta, r_1, \theta$, and corresponding true values row-wise from left to right over time with FNN over 200 epochs. The reference solution was generated using the RK-4 method with $t \in [0, 50]$ and a step size of 0.5. 195

5.7	Evaluation of state variables row-wise from left to right of the behavior model (5.1) with estimated parameters and error estimation. The simulation defined with PINN-based deep neural network structure consists of one input layer and three hidden layers, with each layer having 64 neurons with ReLU (Rectified Linear Unit) activation functions to describe the monotonic behavior of the model. The reference solution was generated using the RK-4 method with $t \in [0, 50]$ and a step size of 0.5. The forecasting trajectories of each state variable are biologically meaningful and collectively capture the complex interaction between social media addiction and depression in individuals.	196
5.8	Comparison of the estimated parameters ν , π , ϕ_1 , ξ , γ , β , r_1 , θ , and corresponding true values row-wise from left to right over time with FNN over 20000 epochs. The reference solution was generated using the RK-4 method with $t \in [0, 50]$ and a step size of 0.5.	196
5.9	Setting 1: Comparison of actual data given in Table 5.1 and estimated FNN and PNN data simulated in Sec 5.7.1 over each compartment $S(t)$, $E(t)$, $I(t)$, $R(t)$, $Q(t)$, $D(t)$ of model (5.1) row wise from left to right with initial condition written in Sec 5.4.1. The simulation perfectly describes the alignment of data-driven models with epidemic trajectories over time. The simulation run over $t \in [0, 50]$ with step size $dt = 0.05$ using higher order RK-4 method.	198
5.10	Setting 2: Evaluation of transition flow and forecasting of addiction and depression states on individuals with respective transmission rates and treatment process. The first row describes the addiction dynamics while the second row shows depression dynamics using model (5.1). The first column explains the variation of addiction and depression variables, peak size of addiction and depression defined in the second column, and time to peak with respect to parameter pairs λ_1, ϕ_1 , λ_2, ϕ_2 of addiction and depression given in the third column. For simulation, we assume $t \in [0, 50]$ and $dt = 0.1$	199
5.11	Setting 3: Simulation of total infection time to peak (first row), total infection peak size (second row) and cumulative infection (third row) with respective parameters cases ν (first column), q (second column), α (third column) and β (fourths column) precisely describe in Table 5.1. The evaluation is defined by perturbing $\pm 50\%$ each parameter from baseline and taking $t \in [0, 50]$ with $dt = 1/10$	199
5.12	Setting 4: Impact of basic reproduction number $\mathbb{R}_0$ (5.4) of model (5.1) on the behavior epidemic spread. Analysis of biological parameters pairs (β, q) , (ϕ_1, r_1) , (β, ξ) , (ϕ_2, q) , (γ, α) , (ν, α) over $\mathbb{R}_0$ given in row one and two from left to right respectively. The dotted red line defines the value $\mathbb{R}_0 = 1$. In third row, we presents the 3D visualization of pairs $(\lambda_1, \phi_1, \gamma)$, (λ_2, ϕ_2, q) and (α, μ, β) over $\mathbb{R}_0$. All parameter descriptions are given in Table 5.1.	200
5.13	Test 1: Simulation of the optimality system (5.41)-(5.42) using numerical scheme expressed in Algorithm3 for evaluation of state variables E, I_1, I_2, R, Q, D row wise from left to right by applying only u_1 control with $u_2 = 0$. We assume social cost $\kappa_1 = 1, \kappa_2 = 2$ and weighted parameters $\kappa_3 = \kappa_4 = 10$ of controls having upper bound $u_{1\max} = 0.6$ and $u_{2\max} = 0$ with $u_i \in [0, 1]$. The simulation is defined with time in years $t \in [0, 50]$ of 1000 discrete time points with step size $0.05/2$ and convergence tolerance 1×10^{-5}	204

5.14	Test 2: Simulation of the optimality system (5.41)-(5.42) using numerical scheme given in Algorithm 3 for evaluation of state variables E, I_1, I_2, R, Q, D row wise from left to right by applying only u_2 control with $u_1 = 0$. We assume social cost $\kappa_1 = 1, \kappa_2 = 2$ and weighted parameters $\kappa_3 = \kappa_4 = 10$ of controls having upper bound $u_{1\max} = 0$ and $u_{2\max} = 0.8$ with $u_i \in [0, 1]$. The simulation is defined with time in years $t \in [0, 50]$ of 1000 discrete time points with step size 0.025 and convergence tolerance 1×10^{-5}	204
5.15	Test 3: Simulation of the optimality system (5.41)-(5.42) using numerical scheme written in Algorithm 3 for evaluation of state variables E, I_1, I_2, R, Q, D row wise from left to right by applying combine controls $u_1 = u_2 \neq 0$. We assume social cost $\kappa_1 = 1, \kappa_2 = 2$ and weighted parameters $\kappa_3 = \kappa_4 = 10$ of controls having upper bound $u_{1\max} = 0.6$ and $u_{2\max} = 0.8$ with $u_i \in [0, 1]$. The simulation is defined with time in years $t \in [0, 50]$ of 1000 discrete time points with step size 0.025 and convergence tolerance 1×10^{-5}	205
5.16	Comparison of all Tests simulation for optimality system (5.41)-(5.42) using numerical scheme define in Algorithm 3. In the first row, the total infection (right) shows a significant reduction in addiction and depression population, while the cumulative population (right) of disease burden presents a comparative relation of interventions. The second row shows both control profiles $u_1(t), u_2(t)$ for all intervention Tests.	205
6.1	6×6 adjacency matrix and corresponding pixel visualization.	211
6.2	Pixel representations of graphs with $n = 10, 100, 1000$	212
6.3	Illustration of the SAS algorithm for two different scenarios.	221
6.4	Estimated graphons for real networks. First row: Collaboration network of arXiv astro physics (ca-AstroPh) $n = 1.8 \times 10^4$. Second row: who-trusts-whom network of Epinions.com (soc- Epinions1) $n = 7.5 \times 10^4$	223
6.5	Function $\kappa(w, w_*)$ in (6.4) for $\eta = 0.1$ (left), $\eta = 0.5$ (middle) and $\eta = 1$ (right). For all cases, $\beta = 0.5$	224
6.6	Equilibrium distribution (6.23) with $\mathcal{B}(x, y) = \frac{9}{16}(xy)^{-1/4}$ for several choices of the parameter $\lambda > 0$, $m_1 = 0$ (first row) and $m_1 = 0.2$ (second row), with $m_2 = 1$. We assume $\lambda = 0.1$. Opinion polarization is observed for $m_1 > 1$, whereas consensus formation corresponds to $m_1 \ll 1$	224
6.7	Comparison between the Boltzmann model (6.19) and the Fokker-Planck equation (6.12), computed via DSMC for $\lambda = 0.15, 0.5, 1.0, 1.5$ (row-wise), with $m_1 = 0$, $m_2 = 1$, and initial distribution (6.30). Simulations use $N = 10^5$ particles over $T \in [0, 5]$ and $\epsilon = \Delta t = 10^{-1}, 10^{-2}, 3 \times 10^{-4}$	225
6.8	Evolution of $f(x, w, t)$ for the separable graphon $\mathcal{B}_1$. Rows correspond to $\epsilon = 10^{-1}$ and 10^{-2} , respectively.	226
6.9	Evolution of $f(x, w, t)$ for the non-separable graphon $\mathcal{B}_2$. Rows correspond to $\epsilon = 10^{-1}$ and 10^{-2} , respectively.	226

6.10	Opinion dynamics on the ca-AstroPh network. Rows correspond to confidence thresholds $\Delta = 0.2$ and $\Delta = 0.8$. The first column shows snapshots of $f(x, w, t)$ at $t = 0, 10, 20, 30$, the second column reports the marginal opinion distribution, and the third column displays the final-time surface of $f(x, w, t)$	227
6.11	Opinion dynamics on the soc-Epinions-1 network. Rows correspond to confidence thresholds $\Delta = 0.2$ and $\Delta = 0.8$. The first column shows snapshots of $f(x, w, t)$, the second column reports the marginal opinion distribution, and the third column displays the final-time surface.	228
6.12	Epidemic model-Setting 1: Time evolution of the compartment masses (first row) and mean opinions (second row) computed from the SEIR kinetic model (6.2) for $\tau = 0.8, 10, 10^2$, using (6.32). Graphon (6.33) is employed. The domain $[0, 1] \times [-1, 1]$ is discretized with $N_x = 30$, $N_w = 10$, $\Delta t = 0.08$, over $[0, 30]$, with $\sigma^2 = 10^{-3}/4$ and $\beta = 0.5$	229
6.13	Epidemic model-Setting 2: Time evolution of the compartment masses (first row) and mean opinions (second row) computed from the SEIR kinetic model (6.2) for $\tau = 0.8, 10, 10^2$, using (6.32), with an estimated graphon from the ca-AstroPh network. The domain $[0, 1] \times [-1, 1]$ is discretized with $N_x = 30$, $N_w = 10$, $\Delta t = 0.08$, over $[0, 30]$, with $\sigma^2 = 10^{-3}/4$, $\beta = 0.5$, and $\eta = 1$	229
6.14	Epidemic model-Setting 3: Time evolution of the compartment masses (first row) and mean opinions (second row) computed from the SEIR kinetic model (6.2) for $\tau = 0.8, 10, 10^2$, using (6.32), with an estimated graphon from the soc-Epinions-1 network. The domain $[0, 1] \times [-1, 1]$ is discretized with $N_x = 30$, $N_w = 10$, $\Delta t = 0.08$, over $[0, 30]$, with $\sigma^2 = 10^{-3}/4$, $\beta = 0.5$, and $\eta = 1$	230
6.15	Time evolution of the marginal opinion distributions for susceptible (first column), exposed (second column), infected (third column), and recovered (fourth column) compartments. Rows correspond to Setting 1, Setting 2 and Setting 3, computed from the Fokker-Planck model (6.24) with $\tau = 10$. The domain $[0, 1] \times [-1, 1]$ is discretized with $N_x = 30$, $N_w = 15$, $\Delta t = 0.2$, over $[0, 30]$, with $\sigma^2 = 10^{-3}/4$ and $\beta = 0.5$	230
6.16	Graphon 14 case. Total marginal opinion density obtained with the bounded confidence model (6.34) and infection-dependent threshold $\Delta(\rho_I)$. Columns correspond to $\varepsilon_I = 5 \times 10^{-2}$, 3×10^{-2} , and 1×10^{-2} , respectively. The domain $[0, 1] \times [-1, 1]$ is discretized with $N_x = 30$, $N_w = 15$, using $\Delta t = 0.5$ over $t \in [0, 50]$	231
6.17	ca-AstroPh network. Total marginal opinion density obtained with the bounded confidence model (6.34) and infection-dependent threshold $\Delta(\rho_I)$, using the graphon estimated from the ca-AstroPh data. Columns correspond to $\varepsilon_I = 5 \times 10^{-2}$, 3×10^{-2} , and 1×10^{-2} , respectively.	232
6.18	soc-Epinions-1 network. Total marginal opinion density obtained with the bounded confidence model (6.34) and infection-dependent threshold $\Delta(\rho_I)$, using the graphon estimated from the soc-Epinions-1 data. Columns correspond to $\varepsilon_I = 5 \times 10^{-2}$, 3×10^{-2} , and 1×10^{-2} , respectively.	232
7.1	6×6 adjacency matrix and corresponding pixel visualization.	235

7.2	Schematic representation of a coupled network modeling opinion formation and disease spreading.	236
7.3	Schematic representation of the update rules for opinion formation and disease spreading.	237
7.4	Comparison between normalized degree distributions of $\mathcal{S}$ and $\mathcal{C}$ in the case of random graphs (left) and in the case of social contacts distributed as a lognormal and physical contacts distributed as a Weibull (right).	244
7.5	Bounded confidence parameter $\Delta = 2$. Final normalized number of infected nodes for different values of β and $q(\mathcal{S}, \mathcal{C})$ (left) and visual representation of the phase diagram (right). Random graphs case. . . .	245
7.6	Bounded confidence parameter $\Delta = 0.5$. Final normalized number of infected nodes for different values of β and $q(\mathcal{S}, \mathcal{C})$ (left) and visual representation of the phase diagram (right). Random graphs case. . . .	245
7.7	Bounded confidence parameter $\Delta = 2$. Final normalized number of infected nodes for different values of β and $q(\mathcal{S}, \mathcal{C})$ (left) and visual representation of the phase diagram (right). Coupled lognormal-Weibull distributions for social and contacts networks.	246
7.8	Bounded confidence parameter $\Delta = 0.5$. Final normalized number of infected nodes for different values of β and $q(\mathcal{S}, \mathcal{C})$ (left) and visual representation of the phase diagram (right). Coupled lognormal-Weibull distributions for social and contacts networks.	246
7.9	Bounded confidence parameter $\Delta = 0.5$. Final time opinion distributions for various values of the parameter $q(\mathcal{S}, \mathcal{C})$, in the case $\beta = 0.5$ (left), $\beta = 0.58$ (center), $\beta = 0.66$ (right).	247

List of Tables

2.1	Values of parameters utilized in the developed model (5.1), (2.33), and (2.37).	32
2.2	Sensitivity index of $\mathcal{R}_0$ of model (5.1)	43
2.3	Sensitivity index of $\mathcal{R}_0$ of model (2.33)	54
2.4	Sensitivity index of $\mathcal{R}_S, \mathcal{R}_R, \mathcal{R}_Q$ and $\mathcal{R}_0$ of model (2.37)	65
3.1	Values of parameters involved in the proposed SMAD model (5.1).	83
3.2	Parameter values used in the proposed model.	103
3.3	Error analysis of the numerical results achieved via Fractional Forward Euler Scheme (FFE) at $\aleph = 1$	129
3.4	Error analysis of the numerical results achieved via Adams-Bashforth Molten Scheme (ABM) at $\aleph = 1$	129
3.5	Error analysis of the numerical results achieved via Toufik-Atangana Scheme (TAS) at $\aleph = 1$ and $N = 2^{19}$ as reference solution	130
3.6	Error analysis of the numerical results achieved via Adams-type Predictor-Corrector Scheme (ATPC) at $\aleph = 1$ and $N = 2^{19}$ as reference solution	130
5.1	Parameters of Model	175
5.2	Representation of estimated parameters with FNN & PINN.	195
5.3	Efficiency analysis of model (5.30) with $\mathbb{E}$.	201
5.4	Cost Effective analysis of model (5.30) with IAR.	202
5.5	Cost Effective analysis of model (5.30) with ACER.	202
5.6	Cost Effective analysis of model (5.30) with ICER.	203

List of Abbreviations

FNN	Feed Forward Neural Network
PINN	Physics Informed Neural Network
NAR	Nonlinear Autoregressive Network
DINN	Disease Informed Neural Network
FPE	Fokker-Planck equation
ODE	Ordinary differential equations
PDE	Partial differential equations
LHS	Latin Hypercube Sampling
PRCC	Partial Rank Correlation Coefficient
SRD	Smoking related diseases
NRT	Nicotine replacement therapy
CBT	Cognitive behavioral therapy
ACT	Acceptance and commitment therapy
NGM	Next generation matrix technique
NFSI	Normalized forward sensitivity index
SMA	Social media addiction
SMAD	Addiction and depression on social media
UH	The ulam hyers stability
CDF	cumulative distribution function
IAR	Infection averted ratio
ACER	Average cost-effectiveness ratio
ICER	Incremental cost-effectiveness ratio
USVT	Universal singular value thresholding
SBA	Stochastic blockmodel approximations
SAS	Sorting-And-Smoothing algorithm
SIS	Susceptible-Infected-Susceptible
SIR	Susceptible-Infected-Recovered
SIAR	Susceptible-Infected-Asymptomatic-Recovered
SEIR	Susceptible-Exposed-Infected-Recovered

*For/Dedicated /To
My Loving Parents for their continuous moral support and
love.*

Chapter 1

Introduction

Complexity is a distinguishing feature of many natural, social, and engineering systems. They are defined by a large number of interacting elements showing emergent phenomena, which frequently arise from local interactions in nonlinear ways. Their diverse nature comprises the emergence of infectious diseases in various communities, the structuring of social interactions, collective behaviors of individual particles in physical dynamics, sharing information through digital mediums, traffic flow in urban areas, evaluation of opinion formation in societies, and determination of financial marketing. Considering their diversity, all mechanisms share common features like heterogeneity, uncertainty, large-scale interaction, and relapse process etc. A major challenge in investigating complex systems is to understand how interactions on a small scale among individuals accumulate to lead to an emergent pattern at the system level. Individual-level interaction at the micro scale in humans, molecules, particles, data format, and spreads across a wide range, resulting in collective dynamics at enormous scales. To observe this transition biologically as well as mathematically is significant. This process resembles the accurate forecasting of epidemic trajectory, which necessitates the understanding of how local contagion factors generate population-level outbreaks. Similarly, computing innovation or rumor dissemination in a social network demands linking bilateral contacts to broad adoption or consensus behaviors.

Modeling provides a mathematical foundation by which researchers can simplify complex phenomena to digestible visualizations, bridging the gap between theoretical knowledge and actual observation. One may test theories, investigate hypothetical potential, and forecast system dynamics beyond the empirical spectrum by using effective mathematical or computing models. The ability of predictions and explanations makes modeling essential for both expanding scientific understanding and directing policy choices in domains including public health, engineering, and economics. In epidemiology, the pioneering study of Kermack and McKendrick [163] presented the compartmental SIR (Susceptible-Infected-Recovered) model, which provides base for disease modeling. The foundation for contemporary epidemic theory was established by this straightforward system of ordinary differential equations (ODEs), which captured the important interaction between susceptible, infected, and recovered individuals. This paradigm has been expanded over decades to include random effects, demographic dynamics, geographical heterogeneity, and latency periods. In physics, the significant work of L.E. Boltzmann [56] defines the Boltzmann equation, which provides a mesoscopic description for defining the evaluation of particle velocity distribution over binary collisions in kinetic paradigms. This equation is a prime example of the kinetic modeling technique, which bridges the microscopic and macroscopic scales by studying the probability density of states rather than following each particle sequentially. Subsequently, the Fokker-Planck

equation [117, 236] extensively described diffusion and stochastic processes, extending the applicability of kinetic theory to statistical physics, molecule interaction in chemical processes, and disease dynamics. In the latter half of the 20th century, network science emerged, initially influenced by Erdos and Renyi's [108] study of random graphs. This model offered a mathematical structure for exploring thresholds and interaction in complex networks. Recent studies reveal that various real-world networks, from the internet and transportation systems to networks involving protein-protein interactions, display non-random structures (scale-free networks) and high clustering (small-world networks). These structural traits have a significant impact on dynamical processes, including the contact network in epidemiology, epidemic propagation, diffusion, and integration. Although each of these modeling strategies has some drawbacks, such as simple compartment models often ignoring the heterogeneity among individual interactions, kinetic models might turn analytically problematic when interactions are complicated, and network models encounter scalability complications when utilized on real-world complex systems.

Concurrent with these theoretical advancements, there has been an unmatched expansion in data accessibility and computing power during the past decades. Due to this, the paradigm for science has evolved to a data-driven system, whereby conventional mechanistic models are either replaced or supplemented by Machine Learning approaches. Currently, neural networks like Feed Forward Neural Network (FNN) [247], Physics Informed Neural Network (PINN) [242] are used extensively as universal function estimators because they can substitute models for complicated dynamical systems and identify hidden patterns in data. The rise of data-driven approaches in epidemics has made simple real-time forecasting and parameter calibration by using large, heterogeneous datasets like genomic sequences, the number of infectious individuals, financial market reporting, etc. However, the lack of real-world data and the noise it introduces continue to be barriers to accurate inference and forecasting. This has inspired the emergence of surrogate modeling techniques and data augmentation methodologies that integrate the advantages of data-driven flexibility with mathematical analysis.

The interdisciplinary convergence of all modeling fields and neural network strategies discussed above is associated with challenges as well as opportunities, as their hybridization is crucial for tackling contemporary scientific and societal issues. Epidemic models require the integration of biological insights with stochastic disease transmission, observing network heterogeneity, and leveraging data science strategies to narrow the gap between predictive and sparse observations. Similarly, kinetic theory can offer a potent approach for converting microscopic interactions into mesoscopic or macroscopic rules, establishing a connection between individual actions and collective results by use of network graphs. Networking theory may generate a solid domain to analyze the complex interaction in real-world dynamics by emphasizing the neural networks, like Graph Neural Network (GNN), whereas machine learning tools offer flexible solutions to deal with simulation complexity and data constraints. The COVID-19 pandemic serves as a stark reminder of the necessity of such interdisciplinary integration to understand the real-world dynamics. This highlighted that traditional compartmental models are helpful as an initial step but inadequate to accurately capture the complexity of real-world dynamics impacted by imperfect monitoring information, time-varying persecution measures, viral evolution, and different contact networks. More realistic representations were provided by kinetic and network-based models due to their ability to capture the small-scale interaction, but data-driven methods were needed for their validation

and calibration. Neural networks with data augmentation approaches enable filling the gap in initial uncertain data, reporting infectious individuals, and creating hypothetical simulations with synthetic data. The pandemic proved that no single strategy is sufficient for accurately communicating the real-time forecasting, which is useful for initial prosecution measures to control the disease spread.

The central motivation of this thesis lies within this interdisciplinary space. Specifically, it seeks the role of the traditional model in epidemics, how disease dynamics can be meticulously modeled using kinetic theory, further extended to structural population through networking modeling, and optimized with machine learning approaches linked with data augmentation and forecasting. The primary objective is to offer a framework that is simultaneously effective in practice and mathematically validated, and can respond to questions such as:

- In what manner are the individual-level microscopic interactions rigorously connected to mesoscopic and macroscopic representations of disease dynamics?
- How may network theory make effective modeling of couple dynamics over multiplex networks, and particularly graphon-based description, generalize the modeling of structured populations over an unlimited bound?
- How can data augmentation in initial observed data with neural networks be integrated over the epidemic models into mathematical frameworks without compromising theoretical integrity or accessibility?
- How traditional mathematical models with homogeneous mixing are significantly capable of predicting the persistence duration of disease dynamics like addiction to smoking dynamics, behavioral dynamics showing addiction & depressive behavior, and respective optimal control models?

1.1 Disease dynamics

Infectious disease dynamics provide a unified mathematical basis, central for epidemiology and mathematical biology to understand the process by which viruses spread through, persist, and ultimately die out throughout host populations. Foundations of all the modern complex disease models laid by the Kermack and McKendrick [163], who present the traditional SIR(Susceptible-Infected-Recovered) compartment model. The model divides the closed population N into three compartments: Susceptible (S), who are at risk of becoming vulnerable, Infected (I) consists of agents who can spread disease, and Recovered (R), with gain immunity, as displayed in Fig 1.1. Most compartment models are defined through a system of ODEs, driven by transmission rate β and recovery γ , providing deterministic outcomes that are mathematically tractable.

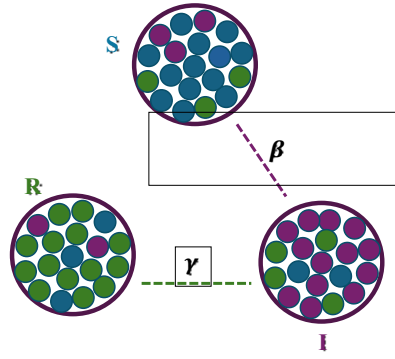


FIGURE 1.1: A schematics diagram of the SIR compartmental model.

The classical SIR ODEs with total population over time are as follows. It's often convenient to work with a normalized form where we use a fraction of the population, significant for theoretical analysis.

Dimensional form

$$\begin{aligned}\frac{dS}{dt} &= -\frac{\beta}{N}SI, \\ \frac{dI}{dt} &= \frac{\beta}{N}SI - \gamma I, \\ \frac{dR}{dt} &= \gamma I,\end{aligned}$$

Initial conditions:

$$S(0) = S_0, \quad I(0) = I_0, \quad R(0) = R_0,$$

and

$$S_0 + I_0 + R_0 = N.$$

Normalized form

$$\begin{aligned}\dot{s} &= -\beta si, \\ \dot{i} &= \beta si - \gamma i, \\ \dot{r} &= \gamma i,\end{aligned}$$

where

$$s = \frac{S}{N}, \quad i = \frac{I}{N}, \quad r = \frac{R}{N}.$$

Initial conditions:

$$s(0) = s_0, \quad i(0) = i_0, \quad r(0) = r_0,$$

and

$$s_0 + i_0 + r_0 = 1.$$

The corresponding homogeneous mixing reproduction number of the SIR model is written as

$$\mathbf{R}_0 = \frac{\beta}{\gamma}$$

The ratio indicates the anticipated number of secondary infections from a single infection in a vulnerable population. Linearising around the disease free equilibrium $i \rightarrow 0, s \approx s_0$ leads to growth condition

$$\beta s_0 - \gamma > 0 \iff \frac{\beta}{\gamma} s_0 > 1 \iff \mathbf{R}_0 s_0 > 1,$$

where considering $s_0 = 1$ give $\mathbf{R}_0 > 1$ indicate the endemic phase while $\mathbf{R}_0 < 1$ define the healthy phase. Real observation of epidemic final size can be formulated analytically by taking the fraction of $\dot{s}$ and $\dot{r}$

$$\frac{ds}{dr} = \frac{\dot{s}}{\dot{r}} = -\frac{\beta}{\gamma} s = -\mathbf{R}_0 s,$$

Integrating with separating variables, we get

$$s_{\infty} = e^{-R_0(r_{\infty}-r_0)}.$$

The expression reveals that an epidemic's end is driven by a decline in infectious individuals, not a complete lack of vulnerable individuals, as not all completely recovered. In the SIR model without vital dynamics, it is rigorously proven that the infectious compartment $I(t)$ tends to zero as time approaches infinity, indicating that $I_{\infty} = 0$. Since the expression

$$\frac{dR}{dt} = \gamma I(t) \geq 0,$$

indicates that the function $R(t)$ is monotone increasing and bounded above by N , it converges to a finite limit R_{∞} . By Integral for R , we get

$$R(t) - R(0) = \gamma \int_0^t I(s) ds,$$

and approaching the limit as $t \rightarrow \infty$ produces

$$\int_0^{\infty} I(s) ds = \frac{R_{\infty} - R(0)}{\gamma} < \infty.$$

Thus, $I(t)$, being a non-negative, continuous, and integrable function on $[0, \infty)$, approaches 0 as t tends to infinity. If it did not exist, continuity would result in the integral diverging, leading to a contradiction. As a result, the proportion of infectious individuals asymptotically diminishes, suggesting that the SIR model cannot have an endemic equilibrium in the absence of demographic parameters.

With the passage of time, numerous extensions in the SIR model, like SEIR, SEIRQ, etc, with the addition of exposed and quarantined classes in the sense of COVID-19, made the traditional model more complex and realistic. To capture biological realism, different interaction behaviors across communities are described by age-structured and metapopulation models, while expanded with stochastic formulations considering random fluctuation factors, to make effective public health policy, directing non-pharmaceutical actions during epidemics and immunization campaigns. Heterogeneity is still a significant obstacle regarding their progress. Real populations differ in contact rates, vulnerability, immunity, and cognitive reactions; they are not well-mixed groups of comparable individuals. Transmission is modulated by temporal changes in contacts, geographical grouping, and social structure. Ignoring these aspects puts models at risk for deliberate error in forecasts and calibrating parameters. Multi-group and network-based models have been developed as a result of the inclusion of heterogeneity; nodes in these models identify people or subpopulations, while edges indicate connections that may vary over time. These methods provide a deeper but more intricate definition by allowing the reproduction number to develop from the spectral features of the contact matrix rather than from a single scalar transmission ratio. Over the decade, the emerging use of traditional models still presents some open challenges [93, 50, 66], including integrating individual-level relations to community patterns, merging diverse networks, and calibration from noisy, uncertain data, which lead to the formulation of new computational and mathematical frameworks.

A natural approach to analyzing notable diversity in social behavior, characterized by differences in contact patterns, mobility, and connectivity, is to adopt a mesoscopic description that classifies the population using distribution functions spanning relevant microscopic variables. This leads to a kinetic formulation of epidemic dynamics, which makes a consistent relationship between individual-level interactions and generalized epidemic regulation. Suppose $w \in \mathbb{R}_+$ be a social variable that denotes personal features, like the number of contacts. The distribution of individuals with social attribute w in each compartments is define by the function $f_{\mathcal{J}}(w, t)$, where $\mathcal{J} \in \{S, I, R\}$ such that

$$f_S(w, t) + f_I(w, t) + f_R(w, t) = f(w, t), \quad \int_{\mathbb{R}_+} f(w, t) dw = 1$$

The macroscopic quantities are recovered as:

$$S(t) = \int_{\mathbb{R}_+} f_S(w, t) dw, \quad I(t) = \int_{\mathbb{R}_+} f_I(w, t) dw, \quad R(t) = \int_{\mathbb{R}_+} f_R(w, t) dw.$$

At the mesoscopic level, infection spread is modeled via binary interactions between susceptible and infected individuals. This defines the SIR system of kinetic equations as

$$\begin{aligned} \partial_t f_S(w, t) &= -\mathcal{K}(f_S, f_I)(w, t) + \mathcal{Q}_S(f_S, f_I, f_R)(w, t), \\ \partial_t f_I(w, t) &= \mathcal{K}(f_S, f_I)(w, t) - \gamma(w) f_I(w, t) + \mathcal{Q}_I(f_S, f_I, f_R)(w, t), \\ \partial_t f_R(w, t) &= \gamma(w) f_I(w, t) + \mathcal{Q}_R(f_S, f_I, f_R)(w, t). \end{aligned} \quad (1.1)$$

The disease transmission is described through the local incidence rate

$$\mathcal{K}(f_S, f_I)(w, t) = f_S(w, t) \int_{\mathbb{R}_+} \beta(v, w) f_I(v, t) dv$$

where the operator $\mathcal{Q}_{\mathcal{J}}$ represents the evaluation of social variable based on individual interactions, while maintaining the total population in each compartment, while $\gamma(w)$ is the recovery rate. Integrating the SIR kinetic system (1.1) with respect to w and assuming interaction operator $\mathcal{Q}_{\mathcal{J}}$ conserve mass

$$\int_{\mathbb{R}_+} \mathcal{Q}_{\mathcal{J}}(\cdot, \cdot) = 0$$

then under the homogeneous limit and closure

$$\beta(v, w) = \beta, \quad \gamma(w) = \gamma$$

The expressions in (1.1) are defined as

$$\int_{\mathbb{R}_+} \mathcal{K}(f_S, f_I)(w, t) dw = \beta SI, \quad \int_{\mathbb{R}_+} \gamma(w) f_I dw = \gamma I$$

leads to the classical SIR model. The SIR model is the macroscopic limit of a kinetic framework that explains individual interactions. The association advocates kinetic theory as an appropriate extension to include actual features such as various interactions and dynamic social networks.

1.2 Kinetic theory and Boltzmann equation

Modern societies confront complex challenges such as greenhouse gas emissions, plasma-based energies, traffic jams, air pollution, urban mobility, climate change, disease spread, and global finance. Each system involves multiple large-scale particles such as dust, aerosol, vehicles, data points, consumers, and human agents that interact in an extremely non-intuitive way as shown in Fig 1.2. Researchers are strictly focusing on current challenges due to the precise interaction of the individual particles, which has a direct impact on the behavior that emerges at large-scale dynamics. Despite advances in computing, most simulation of interacting particle systems is usually done by simplified approximated models to control the computation cost. A mesoscopic approach that captures the finer scale dynamics neglected due to limitations of purely macroscopic models, commonly referred to as kinetic description of the system. Kinetic theory proposes a mathematical base for describing the collective behavior of a system made up of several interacting agents. It allows us to accurately encode the probability of finding an agent in the phase space region using the statistical distribution function without requiring explicit particle description.

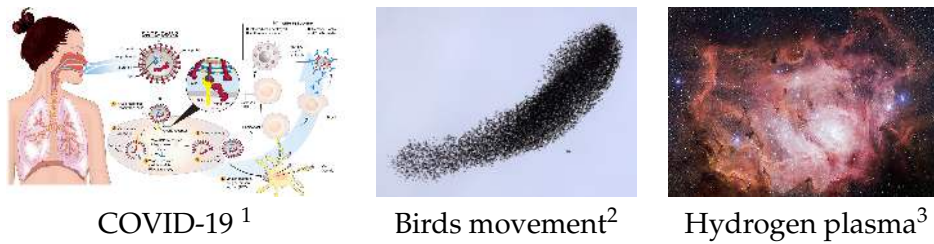


FIGURE 1.2: Visualization of some real-world phenomena of complex interacting particle dynamics.

The kinetic theory, as central to modern physics with application in biology, social, and technological contexts, originated from the classical thermodynamics' concept shaped by the study of [48, 31, 70]. J.C Maxwell long before Boltzmann, produced one of the first mathematical discoveries of what would become the kinetic theory of rarefied gases [200]. He made the case that Saturn's rings must be made up of many tiny satellites colliding with each other in a previous paper on the rings' stability. This article was written almost a decade before the groundbreaking studies of Maxwell and Boltzmann, which later introduced the Boltzmann equation for rarefied gases in its modern form [201, 56]. It is historically the first mathematical occurrence of a kinetic equation. Since it was originally designed to depict a gas as freely moving particles that collided instantly. In mathematical terms, these systems regulate the formation of a particle distribution in six-dimensional position by integrating the linear transport with several stiff and nonlinear interaction components expressed as Boltzmann-like systems. More specifically, the evaluation of distribution function $f^\epsilon = f^\epsilon(x, v, t)$ for $t \geq 0$, $x \in \Pi \subset \mathbb{R}^d$ for $d \leq 3$ and $v \in \mathbb{R}^d$ with given non negative initial condition f_0 , solution to the scaled, initial boundary value problem

¹<https://en.wikipedia.org/wiki/COVID-19>

²https://en.wikipedia.org/wiki/Swarm_intelligence

³https://en.wikipedia.org/wiki/Astrophysical_plasma

$$\epsilon^{\alpha_1} \partial_t f^\epsilon + v \cdot \nabla_x f^\epsilon = \frac{1}{\epsilon^{\alpha_2}} \mathcal{Q}(f^\epsilon), \quad \text{where} \quad f^\epsilon(x, v, 0) = f_0(x, v).$$

refers to a Boltzmann-type model where $\mathcal{Q}$ is the collision operator, ϵ is an marginal quantity that describes the gas's rarefaction. Since the open set Π is a limited Lipschitz-continuous region of $\mathbb{R}^d$, boundary requirements must also be included in the above model. The study of various asymptotic tendencies will be made possible by the non-negative parameters α_1 and α_2 . Now we precisely explain the mathematical perspective behind the kinetic equation in the next step.

1.2.1 Kinetic equation: Mathematical perspective

Nowadays, a vast range of systems of physical and biological significance is covered by kinetic equations. The mathematical formulation of kinetic equations depends on different levels of description. Interacting particle systems are ubiquitous in nature. Particle dynamics, which captures the behavior of individual particles, may be employed to describe the motion of particle systems like Newton equations or Hamilton's equations at the *microscopic level*. Considering the challenge of solving numerous problems simultaneously, which might be difficult, particle dynamics is not feasible. Models such as the Euler or Navier-Stokes equations explain averaged values, local density, momentum, and energy at the *macroscopic level*. However, they depend on equilibrium constraints that might not be genuine or simply accepted analytically. The division between the levels of description must be bridged. This issue was resolved by introducing the *mesoscopic level*, so-called kinetic level, between microscopic and macroscopic scales. In this level, the models are represented by Boltzmann and mean field equations, which describe the time evolution of a system with many particles using a statistical distribution function, which defines the density of particles in phase space. Now we discuss the mathematical description of all scales, respectively.

Microscopic level

Let us consider a system of N interacting particles moving, each of which is described at time $t \in [0, T]$ by its position $x_i(t)$ and velocity $v_i(t)$, for $i = 1, 2, \dots, N$. The entire evolution of the microscopic state of the system is defined through Newton's equation of motion as

$$\dot{x}_i(t) = v_i(t), \quad \dot{v}_i(t) = f_i(x_1(t), \dots, x_N(t)),$$

where f_i presents the total force acting on the i^{th} particle. This equation represents a closed $2N$ system of ODEs describing the precise deterministic dynamics. The force is conservative across various physical and biological contexts, meaning that it originates from an interaction potential $\Xi : (\mathbb{R}^3)^N \rightarrow \mathbb{R}$, resulting in

$$f_i(x_1, x_2, \dots, x_N) = -\nabla_{x_i} \Xi(x_1, \dots, x_N).$$

where potential Ξ commonly works as a pairwise (binary) interaction term illustrating particle-particle interactions, then the total potential is given as

$$\Xi(x_1, \dots, x_N) = \frac{1}{2} \sum_{\substack{j,k=1 \\ j \neq k}}^N \Xi_{\text{int}}(x_j - x_k) + \sum_{j=1}^N \Xi_{\text{ext}}(x_j).$$

To ensure total momentum conservation, the interaction potential between particles, Ξ_{int} , is dependent on relative displacement $x_j - x_k$. The external potential Ξ_{ext} characterizes background fields such as electromagnetic, gravitational, or confining trap forces. The microscopic description is typically a perfect representation of the many-body dynamics, but when N is large, like the Avogadro number, its direct numerical simulation is prohibitive or impossible.

Mesoscopic level (Kinetic formulation)

The mesoscopic scale defines a large scale partial system at time t in single particle phase statistically by means of a non-negative distribution function in which the total density of particles is situated at location $x \in \Pi$ and having velocity $v \in \mathbb{R}^3$ written as

$$f = f(x, v, t).$$

This precisely describe the number density of particle at t in small volume $dx dv$ exist in Φ with velocities in Ψ about point (x, v) given as

$$\iint_{\Phi \times \Psi} f(x, v, t) dx dv,$$

with measurable subset of position $\Phi \subset \mathbb{R}^3$ and velocity $\Psi \subset \Pi$. Additionally, the associated macroscopic quantities like macroscopic/particle density, mean velocity utilizing the current density, and local kinetic energy may be defined from a given distribution function f , respectively

$$\rho(x, t) = \int_{\mathbb{R}^3} f dv, \quad \rho u(x, t) = \int_{\mathbb{R}^3} v f dv, \quad \mathcal{E}(x, t) = \frac{1}{2} \int_{\mathbb{R}^3} |v|^2 f dv.$$

The temperature is written as

$$\rho T(x, t) = \frac{1}{d} \int_{\mathbb{R}^3} |v - u|^2 f dv,$$

with spatial dimension d . The energy dissipation, respectively, with kinetic and internal energy, is described as

$$\text{Total Energy} = \frac{1}{2} \rho |u|^2 dx + \frac{d}{2} \rho T dx.$$

Global quantity like total Mass $\mathcal{M}(t)$ and entropy $\mathcal{H}(t)$ is given as

$$\mathcal{M}(t) = \iint_{\Pi \times \mathbb{R}^3} f(t, x, v) dv dx = \int_{\Pi} \rho(t, x) dx,$$

$$\mathcal{H}(t) = \iint_{\Pi \times \mathbb{R}^d} f(t, x, v) \log(f(t, x, v)) dv dx.$$

Macroscopic level

Macroscopic level consists of the continuous description of a large-scale particle system, applying Newton's laws to infinitesimal control volumes within the spatial domain Π [171], leading to partial differential equations (PDEs) that govern measurable bulk quantities like

$$\text{Macroscopic density: } \rho : (t, x) \mapsto \rho(t, x) \in [0, \infty),$$

$$\text{Mean velocity: } u : (t, x) \mapsto u(t, x) \in \mathbb{R}^3,$$

$$\text{Temperature: } T : (t, x) \mapsto T(t, x) \in [0, \infty).$$

The traditional Euler equations for inviscid flow or the Navier-Stokes equations, which incorporate viscosity and heat conduction, connect these topics in the context of gas dynamics.

1.2.2 Non-Interacting particles system

Starting with the most basic case of *non-interacting particles* is informative when attempting to formulate the kinetic equation achieved by the one-particle distribution function $f = f(x, v, t)$. The internal force disappears in this configuration as the particles do not apply forces to one another, i.e. $f_{\text{int}} = 0$. As a result, only an external force field $f_{\text{ext}}(x, t)$ may affect each particle. Consider that $(\mathcal{X}(t), \mathcal{V}(t))$ represents the phase space trajectory of a particle that originates at time $t = 0$ from the location (x, v) to describe the flow's properties. Newton's equations simplify to following expression with initial data $\mathcal{X}(0) = x$, $\mathcal{V}(0) = v$, since pairwise interactions are absent,

$$\dot{\mathcal{X}}(t) = \mathcal{V}(t), \quad \dot{\mathcal{V}}(t) = f_{\text{ext}}(\mathcal{X}(t), t). \quad (1.2)$$

Every particle that starts from a single initial phase, the space point (x, v) , follows the same path. The phase space vector field $f(x, v, t) := (v, f_{\text{ext}}(x, t))$ corresponds to Eq (1.2). Since this field's divergence with regard to (x, v) disappears, indicating flow is incompressible i.e

$$\nabla_{x,v} \cdot f = \nabla_x \cdot v + \nabla_v \cdot f_{\text{ext}}(x, t) = 0,$$

Thanks to Liouville's theorem, the volume element $dx dv$ is preserved along the direction $(\mathcal{X}(t), \mathcal{V}(t))$. Keep in mind the conservation property of one particle distribution function f , the value of f remains constant (volume element $dx dv$ does not change) throughout each characteristic curve because the number of particles included in every moving phase-space volume is conserved show the vector field $(v, f_{\text{ext}}(x, t))$ is divergence free :

$$\frac{d}{dt} f(\mathcal{X}(t), \mathcal{V}(t), t) = 0. \quad (1.3)$$

The chain rule applied to (1.3) yields

$$\frac{\partial f}{\partial t} + \dot{\mathcal{X}}(t) \cdot \nabla_x f + \dot{\mathcal{V}}(t) \cdot \nabla_v f = 0.$$

The Eq (1.2) can be substituted to produce

$$\partial_t f + v \cdot \nabla_x f + f_{\text{ext}}(x, t) \cdot \nabla_v f = 0. \quad (1.4)$$

This is the *Vlasov equation*, often known as the "free transport equation," for non-interacting particles that are exclusively affected by the external force field F_{ext} . It conveys the idea that the distribution function is merely advanced along the trajectories produced by (v, F_{ext}) in phase space.

1.2.3 Mean field particles system

Consider an N -element system of identical particles that couple by a smooth pairwise potential without any external force field i.e $f_{\text{ext}} \equiv 0$. The objective is to determine the kinetic equation for the one-particle distribution function

$$f = f(x, v, t), \quad (x, v) \in \mathbb{R}^3 \times \mathbb{R}^3,$$

which provides the particle density at time t . Using the microscopic scale, every particle complies with Newton's equations

$$\dot{\mathcal{X}}_i(t) = \mathcal{V}_i(t), \quad \dot{\mathcal{V}}_i(t) = \frac{1}{N} \sum_{\substack{j=1 \\ j \neq i}}^N f_{\text{int}}(\mathcal{X}_i(t) - \mathcal{X}_j(t)). \quad (1.5)$$

where the pairwise contact force is f_{int} . The traditional *mean field scaling* is represented by the factor $1/N$, which guarantees a finite limit as $N \rightarrow \infty$. Mean field limit emphasizes that when N is sufficiently big, it is reasonable to believe that a continuous *mean field* may approximate the impact of all other particles on a given one particular. So, one particle distribution function f with spatial marginal

$$\rho(x, t) = \int_{\mathbb{R}^3} f(x, v, t) dv$$

define the particle density at position x and time t . Next, the average force acting on a particle at x is

$$f_m(x, t) = \int_{\mathbb{R}^3} \int_{\mathbb{R}^3} f_{\text{int}}(x - y) f(y, w, t) dw dy = \int_{\mathbb{R}^3} f_{\text{int}}(x - y) \rho(y, t) dy.$$

This follows directly, as $\rho(y, t)$ is the simple result of the integral in w . Moving to define the kinetic equation, although a smooth mean-field force now mediates particle interaction, the phase-space vector field $(v, f_m(x, t))$ is still divergence-free. Implies Liouville's theorem, we say that f is constant along the characteristic flow. Then f is solution of

$$\partial_t f + v \cdot \nabla_x f + f_m(x, t) \cdot \nabla_v f = 0. \quad (1.6)$$

This formulation explains how the distribution function is transported in phase space when the self-consistent mean-field force f_m is applied. Usually, a smooth interaction potential $\mathcal{P}_{\text{int}} : \mathbb{R}^3 \rightarrow \mathbb{R}$ yields the bilateral force, resulting in

$$f_{\text{int}}(x) = -\nabla_x \mathcal{P}_{\text{int}}(x).$$

In this instance, the gradient of a *mean potential* $\mathcal{P}_m$ may be expressed as the mean field f_m

$$f_m(x, t) = -\nabla_x \mathcal{P}_m(x, t), \quad \mathcal{P}_m(x, t) := \int_{\mathbb{R}^3} \mathcal{P}_{\text{int}}(x - y) \rho(y, t) dy. \quad (1.7)$$

Thus, Eq (1.6), the Vlasov equation, produces

$$\partial_t f + v \cdot \nabla_x f - (\nabla_x \mathcal{P}_m(x, t)) \cdot \nabla_v f = 0. \quad (1.8)$$

The self-consistent evolution of the particle distribution when every individual particle encounters the collective field produced by the whole system is captured by equations (1.6) to (1.8). It is fundamental to stellar dynamics, plasma physics, and other models of collective behavior in the social and biological sciences.

1.2.4 The Boltzmann equation

When particles do not interact with each other, the linear Vlasov equation emerges automatically, while in the case of interacting particles, one can formally and conceptually derive the nonlinear kinetic equation with mean field limit $\mathcal{N} \rightarrow \infty$ as done in Sec 1.2.2-1.2.3. For more details, see Refs [122, 154, 250]. Deriving reliable nonlinear kinetic models from microscopic dynamics is still an important and unsolved scientific problem.

Assume the joint $\mathcal{N}$ particle distribution with position and velocity is

$$f_{\mathcal{N}} = \{t, x_1, x_2, \dots, x_{\mathcal{N}}, v_1, v_2, \dots, v_{\mathcal{N}}\}.$$

We acquire the one-particle marginal of $F_{\mathcal{N}}$ by integrating out the other $\mathcal{N} - 1$ particles to concentrate on a single particle

$$f_{\mathcal{N}}^{(1)}(t, x, v) = \int_{(\mathbb{R}^d \times \mathbb{R}^d)^{\mathcal{N}-1}} f_{\mathcal{N}}(t, x, x_2, \dots, x_{\mathcal{N}}, v, v_2, \dots, v_{\mathcal{N}}) dx_2 \cdots dx_{\mathcal{N}} dv_2 \cdots dv_{\mathcal{N}},$$

where $f_{\mathcal{N}}^{(1)}(t, x, v)$ define the probability density of a desired particle at position x and velocity v regardless the other particles locations. Consider that there's an infinite number of particles right now. If we can demonstrate that as $\mathcal{N} \rightarrow \infty$ the expression $f_{\mathcal{N}}^{(1)}(t, x, v)$ converges to limit

$$f(t, x, v) = \lim_{\mathcal{N} \rightarrow \infty} f_{\mathcal{N}}^{(1)}(t, x, v).$$

shows that $f(t, x, v)$ is a single particle distribution of infinite particle gases. The aim is to show that the limiting f converges to the kinetic equation. However, $f_{\mathcal{N}}^{(1)}$ is connected to the density of two particles, $f_{\mathcal{N}}^{(2)}$. This is connected to the density of three particles, and so forth. The BBGKY hierarchy is this infinite series of connected equations. Therefore, a molecular chaos concept is required to obtain a closed equation for f , which states that two randomly selected particles' locations and velocities are (roughly) independent for an infinite number of particles.

$$f_{\mathcal{N}}^{(2)}(x, v, y, w) \approx f(x, v) f(y, w) \quad \text{as} \quad \mathcal{N} \rightarrow \infty.$$

The hierarchy can close under this presumption, providing a unique equation for f . Two major kinetic models emerge, based on the scale and interaction spectrum

- Long-range interaction yields a mean field equation describing the motion of all particles under a self-consistent field (no direct collision).
- Short-range interaction produces a Boltzmann-type or collisional kinetic equation in which particle interaction is under a binary collision model.

- Hybrid models, which combine both features of the mean field and the Boltzmann equation to study the mathematical and physical systems.

The Boltzmann equation connects classical kinematics to macroscopic dynamics by following the one-particle distribution in phase space to characterize a diluted gas. It describes the way binary collisions preserve energy and momentum despite distributing velocities. This evolution is demonstrated in two standard contexts: the physical mechanical model of hard spheres and theoretical binary collision theory. Geometric representations of both cases are shown in Fig 1.3.

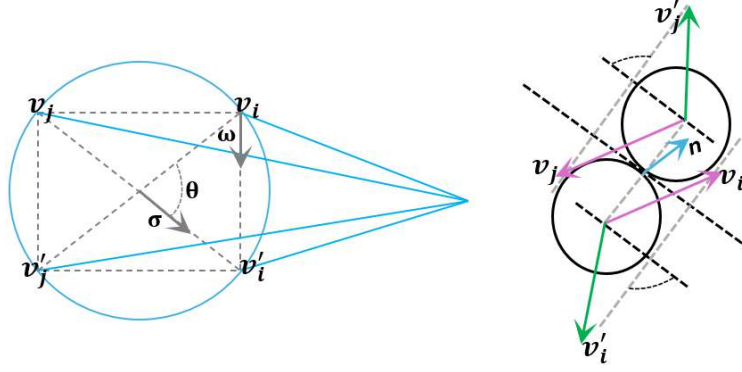


FIGURE 1.3: Geometry of Boltzmann dynamics. The first shows the elastic collisions, while the second describes the case of a hard sphere.

Gas particles in binary collision dynamics depend on several assumptions made by Boltzmann, which we are going to define here. The premise is that particles interact through binary collisions, in which the trajectory of two particles swiftly and significantly diverges. The gas is sufficiently diluted to overlook interactions between more than two particles. Furthermore, collisions are confined in time and space, occurring at a specific location (t, x) . The collision is elastic as it satisfies the conservation of momentum

$$v_i + v_j = v'_i + v'_j$$

and conservation of energy

$$v_i^2 + v_j^2 = v'^2_i + v'^2_j$$

where $v_i, v_j \in \mathbb{R}^d$ are velocity before collision and $v'_i, v'_j \in \mathbb{R}^d$ are velocity after collision. We can characterize each solution with about $d - 1$ free parameters since we have $d + 1$ equations and $2d$ unknowns. Using the elastic collision mechanism, we define the solution in ω representation (impact direction)

$$v'_i = v_i - (\omega \cdot (v_i - v_j))\omega \quad \text{and} \quad v'_j = v_j + (\omega \cdot (v_i - v_j))\omega,$$

where $\omega \in \mathbb{S}^{d-1}$. Another way to written in σ representation (scattering angle)

$$v'_i = \frac{1}{2} \left((v_i + v_j) + \sigma |v_i - v_j| \right) \quad \text{and} \quad v'_j = \frac{1}{2} \left((v_i + v_j) - \sigma |v_i - v_j| \right)$$

for given $\omega \in \mathbb{S}^{d-1}$. It is believed that collisions are microreversible, which means that changes in time and velocity do not affect their dynamics. According to Boltzmann's molecular chaos premise, two particles impacted by a collision have opposite velocities. This suggests that the tensor product of their individual velocities represents the overall likelihood distribution of their velocities.

Hard sphere dynamics comes with the assumption of the particle to be a solid sphere that remains non-interacting until direct contact. The interaction potential is not smooth, compared to the Vlasov formulation, with all pairwise distances obeying

$$f_{int}(x_i - x_j) = 0, \quad \forall x_i, x_j \text{ such that } |x_i - x_j| > \epsilon,$$

where ϵ is the diameter and the precollision center and velocity of two distinct spheres are (x_i, v_i) and (x_j, v_j) . Particles i and j collide when $|x_i - x_j| = \epsilon$. Assume

$$n = \frac{x_i - x_j}{|x_i - x_j|} \in \mathbb{S}^{d-1}$$

be the outward normal at contact (from j to i). The hard sphere case satisfies the conservation of energy and momentum rule, like binary collision particles. The post-collisional velocities solution can be defined using a conserved quantity with unit normal n and conservation of angular momentum

$$v'_i = v_i - [n \cdot (v_i - v_j)] n, \quad \text{and} \quad v'_j = v_j + [n \cdot (v_i - v_j)] n,$$

where the dynamics discrete in time, velocities instantly shift from form v_i, v_j to v'_i, v'_j at collision times. Supposing ω as unit vector with $\omega = g - 2n(n \cdot g)$ and $g = \frac{v_i - v_j}{|v_i - v_j|}$ we write in other form

$$v'_i = \frac{1}{2} \left((v_i + v_j) + \omega |v_i - v_j| \right) \quad \text{and} \quad v'_j = \frac{1}{2} \left((v_i + v_j) - \omega |v_i - v_j| \right).$$

Collision operator

Keep in mind that all particles released from the same place (x, v) have the same trajectory with no collisions

$$\dot{\mathcal{X}}(t) = \mathcal{V}(t), \quad \dot{\mathcal{V}}(t) = 0.$$

and subsequently, along the particle trajectory, the distribution function f is invariant. To model the impact of collision, a term $\mathcal{Q}(f)$ is introduced to compute the variation of f induced by collision interaction, written as

$$\frac{d}{dt} f(\mathcal{X}(t), \mathcal{V}(t), t) = \left(\partial_t f + v \cdot \nabla_x f \right) |_{(\mathcal{X}(t), \mathcal{V}(t), t)} = \mathcal{Q}(f) |_{(\mathcal{X}(t), \mathcal{V}(t), t)},$$

where $\mathcal{Q}(f)$ is the collision operator. The kinetic equation for the case of *hard sphere* is written as

$$\partial_t f + v \cdot \nabla_x f = \mathcal{Q}(f).$$

In case of bilinear interaction structure $\mathcal{Q} = \mathcal{Q}(f, f)$ and required the Boltzmann Grad limit with $\mathcal{N} \rightarrow \infty$ as diameter of sphere $d \rightarrow 0$ such that $\mathcal{N} d^2$ remain constant. Now, the hard sphere collision operator is defined as

$$\mathcal{Q}(f, f) = \int_{\mathbb{R}^d \times \mathbb{S}^{d-1}} |v_i - v_j| \left(f(v'_i) f(v'_j) - f(v_i) f(v_j) \right) d\omega dv_j \quad (1.9)$$

Similarly, for the gas particles, Eq (1.9) describes a non-local bilinear operator.

$$\mathcal{Q}(f, f) = \int_{\mathbb{R}^d \times \mathbb{S}^{d-1}} |v_i - v_j| \sum (|v_i - v_j|, \cos \theta) \left(f(v'_i) f(v'_j) - f(v_i) f(v_j) \right) d\sigma dv_j \quad (1.10)$$

satisfy the σ representation. The term $\Sigma > 0$ represent the cross section with

$$\cos \theta = \left\langle \frac{v_i - v_j}{|v_i - v_j|}, \sigma \right\rangle.$$

The operator $\mathcal{Q}(f, f)$ in Eqs. (1.9)–(1.10) can be split into two terms, which are given by

$$\mathcal{Q}(f, f) = \mathcal{Q}^+(f, f) + \mathcal{Q}^-(f, f).$$

The loss term $\mathcal{Q}^-(f, f)$ describes the decline of the distribution function f when the velocity of particles shifts from $v \rightarrow v'$ in the collision mechanism, while the gain term $\mathcal{Q}^+(f, f)$ models the propagation of distribution function f caused by particles shifting from any other velocity to v . In the generalization of the interaction process, despite not having a comprehensive mathematical theory, the Boltzmann equation is often used for smooth interaction potentials, where the collision operator is expressed as

$$\mathcal{Q}(f, f) = \int_{\mathbb{R}^3 \times \mathbb{S}^2} \mathcal{B}(v_i, v_j, \omega) \left(f(v'_i) f(v'_j) - f(v_i) f(v_j) \right) d\omega dv_j \quad (1.11)$$

The general form of kernel $\mathcal{B}(v_i, v_j, \omega)$ is given as

$$\mathcal{B}(v_i, v_j, \omega) = b_\gamma \cos \theta |v_i - v_j|^\gamma, \quad \gamma = \frac{s+5}{s-1}, \quad \cos \theta = \left\langle \frac{v_i - v_j}{|v_i - v_j|}, \omega \right\rangle.$$

H Theorem

The collision operator preserves the momentum, energy, and mass for f and test function $\varphi \in \mathcal{C}_c^0(\mathbb{R}^d)$

$$\int_{\mathbb{R}^d} \mathcal{Q}(f, f) \varphi(v) dv = 0, \quad \varphi(v) = 1, v^x, v^y, v^z, |v|^2,$$

and satisfy the H Theorem [290] with kinetic entropy inequality

$$\mathcal{H}[f] = \int_{\mathbb{R}^d} \mathcal{Q}(f, f) \log(f(v)) dv \leq 0,$$

and entropy dissipation

$$\mathcal{I}[f] = \int_{\mathbb{R}^d} v \mathcal{Q}(f, f) \log(f(v)) dv \leq 0,$$

then it satisfies the following expression

$$\partial_t \mathcal{H}[f] + \nabla_x \cdot \mathcal{I}[f] \leq 0. \quad (1.12)$$

All these properties proved by taking test function $\varphi \in \mathcal{C}_c^0(\mathbb{R}^d)$

$$\int_{\mathbb{R}^d} \mathcal{Q}(f, f) \varphi(v) dv = -\frac{1}{4} \int_{\mathbb{R}^d \times \mathbb{R}^d \times \mathbb{S}^{d-1}} \mathcal{B}(v_i, v_j, \omega) \left(f'_i f'_j - f_i f_j \right) \left(\varphi'_i \varphi'_j - \varphi_i \varphi_j \right) d\omega dv_i dv_j$$

This inequality is proved by assuming micro reversibility, and the Jacobian transformation is equal to one. Moreover, the test function is collision invariant with the condition on Eq (1.12) that kinetic entropy $\mathcal{H}[f] = 0$ iff f is Maxwellian distribution $\mathcal{M}$ that we describe in next step.

Maxwellian distribution

One of the most crucial objects in kinetic theory is the Maxwellian distribution function $\mathcal{M}$. Considering the gas temperature (T), density (ρ) and mean velocity (u)

$$\rho = \int_{\mathbb{R}^d} f dv, \quad u = \int_{\mathbb{R}^d} v f dv, \quad T = \frac{1}{3\mathcal{R}_\rho} \int_{\mathbb{R}^d} (v - u)^2 f dv,$$

we conclude

$$\mathcal{M}_{(\rho,u,T)}(v,t) = \frac{\rho}{(2\pi\mathcal{R}T)^{3/2}} \exp\left(-\frac{|u-v|^2}{2\mathcal{R}T}\right), \quad (1.13)$$

with $\mathcal{R} = \frac{K_B}{m}$, m is the mass of particle and K_B is the Boltzmann constant. Using the Boltzmann $\mathcal{H}$ Theorem, any function f such that $\mathcal{Q}(f,f) = 0$ is a Maxwellian. If we write the $\mathcal{H}$ function, we have

$$\mathcal{H}(f) = \int_{\mathbb{R}^d} f \log(f) dv,$$

$$\partial_t \int_{\mathbb{R}^d} \mathcal{H}(f) dx = \int_{\mathbb{R}^d \times \mathbb{R}^d} \mathcal{Q}(f,f) \log(f) dv dx.$$

A monotonic decrease in the $\mathcal{H}$ function is observed until f reaches the Maxwellian condition. More precisely, Maxwellian provides a bridge between mesoscopic and macroscopic descriptions. Particularly whenever

$$(\rho, u, T) \longmapsto \mathcal{M}_{(\rho(x,t), u(x,t), T(x,t))}(v, t),$$

solves the free transport equation and then (ρ, u, T) define the hydrodynamics approximation.

Hydrodynamics approximation

The Boltzmann equation for particle distribution $f(x, v, t)$ is used to derive macroscopic fluid equations from the kinetic description.

$$\partial_t f + v \cdot \nabla_x f = \mathcal{Q}(f, f),$$

where $\mathcal{Q}(f, f)$ is the collision operator with its significant property as collision invariants, namely

$$\varphi(v) = 1, v^x, v^y, v^z, |v|^2.$$

Whenever $\varphi(v)$ is added to the Boltzmann equation and integrated over v , the collision term vanishes

$$\int_{\mathbb{R}^d} \mathcal{Q}(f, f) \varphi(v) dv = 0,$$

leads to the derivation of a system of macroscopic conservation laws

$$\int_{\mathbb{R}^d} f \varphi(v) dv + \nabla_x \left(\int_{\mathbb{R}^d} v f \varphi(v) dv \right) = 0,$$

express the conservation of mass, momentum, and energy. Since, conservation laws are not closed as flux $\int_{\mathbb{R}^d} v f \varphi(v) dv$ influenced by higher-order velocity moments of f , but they cannot be understood without knowing more about f . Assuming that the distribution function is approximately a local Maxwellian $\mathcal{M}_{(\rho,u,T)}$ at each point (t, x) is the most straightforward approach. When $f \approx \mathcal{M}_{(\rho,u,T)}$, higher order velocity moments may be directly expressed in terms of ρ , u , and T as indicated by the conservation rules to derive the compressible Euler equations

$$\begin{aligned} \partial_t \rho + \nabla_x \cdot (\rho u) &= 0, \\ \partial_t (\rho u) + \nabla_x \cdot (\rho u \otimes u + p) &= 0, \\ \partial_t \mathcal{E} + \nabla_x \cdot (\mathcal{E} u + p u) &= 0, \\ p &= \rho T = \frac{1}{3} (2 \mathcal{E} - \rho |u|^2). \end{aligned} \tag{1.14}$$

A more accurate hydrodynamic approximation of the Boltzmann moment system may be obtained by employing the Chapman-Enskog expansion to maintain first-order adjustments, leading to compressible Navier-Stokes equations and improving the Euler closure, which eliminates tiny variations from the Maxwellian.

1.2.5 Some kinetic models of interest

After a precise description of kinetic equations and derivation of the Boltzmann equation, we provide some kinetic models as examples that capture in a unified way the dynamics of charge particles in plasma physics, collective behavior in biological swarming, and the spread of infectious disease in a population covered by epidemiological dynamics.

Bhatnagar–Gross–Krook (BGK) model: The Boltzmann equation describes the evolution of the distribution function f of dilute gas particles with the Boltzmann collision operator. BGK model [53] replaces the bilinear collision operator with a simple nonlinear relaxation operator directing the system towards a local Maxwellian equilibrium.

$$\mathcal{Q}_{BGK}(f) = \nu(\rho) \left(\mathcal{M}_{(\rho,u,T)}(f) - f \right),$$

where $\nu(\rho)$ is the collision frequency and $\mathcal{M}_{(\rho,u,T)}$ is the Maxwellian distribution describe in Subsec 1.2.4. The BGK model preserves the conservation law and satisfies the $\mathcal{H}$ Theorem properties, but it does not adequately capture all of the transport coefficients in the whole Boltzmann equation. In particular, the Prandtl number whose is 1 in the BGK model, whereas the correct value for monotonic hard-sphere gas is $\frac{2}{3}$ recovered using $\nu = \nu(\rho, u)$ and Ellipsoidal Statistical BGK. Consequently, the connection between viscosity and thermal conductivity is not correctly predicted by the Navier-Stokes limit estimated by the BGK model. Correction could be utilizing $\nu = \nu(\rho, |v|)$ having dependency on density and molecular speed. A precise extension, Ellipsoidal Statistical BGK (ES–BGK) model [28] substitutes an anisotropic Gaussian equilibrium for the Maxwellian.

$$M_{ES}(f) = \frac{\rho}{\sqrt{\det(2\pi\Theta)}} \exp \left[-\frac{1}{2} (v - u)^\top \Theta^{-1} (v - u) \right],$$

where the required Prandtl number is matched by the temperature tensor, denoted by Θ . For monatomic gases, ES-BGK returns $\mathcal{P}_r$ correctly when the parameters are chosen appropriately.

Kinetic Vicsek model: This is a particle-based model of self-supporting entities that move at a steady speed while subjected to random noise, including fish, birds, germs, or synthesized particles [289]. The model changes from a microscopic to a mean-field kinetic description as the number of individuals rises, and the kinetic Vicsek equation serves as the limiting PDE of distribution function $f(x, w, t)$ of individuals at time $t > 0$, position $x \in \mathbb{R}^d$, and with orientation $w \in \mathbb{S}^{d-1}$, written as

$$\partial_t f + \nabla_x \cdot (\omega f) + \nabla_\omega \cdot [\mathcal{P}_{\omega^\perp}(\mathcal{J}_f(x, t)f)] - \mathcal{D}\Delta_\omega f = 0,$$

wherein $\nabla_\omega \cdot [\mathcal{P}_{\omega^\perp}(\mathcal{J}_f f)]$ denotes each particle's propensity to rotate its orientation in a given direction of the local mean, $\mathcal{J}_f$. To maintain orientation on $\mathbb{S}^{d-1}$, the operator $\mathcal{P}_{\omega^\perp}$ extends onto the tangent plane of the sphere at ω . An angular diffusion with rotational coefficients of diffusion $\mathcal{D} > 0$ that models random variations in direction is $\mathcal{D}\Delta_\omega f$ on the sphere. The local average velocity orientation

$$\mathcal{J}_f(x, t) = \int_{\mathbb{R}^d \times \mathbb{S}^{d-1}} \mathcal{K}(|x - y|) \omega' f(y, \omega', t) d\omega' dy.$$

The integral uses the interaction kernel $\mathcal{K}(|x - y|)$ to compensate for the geographic effect and calculates a weighted mean of the headings ω' of adjacent neighbors. The degradation of alignment interaction with spatial distance is measured by the Interaction kernel.

Kinetic compartment model: The spread of infection population in commuters is modeled by individuals' position $x \in \Omega \subset \mathbb{R}^2$ and moving with velocity direction $v \in \mathbb{S}^1$. The kinetic density of susceptible, infected, and recovered compartments is defined by $f_S(x, v, t)$, $f_I(x, v, t)$, $f_R(x, v, t)$, respectively. The kinetic distribution of commuters can be calculated using the following expression

$$f(x, v, t) = f_S(x, v, t) + f_I(x, v, t) + f_R(x, v, t),$$

with total density

$$\rho(x, t) = \int_{\mathbb{S}^1} f(x, v, t) dv.$$

The total density of persons, independent of their mobility direction, is thus provided by this equation. Consequently, the compartment size is given as

$$\mathcal{S}(x, t) = \int_{\mathbb{S}^1} f_S(x, v, t) dv, \quad \mathcal{I}(x, t) = \int_{\mathbb{S}^1} f_I(x, v, t) dv, \quad \mathcal{R}(x, t) = \int_{\mathbb{S}^1} f_R(x, v, t) dv,$$

with

$$\rho(x, t) = \mathcal{S}(x, t) + \mathcal{I}(x, t) + \mathcal{R}(x, t),$$

define the density fraction at time $t > 0$ with position x of susceptible, infected and recovered population commuters. The evaluation of kinetic density over time is given by the following transport equation dynamics

$$\begin{aligned}
\partial_t f_S + v_S \cdot \nabla_x f_S &= -f_I(f_S, \mathcal{I}_T) + \frac{1}{T_S}(\mathcal{S} - f_S), \\
\partial_t f_I + v_I \cdot \nabla_x f_I &= f_I(f_S, \mathcal{I}_T) - \gamma f_I + \frac{1}{T_I}(\mathcal{I} - f_I), \\
\partial_t f_R + v_R \cdot \nabla_x f_R &= \gamma f_I + \frac{1}{T_R}(\mathcal{R} - f_R).
\end{aligned}$$

More detail can be seen in Ref [61].

1.3 Role of network theory

Generally, well-mixed settings do not support disease transmission, information dispersion, or collective activity, but rather in structured interactions within community meetings, species migrations, and data transferring through digital platforms. Network theory provides a basic framework to model the varied contact structure in complex systems as a graph $\mathcal{G} = (\mathcal{V}, \mathcal{E})$ by describing individuals or objects as nodes $\mathcal{V} = \{1, 2, \dots, \mathcal{N}\}$ and their paired interactions as edges $\mathcal{E} \subseteq \mathcal{V} \times \mathcal{V}$, a subfield of graph theory in computing, mathematics, and network research. Comprehensively exploring the contact heterogeneity and individual-based disease prevention strategies, network models accurately capture the individual influence, enabling the study of local features like clustering, consensus formation, super spreads, and global consequences for disease dynamics. The empirical investigation of network-specific optimization problems is combinatorial optimization, which focuses on determining the most effective means of accomplishing certain goals within a network structure. Network flow, the shortest path issue, resource allocation, and facility placement, opinion polarization, social connectivity, and pandemic outbreak are concrete examples of networks' significance, as shown in Fig 1.4. The disease transmission and opinion formation are the main focus of the thesis, where we utilize the network models.



FIGURE 1.4: Display of various networks.

1.3.1 Network models

We describe some main network models used to simulate social behaviors, opinion propagation, and disease spread. Considering the set of vertices $\mathcal{V} = [n] = \{1, 2, 3, \dots, n\}$. A random graph $\mathcal{G}$ with n vertices is defined by set of edges $\mathcal{E} \subseteq \binom{[n]}{2}$ with $\binom{[n]}{2} = \{\{i, j\} : 1 \leq i < j \leq n\}$, is the set of all potential pairs of different vertices that are not ordered. Uniform $\mathcal{G}_{n,m}$ and Binomial models are the two main

¹https://en.wikipedia.org/wiki/Network_theory

classical models commonly used, which we discuss. Assume

$$\mathcal{G}_{n,m} = \{\mathcal{G} = (\mathcal{V}, \mathcal{E}) : |\mathcal{V}| = n, |\mathcal{E}| = m\},$$

by fixing $n \geq 1$, $0 \leq m \leq \binom{[n]}{2}$, represent the family of all label graphs precisely m edges and a vertex set $[n]$. Within the uniform model, every graph in $\mathcal{G}_{n,m}$ is selected with an equal opportunity

$$\mathcal{P}(\mathcal{G}) = \left(\binom{[n]}{2}\right)^{-1}, \quad \mathcal{G} \in \mathcal{G}_{n,m},$$

which equates to roughly selecting m edges at random from the $\binom{[n]}{2}$ potential edges. Such random graphs denoted by $\mathcal{G}_{n,m} = ([n], \mathcal{E}_{n,m})$ called *uniform random graph model*. Now we define another model by taking $0 \leq p \leq 1$. For $0 \leq m \leq \binom{[n]}{2}$, provide a probability to any graph $\mathcal{G}$ with set of vertices $[n]$ and m edges

$$\mathcal{P}(\mathcal{G}) = p^m (1-p)^{\binom{[n]}{2}-m}, \quad 0 \leq p \leq 1.$$

Alternatively, we begin with an empty graph that has a vertex set of $[n]$, and we carry out $\binom{[n]}{2}$ Bernoulli trials in which we separately insert edges with likelihood p . This kind of random graph is known as a binomial random graph or *Erdős–Rényi–Gilbert graph* [109, 126], and its symbol is $\mathcal{G}_{n,p} = ([n], \mathcal{E}_{n,p})$.

A *scale-free network* is a kind of complex network where the degree distribution monotonically approaches a power-law [77]. According to network science, the degree of a node is determined by the number of edges with other nodes. By formal definition, If $\mathcal{K}$ represents degree, then $\mathcal{P}(\mathcal{K})$ is the likelihood that a randomly selected node has degree $\mathcal{K}$

$$\mathcal{P}(\mathcal{K}) \sim \mathcal{K}^{-\gamma}, \quad \text{for large } \mathcal{K},$$

with $2 < \gamma < 3$, define the power law expectation, then the network is called scale-free. The large tail distributions of these networks make them identical compared to other networks with Poisson or exponential degree distributions. There is a constant likelihood of discovering nodes with extremely high degrees, irrespective of scale, because these networks provide a polynomial decrease in the probability of coming across high-degree nodes or hubs. As a result, certain hubs have a substantially greater number of links than the majority of nodes, demonstrating the networks' scalability. A graph with a large clustering ratio and short distances is called a *small world network* [295]. In particular, a small-world network encoded where the global clustering value is not small, but the average distance $\mathcal{L}$ between two randomly selected nodes

$$\mathcal{L} \propto \log \mathcal{N}$$

increases proportionately to the logarithm of the number of nodes $\mathcal{N}$ in the network.

1.3.2 Mean field limit of large networks

Suppose the undirected graph $\mathcal{G}_{\mathcal{N}} = (\mathcal{V}_{\mathcal{N}}, \mathcal{E}_{\mathcal{N}})$, which has $\mathcal{N}$ nodes and an adjacency matrix $\mathcal{A} = (\mathcal{A}_{ij})$. Node i recovers at a rate of $\gamma > 0$ in a continuous-time susceptible–infected–susceptible (SIS) model, and it acquires infection through every infected adjacent node j at a rate of $\beta > 0$. $\mathcal{X}_i(t) \in \{0, 1\}$ represents the infection state of node i , where $p_i(t) = \mathbb{P}[\mathcal{X}_i(t) = 1]$. A quenched mean-field approximation

enables system closure, resulting in the differential equation

$$\frac{dp_i}{dt} = -\gamma p_i + \beta(1 - p_i) \sum_{j=1}^{\mathcal{N}} \mathcal{A}_{ij} p_j, \quad \text{for } i = 1, \dots, \mathcal{N}.$$

Let $\lambda_{\max}(\mathcal{A})$ be the largest eigenvalue of $\mathcal{A}$. The epidemic threshold is written as

$$\mathbf{R}_0^{\mathcal{N}} = \frac{\beta}{\gamma} \lambda_{\max}(\mathcal{A}).$$

Examining epidemic dynamics on networks is highly challenging due to the extensive nature and diversity of actual networks, which may consist of millions of nodes and edges. Initial investigation of these networks is frequently unfeasible due to numerical intractability and topological complexity. To bridge discrete networks with continuum PDEs or kinetic models, a precise theoretical limiting object is required as $\mathcal{N} \rightarrow \infty$. This evolution maintains the heterogeneity structure while facilitating the transition from node-level dynamics to a configurable limit equation. *Graphon* as a measurable kernel provides a comprehensive solution, serving as the limit of a sequence of dense graphs. Detailed analysis has been formulated in the thesis.

1.4 Data augmentation with neural networks

The study of complex dynamics faces significant challenges due to the nature of real-world data sets that are too scarce, incomplete, and noisy. More practically, the disease transmission rate is usually sparse through temporal and geographic dimensions in epidemiology, and frequently noisy or unreliable individual interaction data in complex networks. Such issues make it tricky to comprehensively understand effective modeling and make it challenging to derive precise inferences or predictions from actual data. All these facts motivate researchers to employ *data augmentation* strategies, generally defined as the generation of new and informative data from a limited observed dataset, to enhance the prediction and forecasting of the dynamics. A pathway of these techniques is represented in Fig 1.5. Considering neural networks exhibit universal approximation features, they are essential to data augmentation in scientific domains. Large-dimensional distributions, temporal interactions, and nonlinear transitions can be accurately approximated using neural networks. They might help with missing data estimation, make it easier to create synthetic data by surrogate modeling, and allow hybrid modeling, guided by physical constraints.

In the context of missing data, assuming a data set $\mathcal{D} = (t_i, y_i)_{i=1}^n$ with observation $y_i \in \mathbb{R}$ over time t_i . Traditional imputation methods, such as expectation-maximization and linear interpolation, depend on homogeneity and basic statistical models that may not be sufficient for stochastic or nonlinear dynamics. Neural networks provide a solution in such instances by using observed values to approximate the conditional distribution of missing samples. Taking Recurrent Neural Network (RNN) capable of capturing temporal dependencies through the learning of nonlinear mappings with function f_ϕ learned from data $\hat{\mathcal{Y}}_t = f_\phi(\mathcal{Y}_{t-1}, \mathcal{Y}_{t-2}, \dots)$, while producing models Variational Autoencoders [166], utilize a latent variable $\mathcal{Z}$ drawn from a distribution $\mathcal{Z} \sim \mathcal{P}(\mathcal{Z})$ and focus on approximation

$$\mathcal{P}_\phi(\mathcal{Y}|\mathcal{Z}) \approx \prod_i \mathcal{P}_\phi(\mathcal{Y}_i|\mathcal{Z}),$$

thereby allowing probabilistic regeneration of missing data.

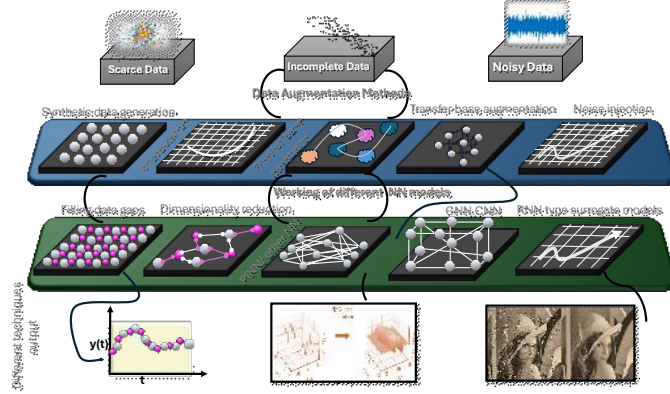


FIGURE 1.5: A schematic diagram of data augmentation with neural network surrogate models.

Surrogate models with neural networks are an effective way to make computation more reliable. Supposing a simulator $\mathcal{Y} = \mathcal{G}(x)$ with x input parameter like disease transmission. Assessing $\mathcal{G}$ frequently involves executing PDEs/ODEs models, both of which are expensive, while f_ϕ as a surrogate with a neural network is trained to estimate $f_\phi \approx \mathcal{G}$, with loss.

$$\mathcal{L}(\phi) = \frac{1}{n} \sum_{i=1}^n |f_\phi(x_i) - \mathcal{G}(x_i)|^2.$$

After training, f_ϕ generates outputs rapidly, allowing for efficient parameter evaluation, data augmentation, and uncertainty quantification [242]. Entirely data-driven augmentation carries the risk of producing synthetic information that is reliable statistically but impractical in a physical sense. This complexity is solved by hybrid models, which use governing equations to integrate data and existing knowledge. Feed Forward Neural Network (FFN), Recurrent Neural Network (RNN), Graph Neural Network (GNN), particularly Physics Informed Neural Network (PINN), well know examples which hybridized in most studies. In a hybrid sense, PINN includes the data fitting and physical constraints in the loss function.

$$\mathcal{L}(\phi) = \mathcal{L}_{\text{data}}(\phi) + \omega \mathcal{L}_{\text{physics}}(\phi).$$

More details can be seen in the next chapters.

Part I

Compartment Modeling of Disease Dynamics

Chapter 2

Smoking dynamics in the context of nicotine gum and media campaigns

Smoking has become a significant global health crisis with profound socio-economic effects. This study presents innovative deterministic models for analyzing smoking dynamics, dividing the population into five sub-groups. It examines treatment for smokers, the influence of media coverage, and the combined impact of advertising across TV, radio, and social media. The models establish disease-free and endemic equilibrium states with stability analyses, indicating stability conditions based on the reproductive number, $\mathcal{R}_0$. Sensitivity analyses and numerical simulations support the findings, suggesting that effective media coverage and medical treatment can reduce smoking rates. Awareness campaigns and targeted advertising are proposed as strategies to enhance public knowledge and facilitate smoking cessation. This chapter is based on the study [13].

2.1 Introduction

Smoking kills almost 7 million people annually, making it one of the global largest ailments. Over 6 million of those are directly smoking, and 0.9 million are non-smokers who are impacted by smokers [297]. The 1.1 billion smokers worldwide are mostly found in nations with middle and low incomes, where smoking has the greatest negative effects on both health and the economy. Smokers who pass away too soon not only lose money from their households but also incur more medical costs and impede economic growth. Smoking is thought to cause damage to the worldwide economy of about 2 trillion dollars a year, or about 2% of the global GDP. About 30% of these losses are connected to medical expenses for addressing disorders brought on by tobacco use, while the majority are attributable to decreased job performance as a result of smoking related diseases (SRD) or fatalities [102]. Smoking is the act of lighting a substance on fire or heating it, then inhaling the smoke through the nose or mouth and into the lungs. The active ingredients in smoking enter the circulation through the lungs and make their way to the brain, where they take action. Tobacco is a highly prevalent smoking drug, used by over 1.3 billion people worldwide [157]. Most invisible diseases, such as cancer, diabetes, cardiovascular issues, and chronic issues, are significantly exacerbated by smoking [62, 27, 165]. Additionally, smoking causes several varieties of cancers, such as those of the mouth, throat, stomach, cervix, breast, and pancreas. Smoking contributes to these

potentially fatal diseases because cigarettes have over 4000 different chemicals and hazardous materials [309].

Mathematical Modeling plays a vital role in understanding any disease dynamics, including smoking, and making critical prevention measures (PM) to control the spread of smoking by their simulation outcomes [79, 15, 112, 209]. The literature on SIR (susceptible, infected, recovered) modeling is a waste. SIR types of other models like SEIR, SEIRQ, etc, play an important role in studying the dynamics of any disease. In epidemic models, the incidence rate that guarantees the establishment of new infection numbers is a significant factor. Different authors use a variety of these, like bilinear, saturated, exponential, nonlinear, fractional, etc, according to their modeling perspective [251, 306, 143, 232]. Many researchers formulate different smoking models with a variety of incidence rates. Castillo-Garsow et al proposed a PSQ (Potential, Recovered, and Quitter smokers) smoking model with linear/nonlinear incident rate [72]. O Sharomi et al formulate the model of smoking in which they split the ex-smoker into temporary and permanent quitters [256]. Later on, Zaman et al extend the smoking dynamics by adding the occasional smokers as a compartment in the model [305]. Z. Alkhudhari et al considered these studies and checked the pre pressure on the temporary quitters in smoking dynamics [19]. After that, Alkhudhari et al also studied these dynamics by dividing the smokers into occasional and heavy smokers and later discussed the impact of occasional and potential smokers on heavy smokers [20]. Ihsanjaya et al discuss the mathematical model of changing smoking behaviors with temporary and permanent quitter compartments [149]. Abdullah et al also elaborate on this model with VGTOW compartments represented as potential, occasional, Smokers, temporarily quitting, and permanently quitting smokers [1]. Later on, Muhafzan et al discuss the PSR smoking model with treatment [39]. Many other authors discuss the smoking dynamics in references [139, 240, 147, 73, 266, 165, 262, 310, 17, 81, 249].

Media awareness and treatment are significant elements of quitting smoking dynamics. Behavioral therapy and Nicotine replacement therapy are two effective treatments that assist in treating the mental and clinical aspects of smoking addiction. In addition, media advertisements promote healthier lifestyles, provide tools for quitting, and increase public understanding of the risks associated with smoking, all of which have the combined effect of lowering smoking rates and assisting people in their attempts to stop. When combined, they provide a comforting atmosphere that raises the possibility of an effective smoking quit. The media plays a key role in educating the public about the dangers of smoking and encouraging quitting. It has the power to influence attitudes and behaviors around tobacco usage by providing tailored messages. Extensive initiatives, including taxes, public smoking prohibitions, safety precautions, and mass media campaigns, have been put into place to successfully lower smoking rates. Even while these programs can inspire some people to give up, smokers who are experiencing stress, worry, or depression may nevertheless succumb to the urge, frequently in an attempt to find momentary solace from their problems. Persistent achievement in quitting smoking needs to address these core concerns [266]. The discussion indicates that by advertising the daily number of infected individuals, making awareness programs through advertisements and slogans on social media apps like YouTube, WhatsApp, Twitter (X), and Facebook, etc, and mainstream media like TV and Radio programs, we can make a significant impact on controlling the smoking dynamics. Also, by implementing clinical treatments for smoking-affected people, we can decrease the mortality rate due to SRD. Numerous studies have been done on epidemics by use of media awareness programs. The risk of disease depends on people's perceptions,

which are influenced by public opinion and the media. Regular smoking advertisements can influence smokers' habits and encourage them to quit. The media plays an important role by providing useful information on the harms of smoking and can influence individuals' thoughts, attitudes, and habits by delivering tobacco cessation messages directly to them. Mass media campaigns combined with strategies at the state and community level can be effective in reducing tobacco use, especially among diverse populations. Awareness campaigns based on media and behavioral interventions are critical during an epidemic and can have a long-term impact. [266, 311, 302, 241, 169, 130]

Designing a mathematical model that considers media influence and treatment might yield significant insights into ways to best implement these strategies and minimize smoking rates. Motivated by the previous literature and considering the complications of smoking dynamics for global health and the death rate due to smoking-related diseases, we formulate multiple mathematical models to gain deep insight into these complex dynamics. In these models, we utilize the impact of daily news on the number of affected smokers considered as fear factors in the community, media advertisement through social apps, TV, and Radio for awareness programs, and implement the different treatment therapies to overcome the smoking-related issues. We also deeply study the combined impact of media awareness and treatment on smokers. To the best of our knowledge, no one has done work on this site. Firstly, we develop a mathematical model to control smoking dynamics with treatment. In this model, we considered potential smokers, active smokers/Smokers, recovered smokers, quitter smokers, and death smokers compartments. We evaluate how smoking addiction affects the population badly and increases the mortality rate due to its harmful nature. Then we believe in the effectiveness of treatment factors to control the increasing mortality rate and addiction to smoking. Secondly, we formulate a mathematical model to advertise daily news of addicted smokers through media and treatment to control smoking dynamics. In this model, we take the same compartments and want to control the transmission of smoking from smokers to potential smokers through effective contact by the use of a bilinear incident rate, in which we take the media awareness parameter, which shows the weight of advertising the daily news of the number of new smokers, which increases when coming in contact with active smokers. So by the simulation of this function, we can analyze the impact of media along with the treatment parameter to control the transmission of smoking and decrease the deaths that come due to SRD. Thirdly, we proposed a mathematical model of the combined impact of TV, Radio, Social media advertisement, and treatment to control smoking dynamics. In this case, we take a saturated incidence rate which is dependent on the media parameters including TV, Radio, and Social media apps like YouTube, WhatsApp, Twitter (X), Facebook, etc, and we try to overcome the smoking habits of the people by increasing the media awareness programs through live streaming, banners on big spots in the country, slogans like "Smoking cause Cancer", "Smoking kill you every second" etc and treating the affected smokers by behavioral or clinical therapies. In this way, we can control the smoking addiction and the mortality rate due to smoking.

Here we present the structure of the chapter as: Sec 2.2 is about the formulation of novel modeling to control smoking dynamics with treatment and a detailed description. Sec 2.3 describes the mathematical analysis of the model to control smoking dynamics with treatment, including feasible region, positivity, and boundedness, the existence of a unique solution, and the calculation of steady states along the threshold parameter. We define the local and global stability analysis of the smoking-free and smoking steady states, and the Sensitivity analysis of the Model to control

smoking dynamics with treatment. We explain the treatment-free smoking model's numerical description. Numerical simulation of the model to control smoking dynamics with treatment to check the impact of treatment on smoking and mortality rate due to smoking deaths is also included. In Sec 2.4 we proposed a mathematical model to advertise daily news of addicted smokers through media and treatment to control smoking dynamics, including the calculation of DFE and EEP, the threshold parameter of the model. We define Sensitivity analysis of the model threshold parameters and numerical simulation of the model to advertise daily news of addicted smokers through media and treatment linked with the effectiveness of daily media news and treatment on smoking and mortality rate due to smoking-related diseases. In Sec 2.5 we propose a mathematical model of the combined impact of TV, Radio, and Social media advertisement and treatment to control smoking dynamics. It also includes the computation of disease-free and endemic steady states and threshold parameters. It shows sensitivity analysis of threshold parameters with PRCC and describes the computation of numerical results of the proposed model and the impact of TV, Radio, and Social media advertisement with treatment on smokers and mortality rate due to smoking deaths. Sec 2.6 is about the comparison of models and future implementation. Sec 2.7 predicts the conclusion of this novel work.

2.2 Mathematical model to control smoking dynamics with treatment

Smoking is the most dangerous addiction in the world, with its extensive effects on mental and physical health. Smokers have a 10% greater probability of lung cancer and a 70% greater chance of heart attacks than non-smokers. Approximately 200 million women and 900 million men smoke worldwide, and smoking-related mortality happens every six seconds. A single pack of cigarettes contains 5–10 mg of nicotine, or around 1-2 mg of nicotine per cigarette for a smoker. Smoking hurts female fertility and hurts the smoker as well as those around them. It causes 12–13 years of lifespan reduction and contributes to 22% of yearly fatalities [149, 269]. Here we discuss some main treatments to overcome the smoking dynamics.

- **Clinical Therapy**

The ingredient in tobacco that makes people addicted is called nicotine. Nicotine in cigarettes is far more addictive than the nicotine in products that help people quit smoking, such as gum or patches. This is because smoking rapidly supplies the brain with nicotine, making it more pleasurable and difficult to stop. [282, 283]. By providing nicotine without the dangerous compounds contained in cigarettes, nicotine replacement therapy (NRT) aids in the cessation of smoking. [284].

- **Cognitive and Behavioral Therapy**

Novel approaches to treating nicotine addiction are the result of research in the behavioral and social sciences. Emotions such as worry, cravings, poor motivation, and self-doubt about quitting might persist long after the effects of nicotine withdrawal have subsided. [187, 285]. Behavioral therapy, cognitive behavioral therapy (CBT), motivational speaking, acceptance and commitment therapy (ACT), and incentive-based therapies are effective approaches for treating tobacco addiction and reliance on nicotine. [284, 271, 220].

Previous studies suggest that the prevention of smoking addiction and overcoming deaths due to smoking-related diseases need more collaboration to find more accurate treatment methods. To consider these points over motive is to formulate a mathematical model of smoking dynamics in which we deal with the treatment of smokers and accurately investigate the mortality rate due to smoking-related disease. In this work, we proposed a model composed of five nonlinear differential equations that address the smoking outbreak. For better understanding, we divide the population $\mathcal{N}(t)$ into five sub classes: Potential smokers $P(t)$, Active Smokers/Smokers $S(t)$, Recovered Smokers $R(t)$, Quitters Smokers $Q(t)$, Death Smokers $D(t)$. Suppose $\mathcal{N}(t)$ represents the whole population at time t and is given as

$$\mathcal{N} = P + S + R + Q + D$$

The compartmental diagram of the smoking dynamics is given in Fig 2.1. The recruitment of potential smokers into the population occurs at a rate Λ , β_1 is effective transmission rate between smokers and potential smokers, the recovery rate of the active smokers is represented by α , μ is natural death rate, the awareness rate of potential smokers is ϕ , β_2 is the rate at which potential smokers moves to quitter class, ϵ is consistent rate of smokers, rate at which smokers died due to smoking is represented by γ , π is the proportion rate at which recovered becomes potential smokers, nicotine gum parameter denoted by ρ and θ is transmission rate from recovered to potential smokers. Table 1 shows the parameter values used in the system (5.1). To describe these dynamics in the best way, we discuss each class independently.

- **Potential Smokers Class $P(t)$**

Potential Smokers $P(t)$ are those who have not yet evolved into habitual smokers but are in danger of executing this. This class may include people who can or start smoking due to the pressure of family, friends, and society, and also include those people who smoke occasionally. Potential Smokers decrease when Smoker $S(t)$ interacts with P at a rate of $\beta_1 PS$. There exist relapse phenomena at a recovery rate α of Smoker S with a determination to become consistent at rate ϵ of not smoking or less smoking and goes to the Recovered class $R(t)$. This happens with probabilities 0 and 1. If $\epsilon = 1$, the individuals become recovered, and if $\epsilon = 0$, they again become potential smokers, due to which this class increased. Also, this class increases with a rate $\pi\theta R$ at which the quitters again become potential smokers. Many individuals are well aware of the bad effects of smoking, and also others directly move to quitters at a rate of ϕ and β_2 respectively. The Natural mortality rate also causes decay in potential smokers.

$$\frac{dP}{dt} = \Lambda - \beta_1 PS + \alpha(1 - \epsilon)S - (\mu + \phi + \beta_2)P + \pi\theta R$$

- **Active Smokers/ Smokers Class $S(t)$**

Active Smokers/ Smokers Class $S(t)$ are those smokers who are addicted to smoking and now continuously actively smoke. This class increases with the higher push up of transmission rate of smoking $\beta_1 PS$ when Smokers S have higher contact with the P . Due to the self-discipline or other types of strategies, active smokers S move to the recovered class at a rate of α along with the natural mortality, which causes decay in $S(t)$. The main point here is the treatment parameter ρ and the deaths due to smoking-related disease γ , which shows our strong motive to control these dynamics. So we introduce the treatment

parameter ρ to control the addiction level of smoking among smokers. The smokers move to Recovered Smokers R due to the proper treatment, which we explained in detail earlier. Also, those smokers who are in serious conditions or are more badly affected by smoking addiction and suffer from SRD like lung cancer, heart disease, mental issues, etc, die and move to the Death class D .

$$\frac{dS}{dt} = \beta_1 PS - (\mu + \alpha + \rho + \gamma)S$$

- **Recovered Smokers Class $R(t)$**

Recovered Smokers Class $R(t)$ are those people who overcome the smoking addiction after successful treatment at a rate of ρS . Also, the smoking individuals who are consistent in quitting smoking recovered with a recovery rate $\alpha \epsilon S$, due to which the recovered class increased. The recovered individuals who permanently quit smoking move to quitter Smokers Q at a rate of θ along with the natural mortality rate μ .

$$\frac{dR}{dt} = \alpha \epsilon S + \rho S - (\mu + \theta)R$$

- **Quitter Smokers Class $Q(t)$**

Quitter Smokers Class $Q(t)$ consists of those individuals who permanently quit smoking after recovering from the smoking addiction at a rate of $(1 - \pi)\theta R$, which indicates the relapse scenario. The recovered smokers who become quitters may be involved in smoking due to the pressure of family, friends, environment, etc, and move to Potential Smokers P at a quitting rate of θ with the probability of π . If the probability of $\pi = 0$, then quitters again become potential smokers, and if $\pi = 1$, then they did not move to P . Also, the natural mortality rate μ causes decay in the Quitter class.

$$\frac{dQ}{dt} = \beta_2 P + \phi P + (1 - \pi)\theta R - \mu Q$$

- **Death Smokers Class $D(t)$**

Death Smokers Class $D(t)$ are those who died due to smoking-related disease. Here γ is the main parameter that indicates the death rate in smoking dynamics, who are badly affected by smoking addiction and can not be treated successfully with some clinical therapies.

$$\frac{dD}{dt} = \gamma S - \mu D$$

The complete Smoking model is written below:

$$\left\{ \begin{array}{l} \frac{dP}{dt} = \Lambda - \beta_1 PS + \alpha(1 - \epsilon)S - (\mu + \phi + \beta_2)P + \pi\theta R \\ \frac{dS}{dt} = \beta_1 PS - (\mu + \alpha + \rho + \gamma)S \\ \frac{dR}{dt} = \alpha \epsilon S + \rho S - (\mu + \theta)R \\ \frac{dQ}{dt} = \beta_2 P + \phi P + (1 - \pi)\theta R - \mu Q \\ \frac{dD}{dt} = \gamma S - \mu D \end{array} \right. \quad (2.1)$$

with the initial conditions

$$P(0) \geq 0, S(0) \geq 0, R(0) \geq 0, Q(0) \geq 0, D(0) \geq 0$$

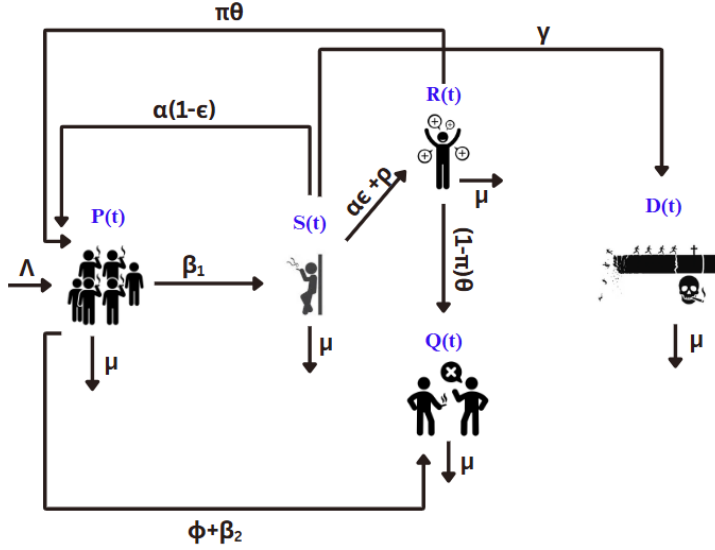


FIGURE 2.1: Schematic diagram of smoking dynamics in model (5.1).

Since actual data were unavailable, we used values from the literature to estimate the parameters $(\beta_2, \theta, \pi, \beta_3, \eta_1, \eta_2)$. After carefully examining several sources, we chose average values that best reflected the behavior of the model. This approach guarantees that the model maintains its theoretical soundness and yields significant outcomes.

2.3 Mathematical analysis of model

In this section, we define some general results from a theoretical perspective that give us the guarantee of validation of the model.

2.3.1 Positively invariant region

We investigate model (5.1) in the closed set based on biological considerations.

$$\Delta = \left\{ P(t), S(t), R(t), Q(t), D(t) \in \mathbb{R}_+^5 \mid 0 < P(t), S(t), R(t), Q(t), D(t) \leq \frac{\Lambda}{\mu} \right\}$$

is positively invariant inside the model's context [213].

Theorem 2.1. *Model (5.1) exhibits positively invariant Δ .*

Proof. Suppose

$$N' = P' + S' + R' + Q' + D'$$

So, the model (5.1) has the form

$$\frac{dN}{dt} \leq \Lambda - \mu N \quad (2.2)$$

TABLE 2.1: Values of parameters utilized in the developed model (5.1), (2.33), and (2.37).

Symbol	Description of Parameters	Values	Source
Λ	Recruitment rate	0.2	[39]
β_1	Effective transmission rate between smokers and potential smokers	0.9	[39, 288]
β_2	Spontaneous or Intrinsic quitting rate of potential smokers	0.03 [0.01 0.03]	Estimate
ϕ	Awareness rate of potential smokers	0.1 [0.05 0.1]	[39]
μ	Natural death rate	0.2	[288]
ϵ	Consistent rate of smokers to quit the addiction of smoking	0.02	[39]
α	Recovery rate of the active smokers	0.1	[39]
γ	Rate at which smokers died due to smoking	0.015	[266]
ρ	Nicotine gum parameter	0.1 [0 0.17]	[39]
θ	Transmission rate from recovered to quitter smokers	0.05	Estimate
π	Proportion rate at which quitters become potential smokers	0.01	Estimate
β_2	Spontaneous or Intrinsic quitting rate of potential smokers of model (2.33)	0.01 [0.01 0.03]	Estimate
ϕ	Awareness rate of potential smokers of model (2.33)	0.05 [0.05 0.1]	[39]
β_3	Effective contact of Potential smokers with Smokers (S)	0.75	Estimate
m	Weighted to share information about smokers	0.5 [0 2]	[130]
η_1	Relative infectivity potential of Recovered Smokers	0.4	Estimate
η_2	Relative infectivity potential of Quitter Smokers	0.2	Estimate
M	Efficiency rate of information shared through S(Social apps) , T(TV) and R(Radio)	50% [0 1]	[169]
β	Baseline transmission rate	0.55	[169]

This implies that $\frac{dN}{dt} < 0$ when $N > \frac{\Lambda}{\mu}$. Thus, $\frac{dN}{dt}$ is bounded by $\Lambda - \mu$. By applying the integrating factor approach to the inequality (5.2), we obtain

$$\frac{d}{dt} (e^{\mu t} N) \leq e^{\mu t} \Lambda \quad (2.3)$$

By using ICs to integrate Equation (5.3), we obtain

$$N(t) \leq N(0)e^{-\mu t} + \frac{\Lambda}{\mu} (1 - e^{-\mu t})$$

As $t \rightarrow \infty$, we get

$$N \leq \frac{\Lambda}{\mu}$$

Consequently, it means that Δ is positively invariant of the model (5.1), i.e., no solution path crosses any of its limits. $\square$

2.3.2 Positivity of solutions

Here, we calculate the solution's positivity. Verifying that every variable in a model (5.1) is non-negative for every t is crucial. To show that the model's solution is positive, we looked at the following theorem [136, 12].

Theorem 2.2. *The model (5.1) yields a positive solution for all $t > 0$, based on the ICs described in Eq (5.1).*

Proof. Using the 1st Eq of system (5.1), assume that

$$\begin{aligned} \frac{dP}{dt} &= \Lambda - \beta_1 PS + \alpha(1 - \epsilon)S - (\mu + \phi + \beta_2)P + \pi\theta R \leq \Lambda - (\mu + \phi + \beta_2)P \\ \frac{dP}{dt} + (\mu + \phi + \beta_2)P &\leq \Lambda \end{aligned}$$

Multiplying by I.F. and after integrating, we obtain

$$Pe^{(\mu+\phi+\beta_2)t} \leq \frac{\Lambda e^{(\mu+\phi+\beta_2)t}}{(\mu + \phi + \beta_2)} + c \quad (2.4)$$

When $t = 0$, we have

$$P(0) \leq \frac{\Lambda}{(\mu + \phi + \beta_2)} + c$$

Using Eq (5.16), as $t \rightarrow \infty$, we get

$$\frac{\Lambda}{(\mu + \phi + \beta_2)} \geq P(t) \geq 0$$

Using the same technique, we can demonstrate that

$$S(0) \geq 0, R(0) \geq 0, Q(0) \geq 0, D(0) \geq 0$$

This demonstrates the positive nature of the model (5.1) solution. $\square$

2.3.3 Boundedness of solutions

In this section, the boundedness of the solution is discussed. The solutions that are obtained remain inside the feasible region denoted by Δ [224, 168].

Theorem 2.3. *Every solution of the model (5.1) is bounded.*

Proof. The total population, represented by N , is described as

$$N' = P' + S' + R' + Q' + D'$$

By adding all equations of model (5.1), we get

$$\frac{dN}{dt} = \Lambda - \mu N$$

Applying Gronwall's inequality, we obtain

$$N \leq \frac{\Lambda}{\mu} + \left(N(0) - \frac{\Lambda}{\mu} \right) e^{-\mu t}$$

Hence, $N \leq \frac{\Lambda}{\mu}$, for all $t \geq 0$, whenever $N(0) \leq \frac{\Lambda}{\mu}$.

$$N \liminf_{t \rightarrow \infty} N \leq \limsup_{t \rightarrow \infty} N \leq \frac{\Lambda}{\mu} > 0.$$

This demonstrates that the feasible area Δ contains a bound on the solution of model (5.1), which shows that the affirmative solution to model (5.1) is contained in the feasible region Δ . $\square$

2.3.4 Existence and Uniqueness of solution

The general first-order ODE takes the following form:

$$\frac{du}{dt} = g(u, t) \quad s(t_0) = u_0 \quad (2.5)$$

To determine the existence and uniqueness of a solution to Eq. (2.5), we analyze the conditions under which a solution exists and the criteria that guarantee a unique solution. Suppose

$$\begin{cases} g_1 = \Lambda - \beta_1 PS + \alpha(1 - \epsilon)S - (\mu + \phi + \beta_2)P + \pi\theta R \\ g_2 = \beta_1 PS - (\mu + \alpha + \rho + \gamma)S \\ g_3 = \alpha\epsilon S + \rho S - (\mu + \theta)R \\ g_4 = \beta_2 P + \phi P + (1 - \pi)\theta R - \mu Q \\ g_5 = \gamma S - \mu D \end{cases}$$

The following theorem demonstrates that our model's solution exists and is unique [168].

Theorem 2.4. (Uniqueness of Solution)

Let Ξ stand for the domain

$$\Xi = \begin{cases} |t - t_0| \leq a, \\ |s - s_0| \leq b, \\ u = (u_1, u_2, u_3, \dots, u_n), \\ u_0 = (u_{10}, u_{20}, u_{30}, \dots, u_{n0}) \end{cases} \quad (2.6)$$

Assume that $g(t, u)$ satisfies the Lipschitz requirement.

$$\|g(t, u_1) - g(t, u_2)\| \leq L\|u_1 - u_2\|, \quad (t, u_1), (t, u_2) \in \Xi \quad (2.7)$$

Then there exists a $\delta > 0$ such that there exists a unique continuous vector solution $u(t)$ of the system (2.5) in the interval $|t - t_0| \leq \delta$. Keep in mind that Eq (2.7) necessitates the following:

$$\left\{ \frac{\partial g_i}{\partial u_j}; \quad i, j = 1, 2, 3, \dots, n \right. \quad (2.8)$$

be continuous and bounded in the domain Ξ .

Lemma 2.5. If it $g(t, u)$ has a continuous partial derivative $\frac{\partial g_i}{\partial u_j}$ on a bounded close convex domain $\mathbf{R}$ where it $\mathbf{R}$ is used to represent real numbers, then it satisfies a Lipschitz condition in $\mathbf{R}$.

Our interest is in the domain $1 < \epsilon < \mathbf{R}$. So, we look for a bounded solution of the form $0 < \mathbf{R} < \infty$. We now prove the following existence theorem.

Theorem 2.6. (Existence of Solution)

Given that $1 \leq \epsilon \leq \mathbf{R}$ holds, assume to Ξ represent the domain as specified in (3.2) s.t (2.7). A solution to (5.1) that is bound in the domain Ξ thus exists.

Proof. Suppose

$$g_1 = \Lambda - \beta_1 PS + \alpha(1 - \epsilon)S - (\mu + \phi + \beta_2)P + \pi\theta R \quad (2.9)$$

$$g_2 = \beta_1 PS - (\mu + \alpha + \rho + \gamma)S \quad (2.10)$$

$$g_3 = \alpha\epsilon S + \rho S - (\mu + \theta)R \quad (2.11)$$

$$g_4 = \beta_2 P + \phi P + (1 - \pi)\theta R - \mu Q \quad (2.12)$$

and

$$g_5 = \gamma S - \mu D \quad (2.13)$$

we demonstrate the continuous and limited nature of $\frac{\partial g_i}{\partial u_j}$, where $i, j = 1, 2, 3, 4, 5$. We looked at the following partial derivatives for every model equation:

From Eq (3.4):

$$\left\{ \begin{array}{l} \frac{\partial g_1}{\partial P} = -\beta_1 S - (\mu + \phi + \beta_2), \left| \frac{\partial g_1}{\partial P} \right| = |-\beta_1 S - (\mu + \phi + \beta_2)| < \infty \\ \frac{\partial g_1}{\partial S} = -\beta_1 P + \alpha(1 - \epsilon), \left| \frac{\partial g_1}{\partial S} \right| = |-\beta_1 P + \alpha(1 - \epsilon)| < \infty \\ \frac{\partial g_1}{\partial R} = -\pi\theta, \left| \frac{\partial g_1}{\partial R} \right| = |-\pi\theta| < \infty \\ \frac{\partial g_1}{\partial Q} = 0, \left| \frac{\partial g_1}{\partial Q} \right| = |0| < \infty \\ \frac{\partial g_1}{\partial D} = 0, \left| \frac{\partial g_1}{\partial D} \right| = |0| < \infty. \end{array} \right. \quad (2.14)$$

From Eq (5.8):

$$\left\{ \begin{array}{l} \frac{\partial g_1}{\partial P} = \beta_1 S, \left| \frac{\partial g_1}{\partial P} \right| = |\beta_1 S| < \infty \\ \frac{\partial g_1}{\partial S} = \beta_1 P - (\mu + \alpha + \rho + \gamma), \left| \frac{\partial g_1}{\partial S} \right| = |\beta_1 P - (\mu + \alpha + \rho + \gamma)| < \infty \\ \frac{\partial g_1}{\partial R} = 0, \left| \frac{\partial g_1}{\partial R} \right| = |0| < \infty \\ \frac{\partial g_1}{\partial Q} = 0, \left| \frac{\partial g_1}{\partial Q} \right| = |0| < \infty \\ \frac{\partial g_1}{\partial D} = 0, \left| \frac{\partial g_1}{\partial D} \right| = |0| < \infty. \end{array} \right. \quad (2.15)$$

Similarly, one can do this for other equations. Our conclusion from the Existence of Solution theorem is that there is a unique solution to the model (5.1) in the domain Ξ by demonstrating the continuity and boundedness of each partial derivative. $\square$

2.3.5 Steady states of model

In mathematical epidemiology, when the variables in a model remain constant throughout time, the steady-state solutions are referred to as equilibrium states. The equilibrium of system (5.1) may be found by solving equations (2.16) to ascertain the long-term behaviors of the system.

$$\left\{ \begin{array}{ll} \Lambda - \beta_1 PS + \alpha(1 - \epsilon)S - (\mu + \phi + \beta_2)P + \pi\theta R & = 0, \\ \beta_1 PS - (\mu + \alpha + \rho + \gamma)S & = 0, \\ \alpha\epsilon S + \rho S - (\mu + \theta)R & = 0, \\ \beta_2 P + \phi P + (1 - \pi)\theta R - \mu Q & = 0, \\ \gamma S - \mu D & = 0. \end{array} \right. \quad (2.16)$$

The steady states of the model are evaluated as

$$\frac{dP}{dt} = 0, \quad \frac{dS}{dt} = 0, \quad \frac{dR}{dt} = 0, \quad \frac{dQ}{dt} = 0, \quad \frac{dD}{dt} = 0$$

Two types of steady states are produced by direct solutions of system (2.16). The trivial (disease free) steady state indicates the absence of smoking, while the positive internal (endemic) steady state characterizes the presence of smoking. From (2.16),

we get DFE of the model is given as

$$E_{q0} = (P^0, S^0, R^0, Q^0, D^0) = \left(\frac{\Lambda}{(\mu + \phi + \beta_2)}, 0, 0, \frac{\Lambda(\phi + \beta_2)}{(\mu + \phi + \beta_2)\mu}, 0 \right). \quad (2.17)$$

DFE will occur when $\mathcal{R}_0 < 1$, which means there is no infection. EEP of the system (2.16) is

$$\begin{aligned} E_{q1} &= (P^*, S^*, R^*, Q^*, D^*). \quad (2.18) \\ P^* &= \frac{\mu + \alpha + \rho + \gamma}{\beta_1}, \\ S^* &= \frac{\Lambda(\mu + \theta) - (\mu + \phi + \beta_2)(\mu + \alpha + \rho + \gamma)(\mu + \theta)}{(\mu + \theta)(\mu + \alpha + \rho + \gamma) - \alpha(\mu + \theta)(1 - \epsilon) - \pi\theta(\alpha\epsilon + \rho)}, \\ R^* &= \frac{(\alpha\epsilon + \rho)S^*}{\mu + \theta}, \\ Q^* &= \frac{(\beta_2 + \phi)P^* + (1 - \pi)\theta R^*}{\mu}, \\ D^* &= \frac{\gamma S^*}{\mu}. \end{aligned}$$

2.3.6 Threshold parameter $\mathcal{R}_0$

The basic reproduction number $\mathcal{R}_0$ [180] is used to quantify the spread of the sickness. The term $\mathcal{R}_0$ describes the infected population that an infected person may produce while they are contagious. Analysis of the stability of steady states depends critically on the model's $\mathcal{R}_0$. The population will not experience an epidemic if $\mathcal{R}_0$ less than 1, and the epidemic will persist if $\mathcal{R}_0$ is greater than 1.

Consider model (5.1) in which the total population is classified into five different classes. To make sure that the first n compartments show the infected individuals, we rearrange the system so that the smoking classes appear first. We now define $\mathbb{F}_i(y)$ represents the rate at which new smokers develop in compartment S , $\mathbb{V}_i^+(y)$ shows the rate at which people come to compartment S through ways apart from the smoking, and $\mathbb{V}_i^-(y)$ is the rate at which people leave compartment S . Equations provide the basis of the smoking model

$$y_i' = \mathbb{F}_i(y) - \mathbb{V}_i(y)$$

where $\mathbb{V}_i(y) = \mathbb{V}_i^-(y) - \mathbb{V}_i^+(y)$

Therefore, the disease-free steady state y_0 already exists, we construct $n \times n$ matrices of $\mathbb{F}$ and $\mathbb{V}$, which are defined by

$$\mathbb{F} = \frac{\partial \mathbb{F}_i}{\partial y_j}(y_0) \quad , \quad \mathbb{V} = \frac{\partial \mathbb{V}_i}{\partial y_j}(y_0)$$

with $1 \leq i, j \leq n$.

Since $\mathbb{F} \geq 0$ and $\det(\mathbb{V}) \neq 0$, then, $\mathbb{V}^{-1} \geq 0$, and also $\mathbb{F}\mathbb{V}^{-1} \geq 0$. Next-generation matrix is defined by the $\mathbb{F}\mathbb{V}^{-1}$. Therefore, $\mathbb{R}_0$ is

$$\mathbb{R}_0 = \rho(\mathbb{F}\mathbb{V}^{-1})$$

where $\rho(\mathbb{FV}^{-1})$ denote the largest eigenvalue of matrix $\mathbb{FV}^{-1}$.

To compute the $\mathcal{R}_0$, the next generation matrix (NGM) technique is utilized. Based on the methodology, from system (5.1), we obtain:

$$\mathbf{F} = \begin{bmatrix} \beta_1 PS \\ 0 \\ 0 \end{bmatrix}, \quad \mathbf{V} = \begin{bmatrix} (\mu + \alpha + \rho + \gamma)S \\ -\alpha\epsilon S + \mu R - \rho S + \theta R \\ -\beta_2 P - (1 - \pi)\theta R + \mu Q - \phi P \end{bmatrix}$$

The following information provides the Jacobi matrices of $\mathbf{F}$ and $\mathbf{V}$ at DFE, represented by $\mathcal{F}$ and $\mathcal{V}$:

$$\mathcal{F} = \begin{bmatrix} \frac{\Lambda\beta_1}{\mu+\phi+\beta_2} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, \quad \mathcal{V} = \begin{bmatrix} \mu + \alpha + \rho + \gamma & 0 & 0 \\ -\alpha\epsilon - \rho & \mu + \theta & 0 \\ 0 & -(1 - \pi)\theta & \mu \end{bmatrix}$$

The greatest eigenvalue is found in the product matrix $\mathcal{F}\mathcal{V}^{-1}$, as seen by

$$\mathcal{R}_0 = \frac{\beta_1\Lambda}{(\mu + \phi + \beta_2)(\alpha + \gamma + \mu + \rho)}. \quad (2.19)$$

2.3.7 Stability analysis of model

We shall evaluate the stability of the steady states of the model. $\mathcal{R}_0$, indicates that the disease's incidence will decline if $\mathcal{R}_0$ is less than 1. nothing change in case $\mathcal{R}_0 = 1$ and $\mathcal{R}_0$ greater than 1 shows sickness will rise. [235].

Local stability analysis

Theorem 2.7. *The DFE is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

Proof. The Jacobin of Eq (5.1) is written as :

$$J = \begin{pmatrix} -\beta_1 S - (\mu + \phi + \beta_2) & -\beta_1 P + \alpha(1 - \epsilon) & \pi\theta & 0 & 0 \\ \beta_1 S & \beta_1 P - (\mu + \alpha + \rho + \gamma) & 0 & 0 & 0 \\ 0 & \alpha\epsilon + \rho & -(\mu + \theta) & 0 & 0 \\ \beta_2 + \phi & 0 & (1 - \pi)\theta & -\mu & 0 \\ 0 & \gamma & 0 & 0 & -\mu \end{pmatrix} \quad (2.20)$$

Simulating Eq (2.20) at the DFE (E_{q0}), we get:

$$J(E_{q0}) = \begin{pmatrix} -(\mu + \phi + \beta_2) & -\beta_1 \frac{\Lambda}{\mu+\phi+\beta_2} + \alpha(1 - \epsilon) & \pi\theta & 0 & 0 \\ 0 & \beta_1 \frac{\Lambda}{\mu+\phi+\beta_2} - (\mu + \alpha + \rho + \gamma) & 0 & 0 & 0 \\ 0 & \alpha\epsilon + \rho & -(\mu + \theta) & 0 & 0 \\ \beta_2 + \phi & 0 & (1 - \pi)\theta & -\mu & 0 \\ 0 & \gamma & 0 & 0 & -\mu \end{pmatrix} \quad (2.21)$$

By transforming (2.21) into block matrices, the eigenvalues are obtained by the characteristic equations as follows:

$$(-(\mu + \phi + \beta_2 + \lambda))(\beta_1 \frac{\Lambda}{\mu + \phi + \beta_2} - (\mu + \alpha + \rho + \gamma) - \lambda)(-(\mu + \theta) - \lambda)(-\mu - \lambda)(-\mu - \lambda) = 0$$

From this equation, the five eigenvalues are $\lambda_1 = -(\mu + \phi + \beta_2)$, $\lambda_2 = \beta_1 \frac{\Lambda}{\mu + \phi + \beta_2} - (\mu + \alpha + \rho + \gamma) = \mathcal{R}_0 - 1$, $\lambda_3 = -(\mu + \theta)$, $\lambda_4 = -\mu$, $\lambda_5 = -\mu$. Hence all eigenvalues are negative, except λ_2 if $\mathcal{R}_0 < 1$ then λ_2 is negative. Hence, prove that E_{q0} is locally asymptotically stable. $\square$

Theorem 2.8. If $\mathcal{R}_0 > 1$, then EEP of Eq. (5.1) is locally asymptotically stable.

Proof.

$$J_1 = \begin{pmatrix} -\beta_1 S^* - (\mu + \phi + \beta_2) & -\beta_1 P^* + \alpha(1 - \epsilon) & \pi\theta & 0 & 0 \\ \beta_1 S^* & \beta_1 P^* - (\mu + \alpha + \rho + \gamma) & 0 & 0 & 0 \\ 0 & \alpha\epsilon + \rho & -(\mu + \theta) & 0 & 0 \\ \beta_2 + \phi & 0 & (1 - \pi)\theta & -\mu & 0 \\ 0 & \gamma & 0 & 0 & -\mu \end{pmatrix}$$

$$\det(J_1 - \lambda I) = 0$$

$$\begin{vmatrix} C_1 & C_2 & \pi\theta & 0 & 0 \\ \beta_1 S^* & C_3 & 0 & 0 & 0 \\ 0 & \alpha\epsilon + \rho & -(\mu + \theta + \lambda) & 0 & 0 \\ \beta_2 + \phi & 0 & (1 - \pi)\theta & -\mu - \lambda & 0 \\ 0 & \gamma & 0 & 0 & -\mu - \lambda \end{vmatrix} = 0$$

where

$$C_1 = -\beta_1 S^* - (\mu + \phi + \beta_2 + \lambda), \quad C_2 = -\beta_1 P^* + \alpha(1 - \epsilon), \\ C_3 = \beta_1 P^* - (\mu + \alpha + \rho + \gamma + \lambda).$$

The characteristic polynomial is expressed as

$$G(\lambda) = (-\mu - \lambda)^2 R(\lambda)$$

with

$$R(\lambda) = \lambda^3 + u_1 \lambda^2 + u_2 \lambda + u_3$$

where

$$u_1 = \beta_1 S^* - \beta_1 P^* + \mu + \alpha + \rho + \gamma + \phi + \beta^2$$

$$u_2 = 2\beta_1^2 S^* - \beta_1 S^* \mu - \beta_1 S^* \alpha - \beta_1 S^* \rho - \beta_1 S^* \gamma - \beta_1 S^* \lambda + 2\mu \beta_1 P^* + \phi \beta_1 P^* + \beta_1 \beta_2 P^*$$

$$u_3 = \beta_1 S^* \mu \alpha - 2\beta_1^2 P^* S^* \mu - 2\beta_1^2 P^* S^* \theta + \beta_1 S^* \mu^2 + \beta_1 S^* \mu \theta + \beta_1 S^* \alpha \theta + \beta_1 S^* \mu \rho + \beta_1 S^* \rho \theta \\ + \beta_1 S^* \mu \gamma - \beta_1 S^* \gamma \theta - \beta_1 P^* \mu^2 + \beta_1 P^* \mu \theta - \beta_1 P^* \mu \phi - \beta_1 P^* \theta \phi - \beta_1 \beta_2 P^* \mu - \beta_1 \beta_2 P^* \theta \\ + \mu \alpha \theta + \mu \rho \theta + \gamma \theta \mu - \mu^2 (\alpha + \rho + \gamma + \theta + \phi + \beta^2 + \mu)$$

(2.22)

Now, the discriminant of $R(\lambda)$ is given as [304]

$$D(R) = 18u_1u_2u_3 + (u_1u_2)^2 - 4u_3u_1^2 - 4u_2^2 - 27u_3^2$$

we have the following results: □

Corollary 2.9. [213] For $\mathcal{R}_0 > 1$, the EEP of model (5.1) is LAS if one of the following requirements is met for $R(\lambda)$

1. If $D(R) > 0$, then $u_1 > 0$, $u_3 > 0$, and $u_1u_2 > u_3$ is a necessary and sufficient condition for D^* to be locally stable.
2. If $D(R) < 0$, $u_1 \geq 0$, $u_2 \geq 0$, $u_3 > 0$, $\theta < \frac{2}{3}$ then D^* is LAS.
3. If $D(R) < 0$, $u_1 < 0$, $u_2 < 0$, and $\theta > \frac{2}{3}$, then all solutions of the eq(13) meet the conditions $|\arg(\lambda_i)| < \theta\frac{\pi}{2}$, $i = 1, 2, 3, 4, 5$.

Global stability analysis

This part identifies a number of key terms and symbols for taking into account the global stability of the model. By the Castillo-Chavez approach model (5.1) would be shown globally asymptotically stable at the DFE [199, 214].

Lemma 2.10. Consider the subsequent structure for an infectious model system:

$$\begin{cases} \frac{ds_1}{dt} = y_1(s_1, s_2) \\ \frac{ds_2}{dt} = y_2(s_1, s_2), y_2(s_1, 0) = 0. \end{cases} \quad (2.23)$$

where $s_1 \in \mathbb{R}^m$ denotes the uninfected population and $s_2 \in \mathbb{R}^n$ represents the infected. $s^* = (s_1^0, 0)$ is DFE Eq (2.23).

As long as $\mathcal{R}_0 < 1$ and the following criteria are met, the point $(y_1^0, 0)$ is a globally asymptotically stable equilibrium for the given system.

B_1 : For $\frac{ds_1}{dt} = y_1(s_1, 0)$, s_1^0 is a globally asymptotically stable.

B_2 : $\frac{ds_2}{dt} = J s_2 - \hat{y}_2(s_1, s_2)$ with $\hat{y}_2(s_1, s_2) \geq 0$ for $(s_1, s_2) \in \Pi$ in which $J = \frac{\partial x_2}{\partial t}(x_1(s_1^0, 0))$ is the Jacobi matrix that Metzler matrix, and Π is the biologically logical area of the model.

Theorem 2.11. The DFE is the global asymptotically stable of Eq (5.1) given that $\mathcal{R}_0 < 1$.

Proof. Assume that in model (5.1), the infected population is $p_2 = (S, R, D) \in \mathbb{R}^3$, and the uninfected population is $p_1 = (P, Q) \in \mathbb{R}^2$. Model (5.1) allows us to write as

$$\begin{cases} \frac{dP}{dt} = \Lambda - \beta_1 P S + \alpha(1 - \epsilon)S - (\mu + \phi + \beta_2)P + \pi\theta R, \\ \frac{dQ}{dt} = \beta_2 P + \phi P + (1 - \pi)\theta R - \mu Q. \end{cases} \quad (2.24)$$

Thus, we have

$$y_1(s_1, s_2) = \begin{pmatrix} \Lambda - \beta_1 P S + \alpha(1 - \epsilon)S - (\mu + \phi + \beta_2)P + \pi\theta R, \\ \beta_2 P + \phi P + (1 - \pi)\theta R - \mu Q. \end{pmatrix} \quad (2.25)$$

At $s^* = (s_1^0, 0)$, above equation becomes

$$\frac{ds_1}{dt} = y_1(s_1, 0) = \begin{pmatrix} \Lambda - (\mu + \phi + \beta_2)P \\ \beta_2 P + \phi P - \mu Q \end{pmatrix} \quad (2.26)$$

Eq (2.26) gives us

$$\begin{cases} P(t) = P(0)e^{-(\mu + \phi + \beta_2)t} + \frac{\Lambda}{\mu + \phi + \beta_2}(1 - e^{-(\mu + \phi + \beta_2)t}) \\ Q(t) = Q(0)e^{-\mu t} + \frac{(\beta_2 + \phi)\Lambda}{(\beta_2 + \phi + \mu)\tau}(1 - e^{-\mu t}) \end{cases} \quad (2.27)$$

As $t \rightarrow \infty$ the solution is:

$$\begin{cases} P(\infty) = \frac{\Lambda}{\mu + \phi + \beta_2} \\ Q(\infty) = \frac{\beta\Lambda}{(\mu + \phi + \beta_2)\mu}. \end{cases} \quad (2.28)$$

This means that (2.26) in Π is globally convergent, therefore satisfying criteria $\mathbf{B}_1$. To find out if condition two will be met, we now use $Y(s_1, s_2) = Zp_2 - \hat{y}_2(s_1, s_2)$. Additionally, we receive:

$$A = \begin{pmatrix} \beta_1 P_0 - (\mu + \alpha + \rho + \gamma) & 0 & 0 \\ \alpha\epsilon + \rho & -(\mu + \theta) & 0 \\ \gamma & 0 & -\mu \end{pmatrix} \begin{pmatrix} S \\ R \\ D \end{pmatrix} - \begin{pmatrix} \hat{y}_2(s_1, s_2) \\ 0 \\ 0 \end{pmatrix} \quad (2.29)$$

$$Z = \begin{pmatrix} \beta_1 P_0 - (\mu + \alpha + \rho + \gamma) & 0 & 0 \\ \alpha\epsilon + \rho & -(\mu + \theta) & 0 \\ \gamma & 0 & -\mu \end{pmatrix} \quad (2.30)$$

with

$$J = \begin{pmatrix} \beta_1(P_0 - P) - (\mu + \alpha + \rho + \gamma) \\ 0 \\ 0 \end{pmatrix} \quad (2.31)$$

The model indicates that the whole population is enclosed by P_0 . Consequently, $\beta_1 P - (\mu + \alpha + \rho + \gamma) \leq \beta_1(P_0) - (\mu + \alpha + \rho + \gamma)$ suggests that $\hat{y}_2(s_1, s_2)$ is positive definite. Additionally, the off-diagonal entries in matrix Z are positive, making it an M-matrix. The global asymptotic stability is therefore demonstrated by the satisfaction of $\mathbf{B}_2$. $\square$

Theorem 2.12. *If $\mathcal{R}_0 > 1$, then the EEP of the proposed model (5.1) is globally asymptotically stable.*

Proof. We consider a Lyapunov function $L_2(t)$ which is define as

$$\begin{aligned} L_2(t) = & \frac{1}{(\mu + \phi + \beta_2)} \left(P - P^* - P^* \ln \frac{P}{P^*} \right) + \Xi \left(S - S^* - S^* \ln \frac{S}{S^*} \right) \\ & + \frac{1}{(\mu + \theta)} \left(R - R^* - R^* \ln \frac{R}{R^*} \right) + \frac{1}{\mu} \left(Q - Q^* - Q^* \ln \frac{Q}{Q^*} \right) \\ & + \frac{1}{\mu} \left(D - D^* - D^* \ln \frac{D}{D^*} \right) \end{aligned}$$

Where $\Xi = \frac{(\mathcal{R}_0-1)}{(\mu+\alpha+\rho+\gamma)}$, $\mathcal{R}_0 > 1$.

The function $L_2(t)$ is defined as positive definite and continuous $\forall t \geq 0$. Now we have

$$\begin{aligned}
\frac{dL_2}{dt} &= \frac{d}{dt} \left[\frac{1}{(\mu + \phi + \beta_2)} \left(P - P^* - P^* \ln \frac{P}{P^*} \right) + \Xi \left(S - S^* - S^* \ln \frac{S}{S^*} \right) \right. \\
&\quad + \frac{1}{(\mu + \theta)} \left(R - R^* - R^* \ln \frac{R}{R^*} \right) + \frac{1}{\mu} \left(Q - Q^* - Q^* \ln \frac{Q}{Q^*} \right) \\
&\quad \left. + \frac{1}{\mu} \left(D - D^* - D^* \ln \frac{D}{D^*} \right) \right] \\
\frac{dL_2}{dt} &\leq \frac{1}{(\mu + \phi + \beta_2)} \left(1 - \frac{P^*}{P} \right) \frac{dP}{dt} + \Xi \left(1 - \frac{S^*}{S} \right) \frac{dS}{dt} + \frac{1}{(\mu + \theta)} \left(1 - \frac{R^*}{R} \right) \frac{dR}{dt} \\
&\quad + \frac{1}{\mu} \left(1 - \frac{Q^*}{Q} \right) \frac{dQ}{dt} + \frac{1}{\mu} \left(1 - \frac{D^*}{D} \right) \frac{dD}{dt} \\
&= \frac{1}{(\mu + \phi + \beta_2)} \left(1 - \frac{P^*}{P} \right) [\Lambda - \beta_1 P S + \alpha(1 - \epsilon)S + \pi\theta R - (\mu + \phi + \beta_2)P] \\
&\quad + \Xi \left(1 - \frac{S^*}{S} \right) [\beta_1 P S - (\mu + \alpha + \rho + \gamma)S] + \frac{1}{(\mu + \theta)} \left(1 - \frac{R^*}{R} \right) [\alpha\epsilon S + \rho S - (\mu + \theta)R] \\
&\quad + \frac{1}{\mu} \left(1 - \frac{Q^*}{Q} \right) [\beta_2 P + \phi P + (1 - \pi)\theta R - \mu Q] + \frac{1}{\mu} \left(1 - \frac{D^*}{D} \right) [\gamma S - \mu D]
\end{aligned} \tag{2.32}$$

using the endemic conditions,

$$\begin{aligned}
\Lambda - \beta_1 P^* S^* + \alpha(1 - \epsilon)S^* + \pi\theta R^* &= (\mu + \phi + \beta_2)P^* \\
\beta_1 P^* S^* &= (\mu + \alpha + \rho + \gamma)S^* \\
\alpha\epsilon S^* + \rho S^* &= (\mu + \theta)R^* \\
\beta_2 P^* + \phi P^* + (1 - \pi)\theta R^* &= \mu Q^* \\
\gamma S^* &= \mu D^*
\end{aligned}$$

in the above equation, we get

$$\begin{aligned}
\frac{dL_2}{dt} &\leq \frac{1}{(\mu + \phi + \beta_2)} \left(1 - \frac{P^*}{P} \right) [(\mu + \phi + \beta_2)P^* - (\mu + \phi + \beta_2)P] \\
&\quad + \Xi \left(1 - \frac{S^*}{S} \right) [(\mu + \alpha + \rho + \gamma)S^* - (\mu + \alpha + \rho + \gamma)S] \\
&\quad + \frac{1}{(\mu + \theta)} \left(1 - \frac{R^*}{R} \right) [(\mu + \theta)R^* - (\mu + \theta)R] \\
&\quad + \frac{1}{\mu} \left(1 - \frac{Q^*}{Q} \right) [\mu Q^* - \mu Q] + \frac{1}{\mu} \left(1 - \frac{D^*}{D} \right) [\mu D^* - \mu D] \\
&= \left(\frac{P - P^*}{P} \right) (P^* - P) + (\mathcal{R}_0 - 1) \left(\frac{S - S^*}{S} \right) (S^* - S) + \left(\frac{R - R^*}{R} \right) (R^* - R) + \\
&\quad \left(\frac{Q - Q^*}{Q} \right) (Q^* - Q) + \left(\frac{D - D^*}{D} \right) (D^* - D)
\end{aligned}$$

$$\frac{dL_2}{dt} \leq -\frac{(P - P^*)^2}{P} - \frac{(S - S^*)^2}{S}(R_0 - 1) - \frac{(R - R^*)^2}{R} - \frac{(Q - Q^*)^2}{Q} - \frac{(D - D^*)^2}{D}$$

This implies,

$$\frac{dL_2}{dt} \leq 0$$

It follows that if $\mathcal{R}_0 > 1$, then we have $\frac{dL_2}{dt} \leq 0$. Therefore, $L_2(t)$ is bounded and decreasing. In addition, we know that $\frac{dL_2}{dt} = 0$ if and only if $S(t) = S^*, R(t) = R^*, Q(t) = Q^*, D(t) = D^*$. Hence, when $\mathcal{R}_0 > 1$, the endemic state of model (5.1) is globally stable [213]. $\square$

2.3.8 Sensitivity analysis

To demonstrate the impact of each parameter on smoking transmission, we conducted a sensitivity study. To do this analysis, we used the normalized sensitivity index idea [130].

Definition 2.13. The normalized forward sensitivity index (NFSI) of $\mathcal{R}_0$ is written as:

$$\Pi_p^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial I_1} \times \frac{A}{\mathcal{R}_0}$$

where $\mathcal{R}_0$ is the basic reproduction number, and A represents all parameters.

$$\mathcal{R}_0 = \frac{\beta_1 \Lambda}{(\mu + \phi + \beta_2)(\alpha + \gamma + \mu + \rho)}.$$

The sensitivity index of $\mathcal{R}_0$ to the model's parameters can be computed as:

TABLE 2.2: Sensitivity index of $\mathcal{R}_0$ of model (5.1)

Parameters	Normalized Sensitivity index	Sensitivity index	Index values
Λ	$\Pi_{\Lambda}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \Lambda} \times \frac{\Lambda}{\mathcal{R}_0}$	+ev	1
β_1	$\Pi_{\beta_1}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \beta_1} \times \frac{\beta_1}{\mathcal{R}_0}$	+ev	1
μ	$\Pi_{\mu}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \mu} \times \frac{\mu}{\mathcal{R}_0} = -\frac{\mu(\alpha + \gamma + 2\mu + \rho + \phi + \beta_2)}{(\alpha + \gamma + \mu + \rho)(\mu + \phi + \beta_2)}$	-ev	-1.088
ϕ	$\Pi_{\phi}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \phi} \times \frac{\phi}{\mathcal{R}_0} = -\frac{\phi}{\mu + \phi + \beta_2}$	-ev	-0.303
β_2	$\Pi_{\beta_2}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \beta_2} \times \frac{\beta_2}{\mathcal{R}_0} = -\frac{\beta_2}{\mu + \phi + \beta_2}$	-ev	-0.091
α	$\Pi_{\alpha}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \alpha} \times \frac{\alpha}{\mathcal{R}_0} = -\frac{\alpha}{\alpha + \gamma + \mu + \rho}$	-ev	-0.241
γ	$\Pi_{\gamma}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \gamma} \times \frac{\gamma}{\mathcal{R}_0} = -\frac{\gamma}{\alpha + \gamma + \mu + \rho}$	-ev	-0.036
ρ	$\Pi_{\rho}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \rho} \times \frac{\rho}{\mathcal{R}_0} = -\frac{\rho}{\alpha + \gamma + \mu + \rho}$	-ev	-0.241

The output of the sensitivity analysis of $\mathcal{R}_0$ is shown in Table 3.2, that presents a normal sensitivity index up to three decimal places along with their graphics in Fig 2.2, which indicates that only Λ and β_1 show the positively increasing behaviors, rest of others shows negative sense. From the figure, we analyze that Λ and β_1 are significant parameters for $\mathcal{R}_0$ in the positive sense, while μ is significant in the negative sense from the rest of the negative sense parameters. As the death rate increases, the probability of disease transmission decreases, according to the negative sensitivity index μ . The value of $-1.088 \approx -1$ indicates a strong sensitivity of disease transmission to changes in the natural death rate, suggesting that even small increases in the natural death rate can significantly reduce the basic reproduction number. We

conclude that to control the transmission of smoking disease, we have to manage the β_1 and Λ by decreasing their value and increasing the values of other parameters in the negative sense. To check the detailed view of $\mathcal{R}_0$ behavior, we vary the different parameters w.r.t $\mathcal{R}_0$ in Fig 2.3. Fig 2.3(a) indicates the variation of parameter α and ϕ . we analyze that both parameters show the decreasing behavior of $\mathcal{R}_0$. Small variation in self-awareness ϕ shows a sudden change in recovery rate of smokers α , due to which $\mathcal{R}_0$ shows positively increasing behaviors till reaching limit 1 at initial values of both parameters. When α along the x-axis and ϕ along the y-axis change from 0 to 1, the reproduction no approaches zero, which shows that self-awareness can increase the recovery rate. Impact of parameters β_1 and ρ on $\mathcal{R}_0$ shown in Fig 2.3(b), which shows that with the increase of these parameters, $\mathcal{R}_0$ increases. when we increase the treatment of smokers ρ transmission of smoking β_1 also decrease almost with same probability. This indicates the effectiveness of treatment for addicts of smoking. Fig 2.3(c) reflects the transmission of smoking β_1 and natural mortality rate μ . This scenario is more important to analyze for the prevention of smokers from smoking. We see that with the increase in smoking, transmission reproduction shows no increase. The natural death rate does not show too much impact. But still overcoming the smoking dynamics β_1 is very important along with the treatment to control the mortality rate due to smoking-related disease. Similarly, one can easily investigate the behaviors of other parameters on $\mathcal{R}_0$.

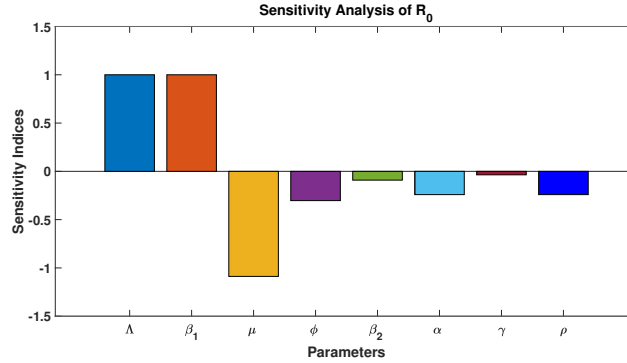
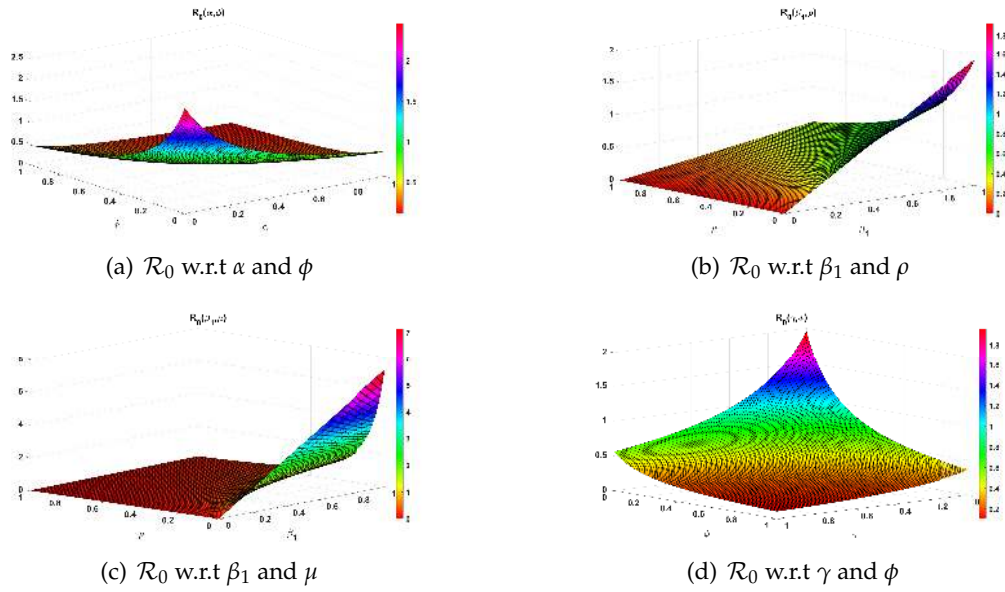


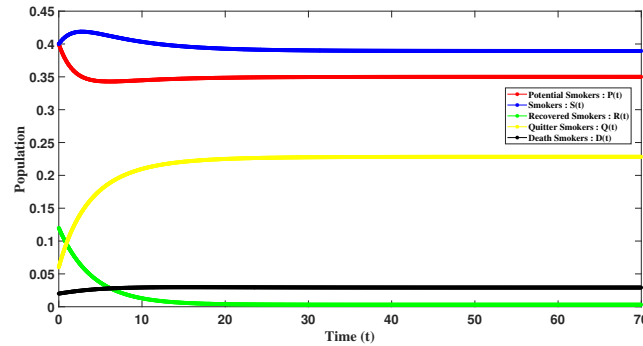
FIGURE 2.2: Sensitivity analysis of $\mathcal{R}_0$ shows the significant parameters of model (5.1).

2.3.9 Numerical simulation of model without treatment

In this section the numerical simulation of mathematical model to control smoking dynamics without treatment is carried out with ICs $\{P(0) = 0.4, S(0) = 0.4, R(0) = 0.12, Q(0) = 0.06, D(0) = 0.02\}$, $h = 0.01$, $T = [0 \ 70]$ and all biological parameters given in Table 3.2 using MATLAB using ode45 routine (RK4 method) shown in Fig 7.2-7.3. It can be analyzed that all positive solutions are in feasible regions. First, we want to show the simple behavior of the model (5.1) without the impact of treatment. So we take model (5.1) with treatment parameter (ρ) zero and predict the model behavior of all classes in Fig 7.2 and separately in Fig 7.3. From these figures, we can easily predict that when the potential smokers P have effective contact with the smokers S , the transmission of smoking increases, due to which the smokers' S curve shows a sudden jump from the initial point as the potential smoker's curve is laid down and reaches its steady state after 10 days. Due to the recovered smokers, R again shows the smoking symptoms move to potential smokers, recovered class decreases, showing equilibrium state after 20 days, and quitter Q increases with the

FIGURE 2.3: Impact of different parameters on $\mathcal{R}_0$ of model (5.1).

immediate effect of self-awareness, and some occasional smokers who quit smoking from the start. As the recovered class decreases, the death of smokers D increases due to the sudden effects of smoking-related disease. From this scenario, we can easily see that the treatment of smokers is an important factor in controlling the addiction to smoking and increasing the recovery rate, along with decreasing the mortality due to smoking-related diseases like cancer, heart disease, mental issues, etc.

FIGURE 2.4: Numerical simulation of model (5.1) without treatment of smokers (ρ) with parameters in Table 3.2, step size $h = 0.01$ and $T = [0 \ 70]$.

2.3.10 Numerical simulation of model with treatment

Here we discuss the numerical simulation of mathematical model to control smoking dynamics with treatment carried out with ICs $\{P(0) = 0.4, S(0) = 0.4, R(0) = 0.12, Q(0) = 0.06, D(0) = 0.02\}$, $h = 0.01$, $T = [0 \ 70]$ and all biological parameters given in Table 3.2 shown in Fig 3.2-2.7. Studies show that every feasible solution is within reach. We want to present the dynamical behavior of the model (5.1) with the impact of the treatment parameter. So we implement model (5.1) with treatment

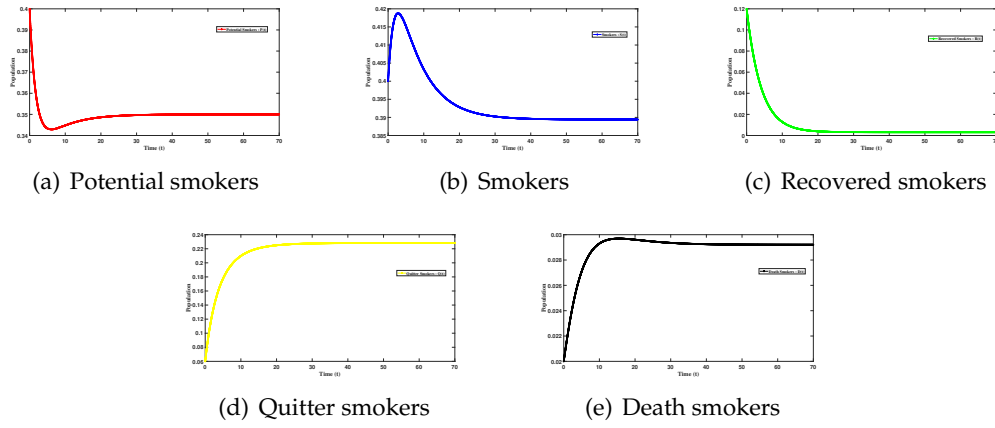


FIGURE 2.5: Numerical simulation of model (5.1) without treatment of smokers (ρ) separately with parameters in Table 3.2, step size $h = 0.01$ and $T = [0 \ 70]$.

parameter $\rho = 0.1$ and forecasts each class's model action in Fig 3.2 and separately in Fig 2.7. Based on this data, we can estimate that when the potential smokers P are in contact with smokers S , the transmission of smoking increases. By applying the treatment, the smoker's S moves to the recovered class. Due to this operation, smokers' S curve shows a sudden downfall from the initial point as the potential smoker's curve lay down and moved up after some time and reached their steady state after 40 days. Due to the increase in the recovery rate and people who were treated successfully move to recovered smokers R shown the less smoking symptoms which move to potential smokers, recovered class increase and reached an equilibrium state after 50 days and quitter Q increases with immediate effect of self awareness, some occasional smokers how quit the smoking from starting and also the high success rate of treatment. As the recovered class increases, the death of smokers D increases due to the sudden effects of smoking-related disease and moves down after some time by showing their equilibrium state after 40 days. From this scenario, we can easily see the strong impact of the treatment of smokers to control the addiction to smoking and increase the recovery rate, along with decreasing the mortality due to smoking-related diseases like cancer, heart disease, mental issues, etc.

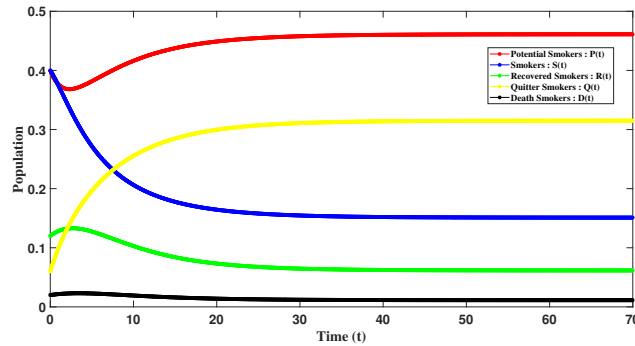


FIGURE 2.6: Numerical simulation of model (5.1) with treatment of smokers (ρ) with parameters in Table 3.2, step size $h = 0.01$ and $T = [0 \ 70]$.

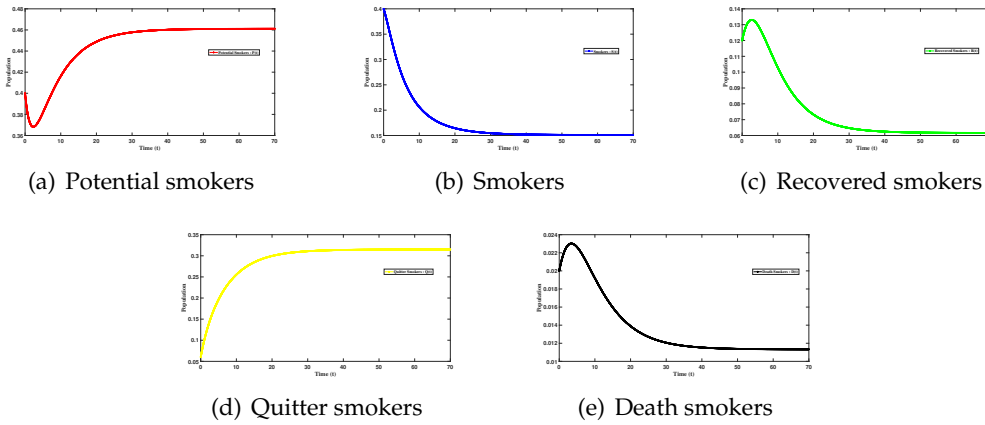


FIGURE 2.7: Numerical simulation of model (5.1) with treatment of smokers (ρ) separately with parameters in Table 3.2, step size $h = 0.01$ and $T = [0 \ 70]$.

Effect of treatment to overcome smoking dynamics

During the formulation of model (5.1), we utilize the treatment factor ρ to control smoking addiction. Here we will study the implementation of ρ on smokers S and describe their complex behavior on $\mathcal{R}_0$ to show the change in transmission of smoking when potential and active smokers are effectively contacted. In Fig 3.6 we present the variation of treatment ρ on model (5.1) smokers compartment. Fig 3.6(a) describes the behavior of active smokers when the treatment parameters change to different values at $\rho = \{0, 0.02, 0.05, 0.1, 0.17\}$. We conclude that when the treatment increases, then the transmission of smoking gradually decreases, which shows that, effectively, the people who are addicted to smoking are treated successfully and move to recovered smokers. If we see the smoker curve without treatment mean at $\rho = 0$, the smokers initially jump and go to their equilibrium state after reaching the peak point. When the full treatment is implemented at $\rho = 0.17$, we see a sudden decrease in the smoking individuals, indicating the effectiveness of ρ . This smoking curve shows their equilibrium state after approximately 40 days. Fig 3.6(b) indicates the 3D view of smoker S when the value of treatment ρ varies to study the model (5.1) characteristics. We can easily see a gradual decrease in the number of smokers with the increase in treatment value. In epidemiology, $\mathcal{R}_0$ is an effective function that grants the stability of the dynamics along with the persistence or dieout of the disease in the community. To keep this thing in mind, we check the impact of the treatment ρ of the addicted smokers to control the smoking transmission by a detailed view of $\mathcal{R}_0$ as a function of parameters ρ and μ in Fig 2.9. From the figure, we see that when we increase the treatment rate of smokers, $\mathcal{R}_0$ decreases with the decrease in the transmission of smoking in other populations. Fig 2.9(a) indicates the $\mathcal{R}_0$ behavior without treatment in which the $\mathcal{R}_0$ curve approaches to 12, which shows that the greater $\mathcal{R}_0$ means the disease persists in the community. If we see Fig 2.9(b) in which we increase the treatment $\rho = 0.05$, the $\mathcal{R}_0$ approaches to 9, and smoking transmission becomes low. When the treatment $\rho = 0.1$ in Fig 2.9(c), then $\mathcal{R}_0$ approaches to 7 and transmission of smoking decreases as compared to the above data. Similarly, when we go with full treatment $\rho = 0.17$, then $\mathcal{R}_0$ approaches to 5, which shows the lowest reproduction no as compared to the above data and smoking transmission gradually decreases, guaranteeing the effectiveness of successful treatment on the addicted smokers shown in Fig 2.9(d). This surprising behavior

shows that to control the smoking dynamics, each needs a different number of doses until it fully recovers. From the above discussion, we come to the point that treatment is effective and a relabel factor in controlling the smoking dynamics in the community.

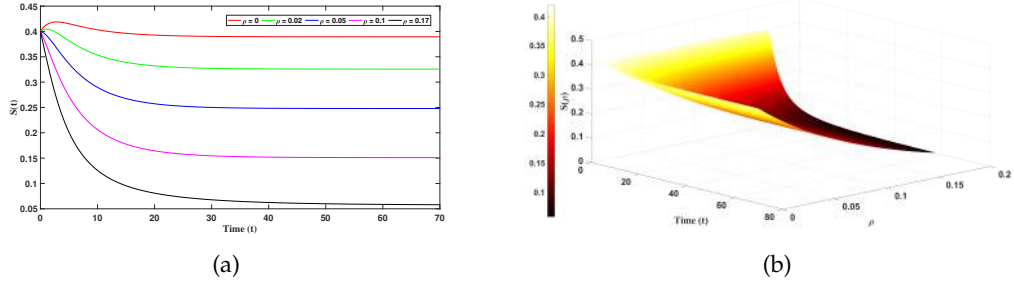


FIGURE 2.8: Numerical simulation of model (5.1) with variation of treatment of smokers (ρ): (a) shows the variation of $\rho = \{0, 0.02, 0.05, 0.1, 0.17\}$ on smoker S , (b) presents the 3D views by variation of $\rho = 0 : 0.001 : 0.17$.

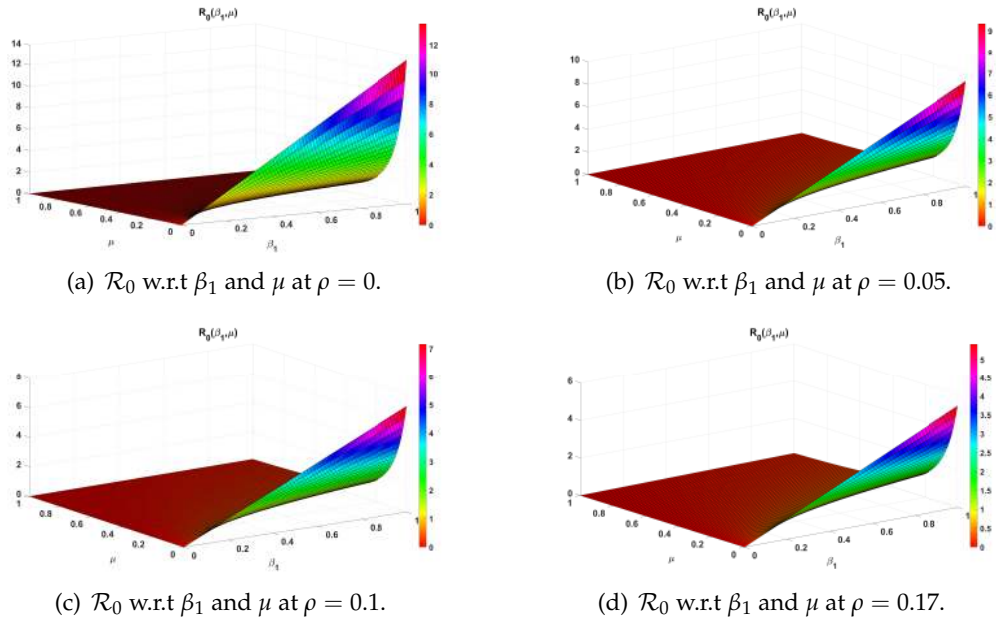


FIGURE 2.9: Simulation of $\mathcal{R}_0$ of model (5.1) w.r.t β_1 and μ with variation of treatment of smokers (ρ): Variation of $\rho = \{0, 0.02, 0.05, 0.1, 0.17\}$ on $\mathcal{R}_0$ w.r.t β_1 and μ are shown.

Effect of treatment on smoking-related disease mortality

During the literature review, we have seen the unexpected death ratio due to SRD. We have seen that most of the people who are addicted to smoking and continuously smoke are forced by their nature and behavioral characteristics and did not quit smoking. These people are always at risk of developing serious diseases like heart disease, lung cancer, mouth cancer, stomach issues, etc, due to smoking. This serious disease may cause the death of these people easily. To study that phenomenon, we

formulate the death smokers D compartment in model (5.1). Here, we analyze this issue with a detailed study and analysis of its mathematical characteristics in real-life scenarios. Numerical implementation of the death smokers D is given in Fig 2.10 with the variation of treatment of smokers ρ . The different values of the treatment parameter are $\rho = \{0, 0.02, 0.05, 0.1, 0.17\}$ predict the death of smokers on different treatment levels shown in Fig 2.10(a). From this figure, we expect that when there is no treatment, $\rho = 0$, death smokers start increasing gradually from the initial and after reaching their higher level, become constant to show an equilibrium state after 40 days. This indicates that the transmission of smoking increases without any restriction, and most people are badly affected by smoking diseases and move to death, showing the higher death rate of addicted smokers. But when we start applying the treatment from basic to full, i.e, 0 to 0.17, the death smoker consistently decreases and initially goes to a peak when the smoking persists, then decreases instantly by showing their equilibrium state after 40 days, as shown in the death smoking curve behavior at $\rho = 0.17$. These states suggest that successful implementation of clinical or behavioral treatment can save many people from death and decrease the death rate γ . The 3D view of death smokers D w.r.t the variation of treatment parameter ρ is given in Fig 2.10(b). In this view, one can easily see how smoothly the death of smokers decreases when we go from the treatment value from 0 to 0.17, and the bar value decreases from 0.025 to 0.005. This is due to the realistic impact of the treatment on the addicted smoker who is badly affected and completes their treatment process carefully, moving to the recovered class. For critical observation of $\mathcal{R}_0$ w.r.t the combined effect of γ and μ , we vary the treatment value and see the explicit behavior of the mortality rate due to smoking in Fig 3.8. Initially, when there is no treatment $\rho = 0$, the reproduction no approaches to 12 with γ taking along the x-axis, μ along the y-axis, and $\mathcal{R}_0$ along the z-axis. with time, when both parameters approach 1, then the reproduction no goes to zero as shown in Fig 3.8(a). If we increase the treatment from 0 to 0.1, then we see the sudden decrease of the reproduction no approaches to 6 now when both the parameters are at their initial points and showing their effect when both approaches to 1, then $\mathcal{R}_0$ goes to zero as seen in Fig 3.8(b). Finally, if we take full treatment $\rho = 0.17$, then we observe the sudden decay of $\mathcal{R}_0$ value from 12 to 5 according to previous data, see Fig 3.8(c). This presents the most realistic and important factor to control the mortality rate due to smoking-related diseases. we have seen a consistent scenario in all images that at initial $\mathcal{R}_0$ is at peak and decreases when γ and μ approach 0. This is due to the sudden increase in the transmission of smoking, due to which the mortality rate is also at its peak and moves down with time. We also observe the combined impact of the transmission rate of smoking β_1 and γ mortality rate due to smoking on the growth of $\mathcal{R}_0$ in Fig 2.12. In this figure, we see that despite the effect of β_1 , the mortality rate due to smoking γ increases with time. In Fig 2.12(a) when $\rho = 0$ we see that the value of $\mathcal{R}_0$ is 0.45, while in Fig 2.12(b)-(c) this value is 0.4 which indicates that when treatment increases the smoking mortality rate is under control until people start to follow the prevention measure to decrease the β_1 . From all the above discussions, we concluded that effective use of clinical or behavioral therapies as treatment for smokers can give realistic results about decreasing the chance of a persistent scenario of smoking dynamics.

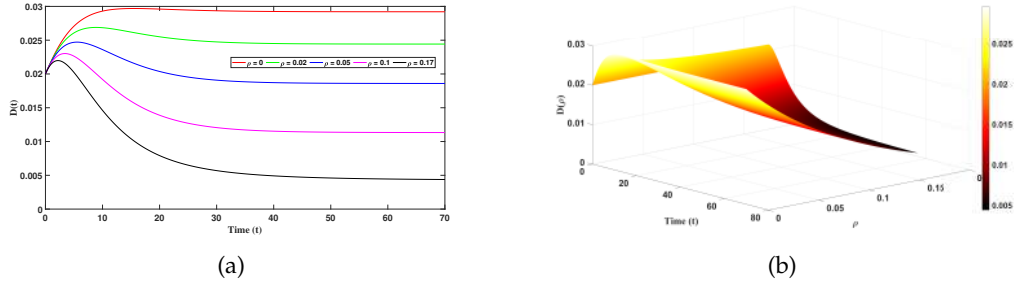


FIGURE 2.10: Numerical simulation of model (5.1) with variation of treatment of smokers (ρ): **(a)** shows the variation of $\rho = \{0, 0.02, 0.05, 0.1, 0.17\}$ on death smoker D , **(b)** presents the 3D views by variation of $\rho = 0 : 0.001 : 0.17$.

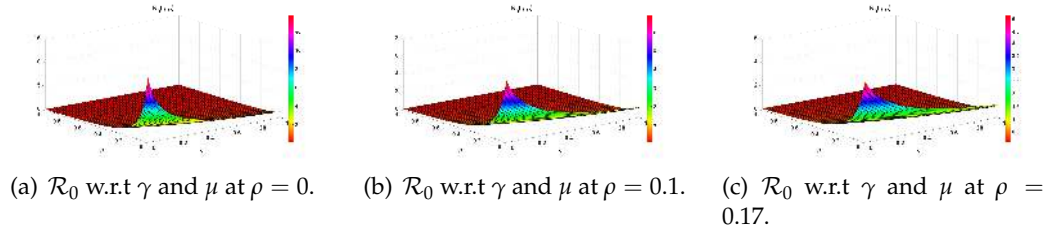


FIGURE 2.11: Simulation of $\mathcal{R}_0$ of model (5.1) w.r.t γ and μ with variation of treatment of smokers (ρ): Variation of $\rho = \{0, 0.1, 0.17\}$ on $\mathcal{R}_0$ w.r.t γ and μ are shown.

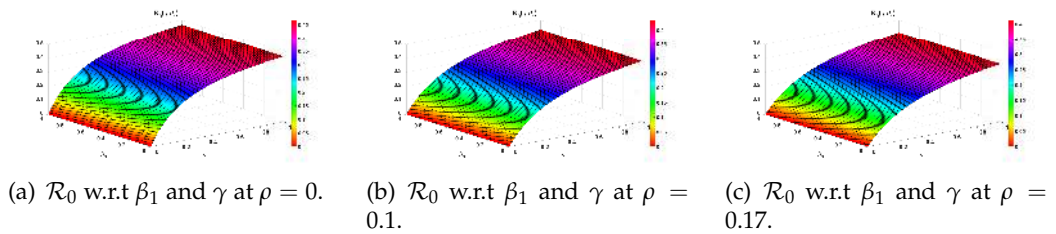


FIGURE 2.12: Simulation of $\mathcal{R}_0$ of model (5.1) w.r.t β_1 and γ with variation of treatment of smokers (ρ): Variation of $\rho = \{0, 0.1, 0.17\}$ on $\mathcal{R}_0$ w.r.t β_1 and γ are shown.

2.4 Mathematical model to advertise daily news of addicted smokers through media and treatment to control smoking dynamics

In this section, we introduce a mathematical model to explain how smoking spreads across society, emphasizing the function of public awareness efforts. By increasing public awareness of smoking's adverse effects, this model underlines the effectiveness of such awareness programs in slowing down the development of smoking addiction. Formulate a model to control the spread of smoking in the community by the use of proper treatment and raising awareness to advertise the number of active smokers S daily as a fear factor, due to which people take self-measures. The primary objective of the model is to assess the efficacy of media initiatives in reducing smoking rates. The approach indicates that smoking may be drastically decreased by advertising the total no of daily smokers addicted and raising awareness through ongoing media coverage, providing a potent means of addressing the smoking dynamics.

People recruited into the community become smokers after effective contact with active smokers S with a bilinear incident rate $\beta_3 e^{-mS} PS$. We use exponential form incident rate $\beta_3 e^{-mS}$ in which β_3 is the effect contact rate of potential smoker P with the active smokers S to become new smokers in population, m is the positive constant which shows the weight of media coverage of active smokers (clinical) to overcome the smoking dynamics and e^{-mS} is the function that represents the average number of active smokers S reported daily through media advertisement. Here we define the different cases for the variation of parameter m to check the behavior of the function $\beta_3 e^{-mS}$.

- When $m=0$

If $m = 0$, then the function $\beta_3 e^{-mS} = 1$, which indicates that there is no impact of media coverage on the transmission of smoking disease from potential to active class. In this case, the transmission rate remains constant irrespective of media coverage of clinically active smokers or how many of people are addicted to smoking. The model assumes that public opinion is unaffected by reporting, awareness efforts, or any preventative actions such as mask-wearing, distancing from the smoker, or getting some clinical treatment. Smoking spreads as it normally would among people who are ignorant of it or don't care about it.

- When $m > 0$

If $m > 0$, then $\beta_3 e^{-mS}$ becomes a decreasing function for the active smoker S which implies that when the number of active smokers cases rises and the active smoker's numbers are covered by the media more frequently, public awareness and caution grow, which slows the spread of the smoking dynamics. People could take up preventative measures like mask wearing when going out, making social separation from smokers, or bettering their gathering habits. The influence of media coverage increases with increasing m value. A larger m indicates that when media awareness rises, the transmission rate will drop more significantly.

- When $m \gg \text{large}$

If $m > 0$, then function $\beta_3 e^{-mS}$ approaches to 0 for the increase of active smoker S . This suggests that when there is extensive media coverage, the rate

of transmission can be significantly decreased since most people will take the necessary precautions, which will result in a large decrease in the number of new smokers. But as we know, smoking dynamics is a global real-world issue. So this identical situation can never happen because there is no 100% effect of media coverage to die out the smoking addiction due to environmental and behavioral factors.

A system of nonlinear differential equations for mathematical models with the addition of a bilinear incidence rate to advertise daily news of addicted smokers through media and treatment to control smoking dynamics is given as:

$$\begin{cases} \frac{dP}{dt} = \Lambda - \frac{\beta_3 e^{-mS} PS}{\mathcal{N}} + \alpha(1 - \epsilon)S - (\mu + \phi + \beta_2)P + \pi\theta R \\ \frac{dS}{dt} = \frac{\beta_3 e^{-mS} PS}{\mathcal{N}} - (\mu + \alpha + \rho + \gamma)S \\ \frac{dR}{dt} = \alpha\epsilon S + \rho S - (\mu + \theta)R \\ \frac{dQ}{dt} = \beta_2 P + \phi P + (1 - \pi)\theta R - \mu Q \\ \frac{dD}{dt} = \gamma S - \mu D \end{cases} \quad (2.33)$$

with the initial conditions

$$P(0) \geq 0, S(0) \geq 0, R(0) \geq 0, Q(0) \geq 0, D(0) \geq 0$$

2.4.1 Steady states of model

The DFE of the model (2.33) evaluated as

$$E_{q0} = (P^0, S^0, R^0, Q^0, D^0) = \left(\frac{\Lambda}{(\mu + \phi + \beta_2)}, 0, 0, \frac{\Lambda(\phi + \beta_2)}{(\mu + \phi + \beta_2)\mu}, 0 \right).$$

Let the EEP of model (2.33) be denoted by $E_{q1} = (P^*, S^*, R^*, Q^*, D^*)$ given as

$$\begin{aligned} P^* &= \frac{\mu + \alpha + \rho + \gamma}{\beta_3 e^{-mS^*}} \\ S^* &= \Lambda - (\mu + \phi + \beta_2) \frac{\mu + \alpha + \rho + \gamma}{\beta_3 e^{-mS^*}} \\ R^* &= \frac{\alpha\epsilon + \rho}{\mu + \theta} \left(\Lambda - (\mu + \phi + \beta_2) \frac{\mu + \alpha + \rho + \gamma}{\beta_3 e^{-mS^*}} \right) \\ Q^* &= (\beta_2 + \phi) \frac{\mu + \alpha + \rho + \gamma}{\mu \beta_3 e^{-mS^*}} + (1 - \pi)\theta \frac{\alpha\epsilon + \rho}{\mu(\mu + \theta)} \left(\Lambda - (\mu + \phi + \beta_2) \frac{\mu + \alpha + \rho + \gamma}{\beta_3 e^{-mS^*}} \right) \\ D^* &= \frac{\gamma S^*}{\mu} \end{aligned} \quad (2.34)$$

2.4.2 Threshold parameter R_0

According to [130], the next-generation matrix is used to determine the basic reproduction number. The new infection terms' matrix and the leftover transition terms'

matrix are provided, respectively, in the following

$$\mathbf{F} = \begin{pmatrix} \frac{\beta_3 e^{-mS} PS}{N} \\ 0 \\ 0 \end{pmatrix}, \quad \mathbf{V} = \begin{pmatrix} (\mu + \alpha + \rho + \gamma)S \\ -\alpha\epsilon S + \mu R - \rho S + \theta R \\ -\beta_2 P - (1 - \pi)\theta R + \mu Q - \phi P \end{pmatrix}$$

The following information provides the Jacobi matrices of $\mathbf{F}$ and $\mathbf{V}$ at DFE, represented by $\mathcal{F}$ and $\mathcal{V}$:

$$\mathcal{F} = \begin{pmatrix} \frac{\Lambda\beta_3(e^{-mS}(-mS) + e^{-mS})}{\mu + \phi + \beta_2} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad \mathcal{V} = \begin{pmatrix} \mu + \alpha + \rho + \gamma & 0 & 0 \\ -\alpha\epsilon - \rho & \mu + \theta & 0 \\ 0 & -(1 - \pi)\theta & \mu \end{pmatrix}$$

The greatest eigenvalue is found in the product matrix $\mathcal{F}\mathcal{V}^{-1}$, as seen by

$$\mathcal{R}_0 = \frac{\Lambda\beta_3}{(\mu + \phi + \beta_2)(\mu + \alpha + \rho + \gamma)} \quad (2.35)$$

2.4.3 Sensitivity analysis

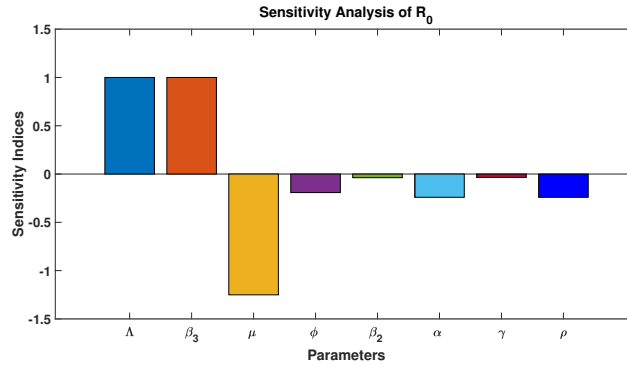
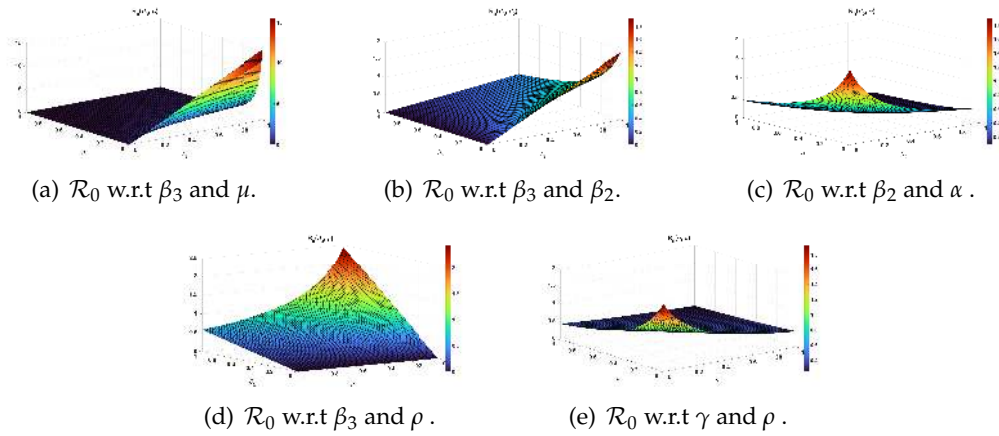
We performed a sensitivity analysis using the normalized sensitivity index concept [130]. For calculating the sensitivity index of $\mathcal{R}_0$ to the parameters of model (2.33), implementation given in Table 3.4. Graphical results of the sensitivity of model (2.33) are shown in Fig 2.13. From this analysis, we concluded that only Λ , β_3 , and μ are the significant parameters. We see that parameters Λ , β_3 are significant in the positive sense, while μ is significant in a negative sense. To control the smoking dynamics, we have to minimize the parameters Λ , β_3 , due to which the transmission of smoking in the population decreases; also, the new individuals who will be added or removed from the potential smokers will be managed. The behavior of $\mathcal{R}_0$ w.r.t different parameters is expressed in Fig 2.14. This behavior explains the persistence or die-out probability of the model. The transmission of smoking β_3 and natural mortality μ variation on $\mathcal{R}_0$ seen in Fig 2.14(a). With the increase of the β_3 , the reproduction value also increases, and the mortality rate slightly increases, showing the proportional relation. To decrease that dynamics, we have to manage the smoking transmission value concluded from Fig 2.14(b). To check the combined impact of smoking transmission β_3 and the percentage of potential smokers who quit smoking β_2 on $\mathcal{R}_0$, we gave a surface view in Fig 2.14(c), which indicates that β_3 has more impact on $\mathcal{R}_0$ as compared to β_2 . When smoking transmission increases, reproduction also increases, which gives certainty of the persistence of smoking dynamics. More clear behavior can be seen in Fig 2.14(d). To check the effect of treatment of smokers ρ and recovery rate γ on $\mathcal{R}_0$, Fig 2.14(e) implies that when the smoking dynamics starts, then the reproduction is not high when the treatment is applied to smokers with time, the recovery rate of smokers increases, and reproduction decreases approach to zero.

2.4.4 Numerical simulation of model with daily media advertisement of smokers and treatment

In this section, we explain the numerical results of model (2.33) with the combined use of treatment of smokers and media to advertise a daily number of smokers in the community, which will be utilized to increase fear in individuals. In this way, they

TABLE 2.3: Sensitivity index of $\mathcal{R}_0$ of model (2.33)

Parameters	Normalize Sensitivity Index	Sensitivity index	Index values
Λ	$\Pi_{\Lambda}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \Lambda} \times \frac{\Lambda}{\mathcal{R}_0}$	+ev	1
β_3	$\Pi_{\beta_3}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \beta_3} \times \frac{\beta_3}{\mathcal{R}_0}$	+ev	1
μ	$\Pi_{\mu}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \mu} \times \frac{\mu}{\mathcal{R}_0} = -\frac{\mu(\alpha+\gamma+2\mu+\rho+\phi+\beta_2)}{(\alpha+\gamma+\mu+\rho)(\mu+\phi+\beta_2)}$	-ev	-1.251
ϕ	$\Pi_{\phi}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \phi} \times \frac{\phi}{\mathcal{R}_0} = -\frac{\phi}{\mu+\phi+\beta_2}$	-ev	-0.192
β_2	$\Pi_{\beta_2}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \beta_2} \times \frac{\beta_2}{\mathcal{R}_0} = -\frac{\beta_2}{\mu+\phi+\beta_2}$	-ev	-0.038
α	$\Pi_{\alpha}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \alpha} \times \frac{\alpha}{\mathcal{R}_0} = -\frac{\alpha}{\alpha+\gamma+\mu+\rho}$	-ev	-0.241
γ	$\Pi_{\gamma}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \gamma} \times \frac{\gamma}{\mathcal{R}_0} = -\frac{\gamma}{\alpha+\gamma+\mu+\rho}$	-ev	-0.036
ρ	$\Pi_{\rho}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \rho} \times \frac{\rho}{\mathcal{R}_0} = -\frac{\rho}{\alpha+\gamma+\mu+\rho}$	-ev	-0.241

FIGURE 2.13: Sensitivity analysis of $\mathcal{R}_0$ of model (2.33) .FIGURE 2.14: Simulation of $\mathcal{R}_0$ of model (2.33) w.r.t different parameters .

take PM from smoking. To see how the model behaves when these measures are utilized in MATLAB using ode45 routine(RK4 method) with ICs $\{P(0) = 0.4, S(0) = 0.4, R(0) = 0.12, Q(0) = 0.06, D(0) = 0.02\}$, $h = 0.01$, $T = [0 \ 70]$ and all biological parameters given in Table 3.2 shown in Fig 2.15-3.9. From the graphics view, we concluded that all the positive solutions are in the feasible region. With the implementation of treatment of smokers ρ and media advertising of the daily no of smokers, the transmission suddenly decreases as people become aware more quickly of the bad effects of smoking, due to which the effective contact rate between smokers S and potential smokers P decays. The sudden increase in the potential smoker P curve is due to a couple of relapse phenomena. One of the main reasons to decrease the smokers' S curve is the accurate implementation of treatment to addicted smokers, due to which these individuals move to recovered smokers R , and the recovery rate increases. This indicates the strong impact of smoking treatment ρ , and smokers S become stable after 20 days. Due to the success rate of media coverage and treatment, the recovered smoker R increases and becomes stable after 20 days because the quitter rate increases and many recovered individuals move to the quitter class Q . The curve of quitter smokers Q increases initially because initially many potential smokers quit smoking due to self-awareness, and also occasional smokers move to this class. After some time, this class reached a peak and moved down to its stable point. Many smokers who are badly affected by smoking and suffer from the smoking disease can not be treated and die at a rate γ moves to the death class D . Due to this, the death class increases and is stable with time. The separate results of each compartment of model 2.33 are shown in Fig 3.9. We can easily check the clear behavior of each solution curve from this Fig. After all discussion we concluded that media coverage along with treatment become a superpower to overcome the smoking dynamics because in this way individuals can start following the PM from smokers and those who are affected from smoking get treatment on earlier biases due to these points the individual's behavior play an important role to die out the smoking dynamics.

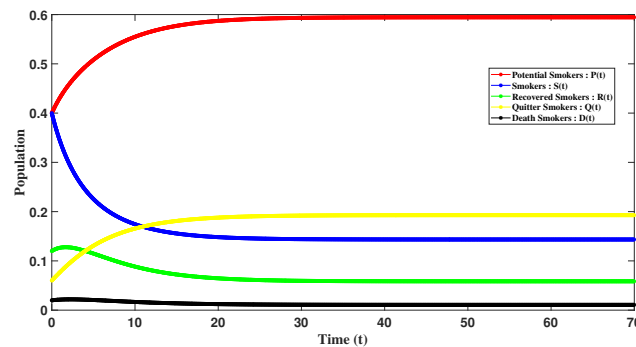


FIGURE 2.15: Numerical simulation of model (2.33) with daily media advertisement (m) and treatment of smokers (ρ) with parameters in Table 3.2, step size $h = 0.01$ and $T = [0 \ 70]$.

Impact of daily media advertisement of smokers and treatment

We have seen the impact of media during COVID-19 to raise awareness among the population aware of the critical situation and follow the PM implemented by the government authorities. Inspired by this thing, we collectively apply the media advertisement of the daily no of smokers $\beta_3 e^{-mS}$ and treatment of smokers ρ to control

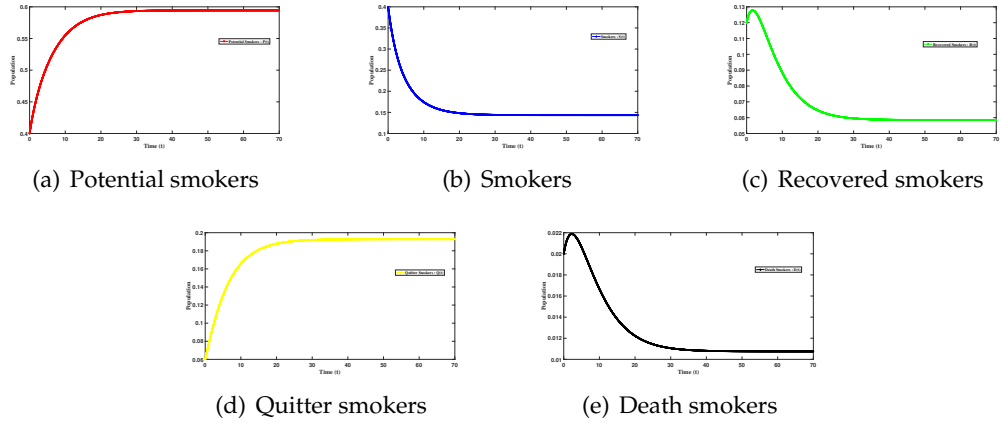


FIGURE 2.16: Numerical simulation of model (2.33) with daily media advertisement of smokers (m) and treatment of smokers (ρ) separately with parameters in Table 3.2, step size $h = 0.01$ and $T = [0 \ 70]$.

the impact of smoking on the population. If we see the function $\beta_3 e^{-mS}$, which is working as a bilinear incidence rate to give deep information about the transmission of smoking when other individuals are in effective contact with active smokers. As we already discussed, this is a decreasing function when you increase the value of m , which is the weight of strong media coverage. Initially, this function does not play any role in managing the smoking dynamics because $\beta_3 e^{-mS(0)}$ becomes zero. Due to this point, we have seen that $\beta_3 e^{-mS(0)}$ is not more effective in $\mathcal{R}_0$. However, in the model, this function plays a keen role in overcoming the smoking dynamics with time. Here we simulate the numerical results of the mathematical model to advertise daily news of addicted smokers through media and treatment to control smoking dynamics in MATLAB using ode45 routine (RK4 method) with ICs $\{P(0) = 0.4, S(0) = 0.4, R(0) = 0.12, Q(0) = 0.06, D(0) = 0.02\}$. In Fig 3.10, we deeply analyze the smoker S compartment of model (2.33) using media coverage and treatment of smokers. When we fixed the media coverage and checked the variation of treatment of smokers $\rho = \{0, 0.02, 0.05, 0.1, 0.17\}$, the smokers S compartment gradually decreased with the increase of treatment as shown in Fig 3.10(a). This is due to the success rate of treatment of smokers. When people recover from their addiction to smoking, then not too many people deny the PM to smokers. So a small amount moved to potential smokers when they recovered come in contact with these individuals. The curve of smoker S at $\rho = 0$ indicates that smoker individuals move slightly down and are stable after 15 days, and only a small number of individuals recovered. If we see the curve of smoker S at $\rho = 0.1$ describes that it suddenly moved down and reached the steady state after almost 20 days. Similarly, the curve of smoker S at $\rho = 0.17$ shows a high rate of recovery because the curve comes close to 0.1 population and is stable after 20 days, which shows the strong impact of treatment on smokers S . We also show the complete 3D behavior of treatment ρ while fixing media coverage strength m in Fig 3.10(b), which shows the smooth decrease in smokers with the increase of treatment when the dynamic started. Now we fixed the treatment of smoker ρ and vary the media coverage weighted m on smokers S in Fig 3.10(c). We fixed $\rho = 0.1$ and see that with the increase of m , the transmission of smoking decreases slightly from 0 to 0.15. But at $m = 2$, the transmission of smoking suddenly dropped, and the recovery rate was so high approached a stable state after 20 days. The 3D view of media coverage

variation is also seen in Fig 3.10(d). Our analysis indicates that the combined utilization of treatment and media coverage can play an important role in decreasing the persistence probability of smoking dynamics.

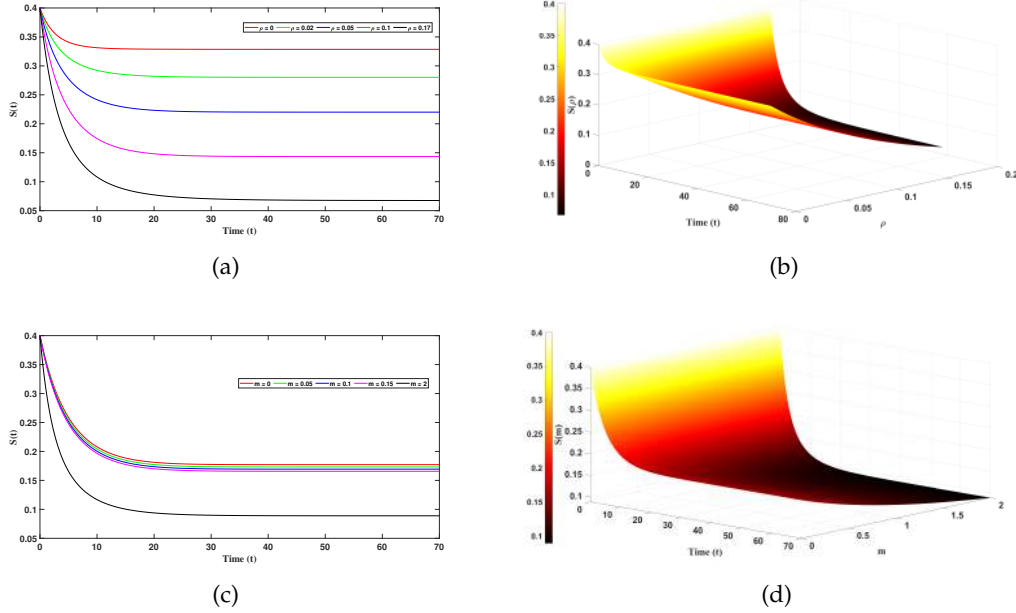


FIGURE 2.17: Numerical simulation of model (2.33) with variation of treatment of smokers (ρ) and daily media advertisement (m): (a) shows the variation of $\rho = \{0, 0.02, 0.05, 0.1, 0.17\}$ on smoker S while taking daily media advertisement of smokers (m) fixed, (c) shows the variation of $m = \{0, 0.5, 1, 1.5, 2\}$ on smoker S while taking treatment (ρ) fixed, (b) and (d) presents the 3D views by variation of $\rho = 0 : 0.001 : 0.17$ and $m = 0 : 0.01 : 2$ respectively.

As we already discussed, the bilinear incidence rate does not have a greater impact on initial smoking dynamics, but treatment can play an effective role in controlling the transmission of smoking. In Fig 3.11 we see the effectiveness of treatment of smokers ρ on $\mathcal{R}_0$ of model (2.33). When there is no treatment of smokers $\rho = 0$, then the value of $\mathcal{R}_0 = 25$ increases the treatment of smokers $\rho = 0.1$ shows value of $\mathcal{R}_0 = 15$ also when $\rho = 0.17$, then $\mathcal{R}_0 = 10$. This indicates that smoking transmission decreases when effective treatment is applied, and the natural death rate does not affect the behavior of $\mathcal{R}_0$. We summarize the discussion with the media coverage of smokers daily, and increasing the success rate of treatment, along with changing the behavior of smokers toward completing the treatment, can overcome the increasing rate of smoking addiction.

Impact of daily news on smoking-related diseases mortality

Smoking has been an active global issue for decades. Despite a lot of restrictions on purchasing, illegal growth of tobacco, and smuggling of smoking drugs, no country has complete control of this epidemic. Due to this worst nature, the smoking epidemic is a serious killer for people who are addicted to smoking. Different WHO reports, medical studies, and literature reviews show a huge amount of deaths occur due to SRD, which is also a big issue for the world. To understand this scenario in model (2.33), we study the death smokers D deeply and investigate how quickly

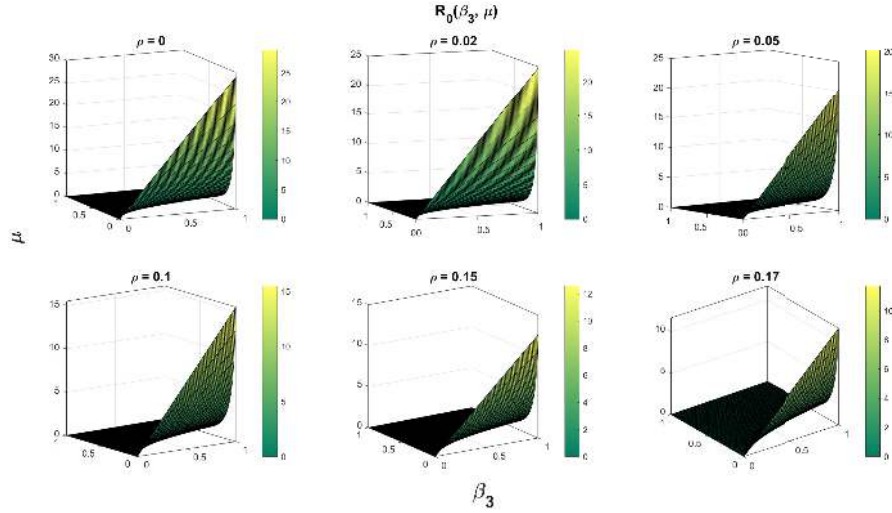


FIGURE 2.18: Simulation of $\mathcal{R}_0$ of model (2.33) w.r.t β_3 and μ with variation of treatment of smokers $\rho = \{0, 0.02, 0.05, 0.1, 0.17\}$.

the mortality rate due to smoking-related disease is affected when treatment and media coverage of the daily no of smokers is shown in Fig 2.19. By fixing the media coverage m and varying the treatment of smokers ρ to check the behavior of dead smokers D , we give the graphics analysis in Fig 2.19(a-b). When there is no treatment, $\rho = 0$, the death of smokers increases from the initial and reaches its maximum value, showing that the mortality rate due to smoking is high. When we apply the treatment $\rho = 0.02$ death of smokers starts increasing and after some move down, shows a slight decrease in cure. Similarly, when we increase the treatment from 0.02 to 0.17, the death smoker curve suddenly goes down, showing a quick downfall of the smoking dynamics and reaching their steady states after 40 days. This behavior of the dead smokers is deeply visualized in 2D view by Fig 2.19(b), which shows a smooth decrease in the death class with time when treatment is applied to their maximum strength. Also, fixing the treatment of smokers ρ and varying the media coverage parameter m , we see the solution of the death smokers compartment. We have seen that when media coverage gradually increases from 0 to 2, then the curve of deaths among smokers decreases and is stable after 30 days. This decrease is due to the fear created in the population against the smoking effects by the proper media coverage as seen in Fig 2.19(c). In this way, people change their behavior towards accepting the PM from smoking, and transmission of smoking decreases, resulting in deaths decrease. The 2D analysis is seen in Fig 2.19(d). To describe the combined effort on the death of smokers by media coverage and treatment of smokers, we give the graphics in Fig 2.19 (e). This combined impact shows the clear view of the implementation of treatment of smokers ρ and media coverage of daily smokers m . We have seen that strong and successful use of media to advertise public messages and proper treatment of smokers can easily decrease the deaths of smokers due to the sudden downfall of smoking-related mortality rates. Reproduction no $\mathcal{R}_0$ of model (2.33) is also a very good tool to express the smoking dynamics. In Fig 2.20 we see the variation of transmission of smoking on $\mathcal{R}_0$. The simulation indicates that with the increase in treatment of smokers, the effective contact of smokers with other populations decreases, due to which the smoking transmission also slows down and

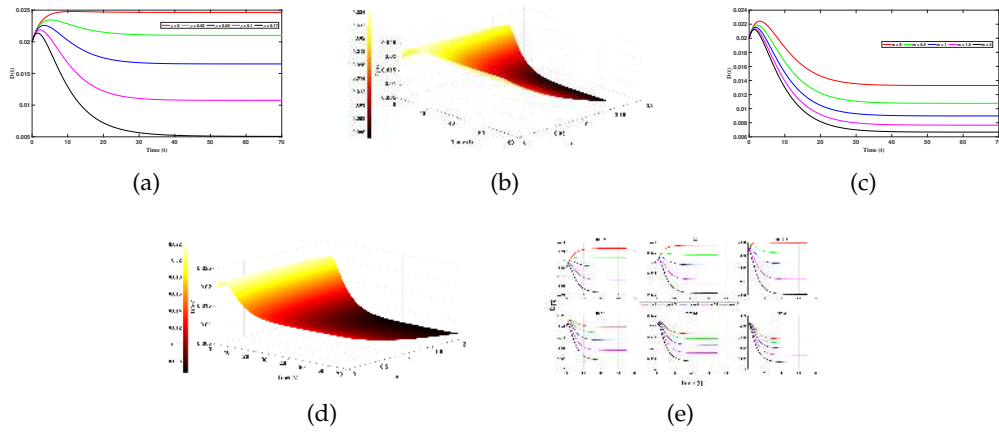


FIGURE 2.19: Numerical simulation of model (2.33) with variation of treatment of smokers (ρ) and daily media advertisement (m): (a) shows the variation of $\rho = \{0, 0.02, 0.05, 0.1, 0.17\}$ on death smoker D while taking daily media advertisement of smokers (m) fixed, (c) shows the variation of $m = \{0, 0.5, 1, 1.5, 2\}$ on death smoker D while taking treatment (ρ) fixed, (b) and (d) presents the 3D views by variation of $\rho = 0 : 0.001 : 0.17$ and $\rho = 0 : 0.01 : 2$ respectively. (e) is death compartments with variation of treatment of smokers $\rho = \{0, 0.02, 0.05, 0.1, 0.17\}$ and daily media advertisement of smokers $m = \{0, 0.5, 1, 1.5, 2\}$.

the smoking-related mortality rate decreases. When the treatment of smokers ρ increases from 0 to 0.17, the value of $\mathcal{R}_0$ decreases from 25 to 9, indicating the effectiveness of treatment to overcome the death due to smoking dynamics. The relation of smoking transmission β_3 and smoking-related mortality γ is also shown in Fig 2.21. The contour views predict that β_3 has more impact on $\mathcal{R}_0$ as compared to γ . But it concludes that when ρ increases, then $\mathcal{R}_0$ decreases and the mortality rate due to smoking decreases slowly with the decrease in transmission of smoking in the population.

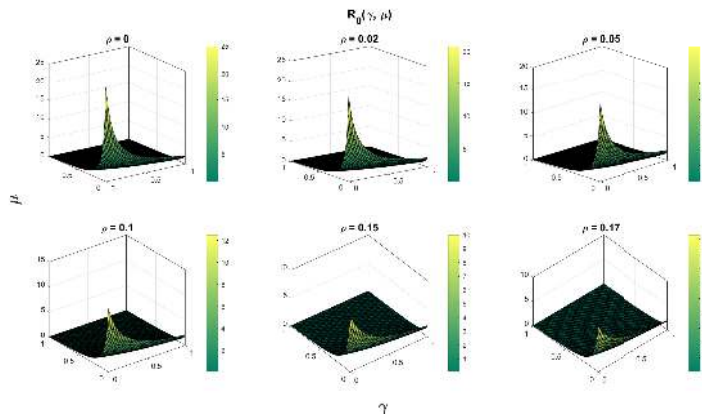


FIGURE 2.20: Numerical simulation of model (2.33) with variation of treatment of smokers (ρ) and daily media advertisement (m) on $\mathcal{R}_0(\gamma, \mu)$.

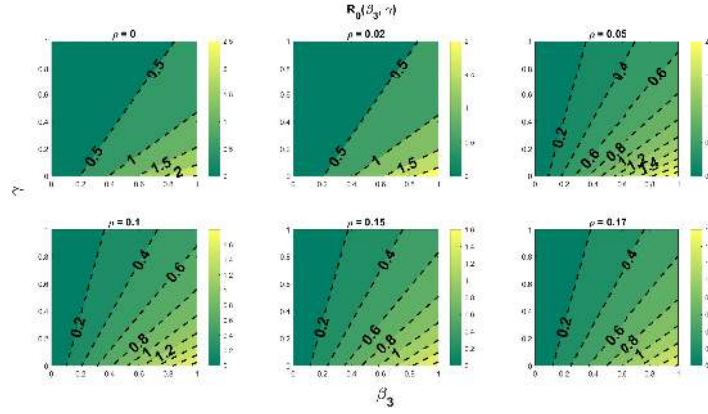


FIGURE 2.21: Numerical simulation of model (2.33) with variation of treatment of smokers (ρ) and daily media advertisement (m) on $\mathcal{R}_0(\beta_3, \gamma)$.

2.5 Mathematical model of combined impact of TV, Radio, Social media advertisement, and treatment to control smoking dynamics

Acquiring public attention to control techniques is a major difficulty for public health authorities, as it is frequently influenced by disinformation and pretended opinions. Oversight throughout the epidemic has been greatly aided by media coverage. Unfortunately, the dynamic and even unconfirmed nature of released data has made it difficult for reporters and scholars to provide the public with reliable information. A massive volume of information, both accurate and false, has been disseminated via social media sites like Facebook, Twitter, YouTube, etc, as a result of this issue. Considering these barriers, social media has mostly resulted in behavioral shifts that have increased the efficacy of federal attempts to stop the spread of the disease. As a result, by promoting the emergence of defensive tactics, media coverage has significantly influenced the dynamics of the disease and is crucial for controlling the spread.

To understand all these challenges, we formulate a deterministic mathematical model of smoking addiction through media advertisement that includes a media coverage parameter (M) for controlling and utilizing the restriction measures to prevent the population from smoking addiction, which takes into consideration the role of all types of media advertisements (Social media Apps, Television and Radio) in changing peoples behavior. A standard incident rate utilized to get information about the transmission of smoking by effective contact between potential and active smokers for this model is given as

$$f(t) = (1 - M)\beta \frac{\eta_1 R + \eta_2 Q + S}{N} P \quad (2.36)$$

Here M is the efficiency of media advertisement and coverage, which raises public awareness and slows the spread of smoking through $S_{social\ apps}$ (including Facebook, YouTube, Twitter(X), WhatsApp, Ticktock, Instagram, Snapchat, Telegram, etc), T_{tv} (all mainstream media like TV) and R_{radio} (Radio programs).

$$M = S_{social\ apps} + T_{tv} + R_{radio}$$

The decline in transmission resulting from the influence of media is represented via $1 - M$, along with β , which is the baseline transmission rate from other classes to active smokers. It shows how quickly smoking spreads from active to target smokers when there is no media coverage or preventative action taken. A greater value of β indicates a faster rate of disease transmission. The fraction $\frac{\eta_1 R + \eta_2 Q + S}{N} P$ is the percentage of contagious people in the population. Recovered smokers R . Despite not having direct contact with the general public, these persons may nevertheless be at risk of transmission due to family functions, friends' pre-pressure, unfavorable gatherings, etc. This is indicated by η_1 , the relative infectivity of Recovered smokers. Quitter smokers Q are those individuals who have completely stopped smoking and do not exhibit any symptoms; their relative infectivity as quitters is represented by η_2 . S are Active smokers who smoke often, as they are most prone to spreading the habit. By dividing the total population, N , you can make sure that the rate of new infections is in line with the size of the population. Potential smokers P is the total number of potential smokers who are at risk of smoking at time t . To check the variation of media impact, we check the detailed view of the function $f(t)$ as

- **No Media Impact $M = 0$**

When $M = 0$, then the function $f(t) = \beta \frac{\eta_1 R + \eta_2 Q + S}{N} P$. This suggests that smoking spreads at its fastest possible rate and that the media has no effect on public opinion. In this way, the general public has not received enough information about preventative measures or is uninformed about the risk factors of smoking. As a result, people carry on with their regular, pre-smoking operations (behaviors), which facilitates the transmission of smoking through contact between active and potential smokers. There are no measures to delay the transmission, as indicated by the fact that the value of β stays at its baseline. The worst-case scenario is reflected in this circumstance when unrestrained smoking transmission results from a lack of media-driven advertisement for awareness or education.

- **Partial Media Impact $0 < M < 1$**

When $0 < M < 1$, then the function $f(t) = (1 - M) \beta \frac{\eta_1 R + \eta_2 Q + S}{N} P$ shows the partial media impact due to which the transmission of smoking decreases at a rate $1 - M$. In this case, media coverage helps partially to increase public awareness through media advertisement and encourages changes in attitudes, including wearing masks, avoiding smoky crowds, smoking under pressure from family, friends, or community, and so on. People take preventative actions as a result of media efforts, which lowers their risk of contracting smokers. On the other hand, the efficiency of the media determines how much the transmission rate is reduced. When M approaches zero, there is less of an impact from the media, which results in fewer people adopting preventative measures, but transmission remains important. There is a noticeable decrease in transmission as M gets closer to 1. This indicates a more grounded situation where media awareness initiatives are operating, but not flawlessly. Because of these measures, smoking is still spreading, but it is doing so more slowly.

- **Complete Media Impact $M = 1$**

When $M = 1$ then function $f(t) = (1 - 1) \beta \frac{\eta_1 R + \eta_2 Q + S}{N} P = 0$. In this instance, media coverage is incredibly successful in encouraging the public to fully abide by preventative and health-related advice. Everybody follows the

advice given by the media. The smoking transmission rate drops to zero, indicating that the medium was successful in stopping the smoking spread altogether. This is the perfect example of how public outreach by the media has persuaded people to take preventative measures. In this case, the smoking dynamics would presumably come to a stop because smoking could no longer propagate.

In practical applications, M tends to be 1, as achieving perfect media efficacy is a challenging task. Several variables restrict the effect of the media, like Social media misinformation or deception, behavioral exhaustion, demographics, and public mistrust. Consequently, advertising efforts are essential, but they also need to be used in conjunction with additional measures. System of non-differential equations of the mathematical model of the combined impact of TV, Radio, Social media advertisement, and treatment to control smoking dynamics is given as

$$\begin{cases} \frac{dP}{dt} = \Lambda - (1-M)\beta \frac{\eta_1 R + \eta_2 Q + S}{N} P + \alpha(1-\epsilon)S - (\mu + \phi + \beta_2)P + \pi\theta R \\ \frac{dS}{dt} = (1-M)\beta \frac{\eta_1 R + \eta_2 Q + S}{N} P - (\mu + \alpha + \rho + \gamma)S \\ \frac{dR}{dt} = \alpha\epsilon S + \rho S - (\mu + \theta)R \\ \frac{dQ}{dt} = \beta_2 P + \phi P + (1-\pi)\theta R - \mu Q \\ \frac{dD}{dt} = \gamma S - \mu D \end{cases} \quad (2.37)$$

with the initial conditions

$$P(0) \geq 0, S(0) \geq 0, R(0) \geq 0, Q(0) \geq 0, D(0) \geq 0$$

2.5.1 Computation of steady states

The DFE of model (2.37) is given by

$$E_{q0} = (P^0, S^0, R^0, Q^0, D^0) = \left(\frac{\Lambda}{(\mu + \phi + \beta_2)}, 0, 0, \frac{\Lambda(\phi + \beta_2)}{(\mu + \phi + \beta_2)\mu}, 0 \right).$$

System (2.37) admits a unique positive endemic equilibrium $E_{q1} = (P^*, S^*, R^*, Q^*, D^*)$.

$$\begin{aligned} P^* &= \frac{\mu(\mu + \theta)(\mu + \alpha + \rho + \gamma)S^*}{(1-M)\beta \left(\eta_1 \frac{\alpha\epsilon + \rho}{\mu + \theta} S^* + \eta_2(\mu + \theta(\beta_2 + \phi) + \theta(1-\pi)(\alpha\epsilon + \rho) + S^*) \right)} \\ S^* &= \frac{\Lambda - \mu(\mu + \phi_2 + \beta_2)(\mu + \theta)(\mu + \alpha + \rho + \gamma)}{\left((\mu + \alpha + \rho + \gamma) - \alpha(1-\epsilon) - \pi\theta \frac{\alpha\epsilon + \rho}{\mu + \theta} \right) l_1} \\ R^* &= \frac{(\alpha\epsilon + \rho)S^*}{\mu + \theta} \\ Q^* &= \frac{(\beta_2 + \phi)(\mu + \theta)(\mu + \alpha + \rho + \gamma)S^* + (1-\pi)\theta(\alpha\epsilon + \rho)S^*}{(\mu + \theta)(1-M)\beta \left(\eta_1 \frac{\alpha\epsilon + \rho}{\mu + \theta} S^* + \eta_2(\mu + \theta(\beta_2 + \phi) + \theta(1-\pi)(\alpha\epsilon + \rho) + S^*) \right)} \\ D^* &= \frac{\gamma S^*}{\mu} \end{aligned} \quad (2.38)$$

where $l_1 = \left((1-M)\eta_1 \frac{\alpha\epsilon + \rho}{\mu + \theta} \beta + \eta_2(\mu + \theta)(\beta_2 + \phi) + \theta(1-\pi)(\alpha\epsilon + \rho) \right)$.

2.5.2 Threshold parameter R_0

To calculate the basic reproduction number of the model (2.37), we use the next-generation matrix approach shown in [130].

$$\mathbf{F} = \begin{pmatrix} (1-M)\beta\eta_1 RP + (1-M)\beta\eta_2 QP + (1-M)\beta SP \\ 0 \\ 0 \end{pmatrix}$$

$$\mathbf{V} = \begin{pmatrix} \mu + \alpha + \rho + \gamma S \\ -\alpha\epsilon S + \mu R - \rho S + \theta R \\ -\beta_2 P - (1-\pi)\theta R + \mu Q - \phi P \end{pmatrix}$$

The following information provides the Jacobi matrices of $\mathbf{F}$ and $\mathbf{V}$ at DFE, represented by $\mathcal{F}$ and $\mathcal{V}$:

$$\mathcal{F} = \begin{pmatrix} (1-M)\beta P^0 & (1-M)\beta\eta_1 P^0 & (1-M)\beta\eta_2 P^0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

$$\mathcal{V} = \begin{pmatrix} \mu + \alpha + \rho + \gamma & 0 & 0 \\ -\alpha\epsilon - \rho & \mu + \theta & 0 \\ 0 & -(1-\pi)\theta & \mu \end{pmatrix}$$

The definition of $\mathcal{R}_0$ in the mathematical context is the spectral radius of $\mathcal{F}\mathcal{V}^{-1}$. Thus, $\mathcal{R}_S$, $\mathcal{R}_R$, and $\mathcal{R}_Q$ are the secondary infection numbers that were sown by close interaction with smokers, removed, and quitter people, respectively.

$$\mathcal{R}_0 = \mathcal{R}_S + \mathcal{R}_R + \mathcal{R}_Q$$

$$\mathcal{R}_S = \frac{\Lambda\beta(1-M)}{(\beta_2 + \mu + \phi)(\alpha + \gamma + \mu + \rho)}$$

$$\mathcal{R}_R = \frac{\Lambda\beta\eta_1(1-M)(\rho + \alpha\epsilon)}{(\mu + \theta)(\beta_2 + \mu + \phi)(\alpha + \gamma + \mu + \rho)}$$

$$\mathcal{R}_Q = \frac{\Lambda\beta\eta_2\theta(1-M)(1-\pi)(\rho + \alpha\epsilon)}{\mu(\mu + \theta)(\beta_2 + \mu + \phi)(\alpha + \gamma + \mu + \rho)}$$

2.5.3 Sensitivity analysis

Sensitivity analysis helps to find significant parameters that affect the results of the model and may slow the smoking dynamics. To do this, the Latin Hypercube Sampling (LHS) approach is employed here to estimate the PRCC, which is a statistical tool. PRCC is used to assess the degree of correlation between the parameters and the model reproduction number. This quantifies the extent to which changes in the input parameters account for variations in the output variables while accounting for the impact of additional input parameter variations. PRCC values of the model (2.37) $\mathcal{R}_0$ are given in Fig 3.13. We use the $N = 2000$ sample for LHS and run over the timespan 0:1:700 in days. The sensitivity analysis of $\mathcal{R}_S$ are seen in Fig 3.13(a) which shows that Λ, β are the significant parameters in positive sense while M, α, γ, μ are significant parameters in negative sense. The graphics indicate that to control the transmission of smoking, we have to increase the ρ because this shows a low value as compared to other parameters. Fig 3.13(b) describe the PRCC analysis of $\mathcal{R}_R$ in which $\Lambda, \beta, \rho, \eta_1$, are the significant parameters in positive sense while M ,

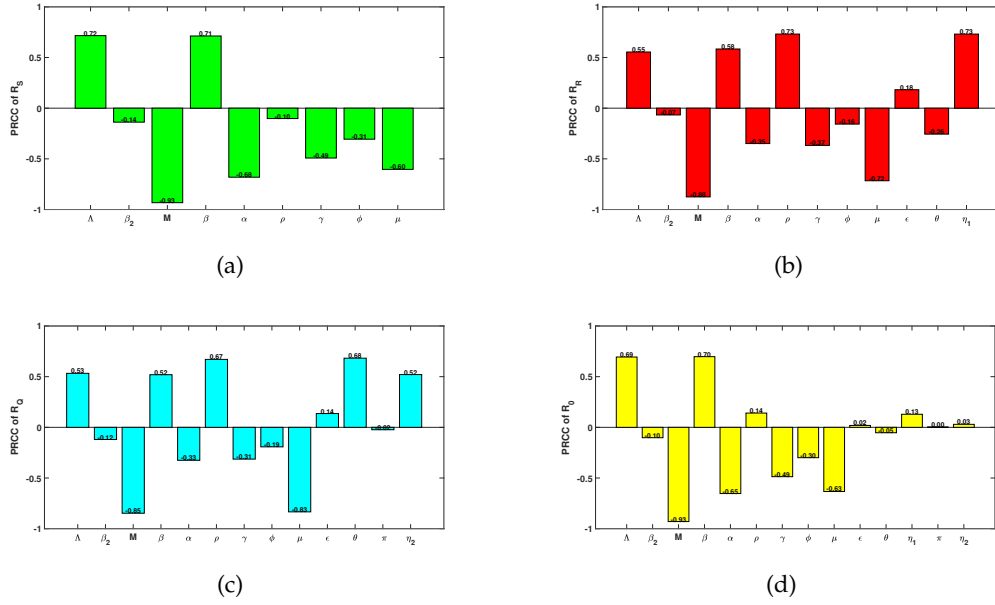


FIGURE 2.22: Sensitivity analysis of $\mathcal{R}_S$, $\mathcal{R}_R$, $\mathcal{R}_Q$ and $\mathcal{R}_0$ of model (2.37).

α , γ , μ are significant parameters in negative sense. The plot shows that to increase the recovery rate of smoking, we have to increase the ρ and control β , η_1 . $\mathcal{R}_Q$ shows the transmission of smoking in quitter smokers. The graphical representation in Fig 3.13(c) concluded that Λ , β , ρ , θ , η_2 , are the significant parameters in positive sense while M , μ are significant parameters in negative sense. So to overcome the smoking probability of recovered smokers becoming potential smokers and increase the quitting parameter, we have to decrease the β , η_2 . Similarly, the PRCC values of $\mathcal{R}_0$ shown in Fig 3.13(d) predicts that Λ , β are the significant parameters in positive sense while M , α , μ , γ are significant parameters in negative sense. This behavior shows that we have to manage β parameters while increasing ρ and decreasing γ , μ to overcome the smoking dynamics. The PRCC values of the receptive $\mathcal{R}_S$, $\mathcal{R}_R$, $\mathcal{R}_Q$ and $\mathcal{R}_0$ given in Table 3.6. To check the impact of some parameters on the $\mathcal{R}_0$ are shown in Fig 3.14. Variation of β smoking transmission and γ mortality rate due to smoking on $\mathcal{R}_0$ are given in Fig 3.14(a) shows that with the increase of β reproduction no also increase but in γ there is slight change indicate that γ is not more effective to change the behavior of $\mathcal{R}_0$. The combined impact of awareness of potential smoker ϕ and ϵ determination to quit smoking in Fig 3.14(b) presented that ϕ is more effective in varying the $\mathcal{R}_0$. Similarly, one can analyze the other graphics.

2.5.4 Numerical simulation of model of combined impact of TV, Radio, Social media advertisement and treatment to control smoking dynamics

In this section, we describe the numerical implementation of the combined Impact of TV, radio, social media advertisement, and treatment on smokers to overcome the smoking dynamics. To see how the model (2.37) behave with this combine Impact in MATLAB using ode45 routine(RK4 method) with ICs $\{P(0) = 0.4, S(0) = 0.4, R(0) = 0.12, Q(0) = 0.06, D(0) = 0.02\}$, $h = 0.01$, $T = [0 \ 70]$ and all biological

TABLE 2.4: Sensitivity index of $\mathcal{R}_S$, $\mathcal{R}_R$, $\mathcal{R}_Q$ and $\mathcal{R}_0$ of model (2.37)

Parameters	Sensitivity Index			
	$\mathcal{R}_S$	$\mathcal{R}_R$	$\mathcal{R}_Q$	$\mathcal{R}_0$
Λ	0.72	0.55	0.53	0.69
β_2	-0.14	-0.07	-0.12	-0.10
M	-0.93	-0.88	-0.85	-0.93
β	0.71	0.58	0.52	0.70
α	-0.68	-0.35	-0.33	-0.65
ρ	-0.10	0.73	0.67	0.14
γ	-0.49	-0.37	-0.31	-0.49
ϕ	-0.31	-0.16	-0.19	-0.30
μ	-0.60	-0.72	-0.83	-0.63
ϵ	-	0.18	0.14	0.02
θ	-	-0.26	0.68	-0.05
η_1	-	0.73	-0.02	0.13
π	-	-	0.52	0
η_2	-	-	-	0.03

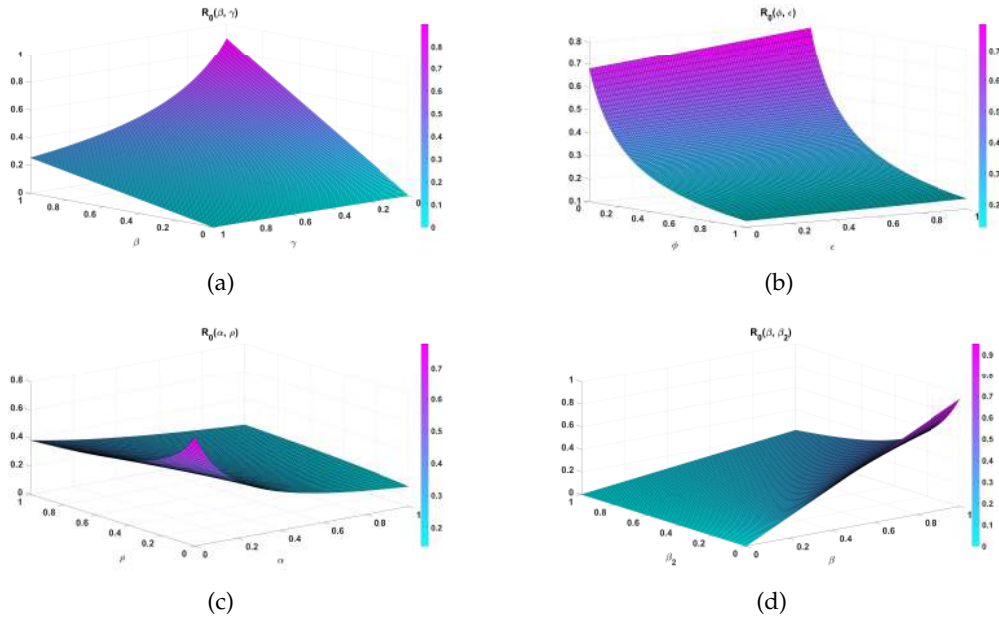


FIGURE 2.23: Variation of parameters on $\mathcal{R}_0$ of model (2.37).

parameters given in Table 3.2 shown in Fig 3.15-3.16. With the use of proper treatment with media awareness using TV, radio, and all social media apps, transmission of smoking decreases suddenly due to low effective contact between the smoker and other populations. This sudden decrease guarantees the successful treatment and effective media awareness programs, due to which the level of smoking dynamics becomes very low. With this effectiveness, the smokers moved to the recovered class, due to which we see the curve of recovered smokers approach its peak quickly and suddenly go to their steady states, which can be seen in Fig 3.16(c). An increase in recovery rate causes people to manage their behavioral factors and quit smoking. Due to this, the number of quitter smokers increases continuously. Similarly, in this way, the mortality rate due to smoking-related disease slows down suddenly. To show these clear behaviors, we made separate plots of model (2.37) in Fig 3.16.

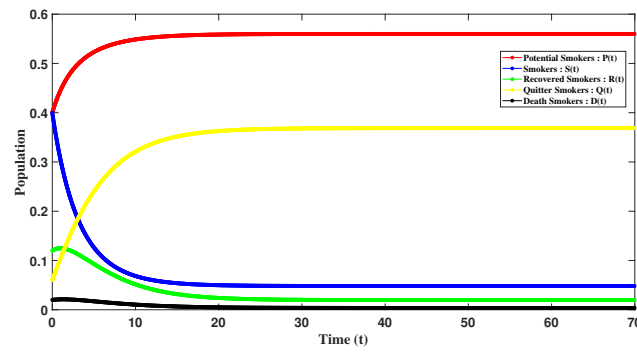


FIGURE 2.24: Numerical simulation of model (2.37) with media awareness program (M) and treatment of smokers (ρ) with parameters in Table 3.2, step size $h = 0.01$ and $T = [0 \ 70]$.

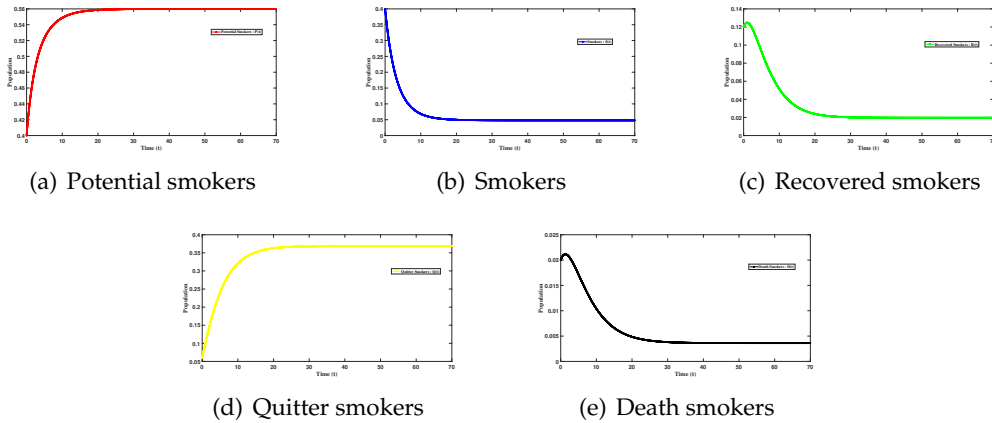


FIGURE 2.25: Numerical simulation of model (2.37) with media awareness program (M) and treatment of smokers (ρ) separately with parameters in Table 3.2, step size $h = 0.01$ and $T = [0 \ 70]$.

Efficiency of TV, Radio, and Social media advertisement with treatment on the model

Media awareness programs and camping have played an active role in any kind of natural disaster for decades. Now, the conman's use of social media to reach everyone has become an effective source of information and fast advertising of the

PM announcements by any government during any social epidemic. Treatment is also an important factor during such occasions, as initial persecution becomes an effective tool to slow down dynamics. Here, we are deeply studying the sudden impact of TV, radio, and social media advertisements for awareness programs and treatment for smokers who are badly affected by smoking-related diseases. We will investigate the smoking compartment S of model (2.37) that suddenly changes its behavior when we collectively apply the media awareness and treatment as persecution to overcome the smoking dynamics in the population. The implementation of TV, radio, and social media advertisement as a control factor on smokers S without using treatment is shown in Fig 2.26(a-b). When there is no media use, $M = 0$, the smoker's curve goes down slightly and stabilizes after 20 days, with the maximum amount of population addicted to smoking. With the initial use of media, $M = 0.25$ smoker class moves down as compared to earlier and gets their equilibrium state before 20 days. Similarly, when we increase the value of media awareness programs M from 0.25 to 0.75, the smoker class moved down suddenly and stable after 15 days showing the effectiveness of a media campaign to aware people about the bad effects of smoking due to which people start self prevention and government measure due to which the effective contact of smokers with other population decrease cause the sudden decrease in transmission of smoking. When we apply the full strength of media awareness, $M = 1$, we see how suddenly the smoker population goes down and approaches zero, showing the effectiveness of media use. A smooth decrease in smokers S is also seen in 3D Fig 2.26(b), which shows that media use in the correct way can become effective behavioral changes in the population. Similarly, in Fig 2.26(c-d), we utilize the treatment factor only as a percussion measure without the effective use of media as advertisements and awareness programs. We see that when there is no treatment, $\rho = 0$, the smoker population S is much higher, while taking treatment as an initial dose $\rho = 0.02$, the smoker's curve moves down as compared to earlier, showing the slight decrease in transmission of smoking in the other population. Following this thing, when we increase the treatment of smokers ρ from 0.02 to 0.1, we see that the smoker population decreases suddenly, and the curve at $\rho = 0.1$ indicates that the recovery rate increases rapidly, due to which a huge amount of smokers recovered, and the curve shows stable behavior after 15 days. With the full strength of treatment, $\rho = 0.17$, the smoker recovered more compared to previous data, indicating that proper treatment can be a more effective tool to overcome the smoking dynamics. The smooth behavior of this compartment is shown in 3D Fig 2.26 (d). We see that only the media use the distance between the curve of the smoker class for a different value of M , which is more, as compared to the implementation of treatment of smokers ρ , concluded that treatment works more quickly than media impact. Combine impact of TV, radio, and social media advertisement for awareness programs M and treatment to the smokers ρ presents in Fig 2.26 (e) at $M = \{0, 0.2, 0.4, 0.6, 0.8, 1\}$ and $\rho = \{0, 0.02, 0.05, 0.1, 0.14, 0.17\}$. If we compare the results at $M = 0$, $\rho = \{0, 0.02, 0.05, 0.1, 0.14, 0.17\}$ (variation of treatment and no media use) and $M = 1$, $\rho = \{0, 0.02, 0.05, 0.1, 0.14, 0.17\}$ (full strength of treatment and media use) show a clear difference in the smokers individuals. When $M = 0$, we see more distance in the smokers' curve at different values of ρ , showing that the smokers population decreases gradually. If we see the second case when $M = 1$ with full treatment of smokers, it shows the smoker population is zero, predicts the successful use of media with treatment to die out the smoking dynamics. Next, we see the variation of reproduction number due to effective parameters because $\mathcal{R}_0$ plays a key role in establishing a clear view of the probability of persistence of smoking dynamics in any population. Transmission of smoking β and

mortality rate μ variation on $\mathcal{R}_0$ shown in Fig 2.27. We check the combine impact at $M = \{0, 0.3, 0.7, 1\}$ and $\rho = \{0, 0.05, 0.1, 0.17\}$. If we see the diagonal subplots in the first case (no treatment and no media use) value of $\mathcal{R}_0$ is 0.02, in second case ($\rho = 0.05, M = 0.3$) value of $\mathcal{R}_0$ is 0.015, in third case ($\rho = 0.1, M = 0.7$) value of $\mathcal{R}_0$ is 4×10^{-3} and in fourth case (full strength of media use and treatment) value of $\mathcal{R}_0$ is 0. This shows how fast the reproduction number decreases to zero when we collectively implement the treatment of smokers and utilize the full power of TV, radio, social media advertisement, and camping. But one thing we note is that β has more impact on $\mathcal{R}_0$ as compared to μ . Variation of β and μ on $\mathcal{R}_S$ expressed in Fig 2.28. All cases of $\mathcal{R}_S$ gets the same values as $\mathcal{R}_0$ instead third case ($\rho = 0.1, M = 0.7$) where $\mathcal{R}_0$ is 6×10^{-3} shows a smooth decrease smoking dynamics. In Fig 2.29 the prediction of $\mathcal{R}_R$ shown w.r.t β and μ . The diagonal subplots in first case (no treatment and no media use) value of $\mathcal{R}_R$ is 8×10^{-7} , in second case ($\rho = 0.05, M = 0.3$) value of $\mathcal{R}_R$ is 2×10^{-5} , in third case ($\rho = 0.1, M = 0.7$) value of $\mathcal{R}_R$ is 1×10^{-5} and in fourth case (full strength of media use and treatment) value of $\mathcal{R}_R$ is 0 shows that the probability in the recovered smokers to become smokers decreases. Similarly, $\mathcal{R}_Q$ values are described in Fig 2.30. By viewing the diagonal subplots in the first case (no treatment and no media use) value of $\mathcal{R}_Q$ is 2×10^{-9} , in the second case ($\rho = 0.05, M = 0.3$) value of $\mathcal{R}_Q$ is 3×10^{-8} , in third case ($\rho = 0.1, M = 0.7$) value of $\mathcal{R}_Q$ is 2×10^{-8} and in fourth case (full strength of media use and treatment) value of $\mathcal{R}_Q$ is 0 indicates smoking scenario in quitter smoker. All the discussion about the prediction of $\mathcal{R}_S, \mathcal{R}_R, \mathcal{R}_Q$, and $\mathcal{R}_0$ describes that β is more effective than μ , showing a gradual decrease of smoking transmission in smokers, recovered, quitters, and the whole population. All this simulation concluded that media awareness programs and treatment of smokers by different therapies collectively overcome the smoking dynamics with time.

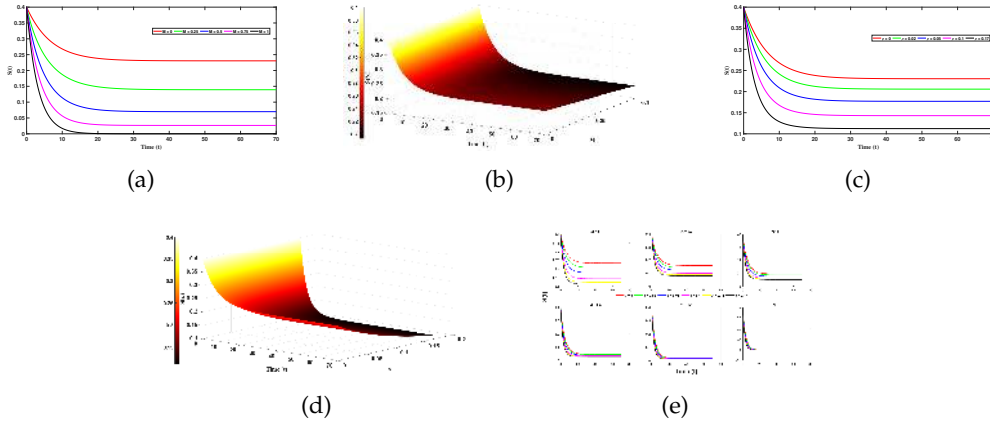


FIGURE 2.26: Numerical simulation of model (2.37) with variation of treatment of smokers (ρ) and TV, radio, social media advertisement (M): (a) shows the variation of $\rho = \{0, 0.02, 0.05, 0.1, 0.17\}$ on smoker S while no TV, radio, social media advertisement (M), (c) shows the variation of $M = \{0, 0.25, 0.5, 0.75, 1\}$ on smoker S while no treatment (ρ) of smokers, (b) and (d) presents the 3D views by variation of $\rho = 0 : 0.001 : 0.17$ and $M = 0 : 0.001 : 1$ respectively. (e) smoker compartments with variation of treatment of smokers (ρ) and TV, radio, social media advertisement (M) at $M = \{0, 0.2, 0.4, 0.6, 0.8, 1\}$ and $\rho = \{0, 0.02, 0.05, 0.1, 0.14, 0.17\}$.

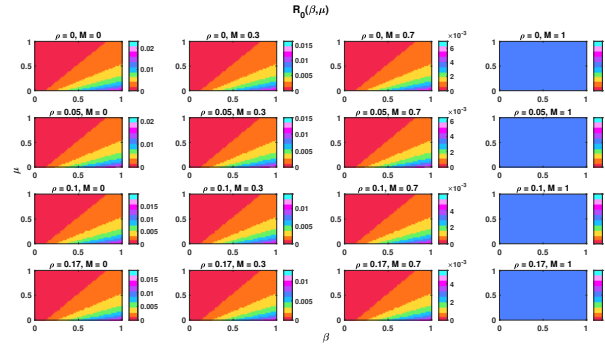


FIGURE 2.27: Simulation of $\mathcal{R}_0$ of model (2.37) w.r.t β and μ with variation of treatment of smokers $\rho = \{0, 0.05, 0.1, 0.17\}$ and TV, radio, social media advertisement $M = \{0, 0.3, 0.7, 1\}$.

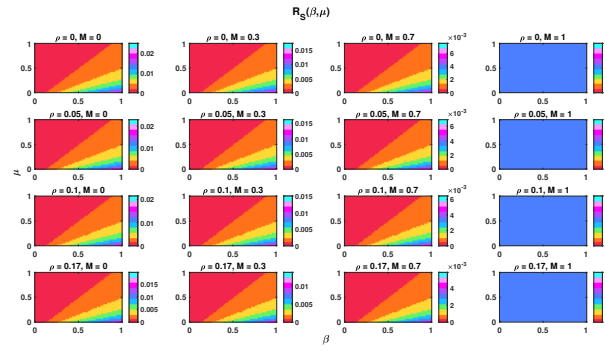


FIGURE 2.28: Simulation of $\mathcal{R}_S$ of model (2.37) w.r.t β and μ with variation of treatment of smokers $\rho = \{0, 0.05, 0.1, 0.17\}$ and TV, radio, social media advertisement $M = \{0, 0.3, 0.7, 1\}$.

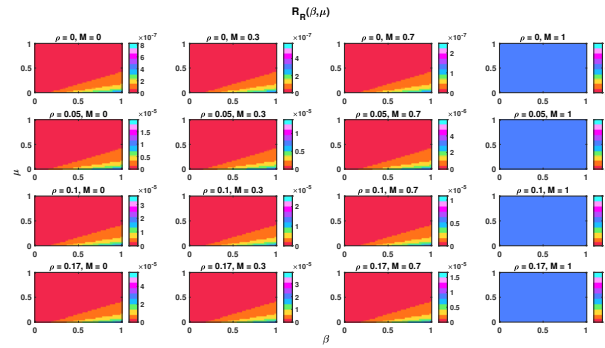


FIGURE 2.29: Simulation of $\mathcal{R}_R$ of model (2.37) w.r.t β and μ with variation of treatment of smokers $\rho = \{0, 0.05, 0.1, 0.17\}$ and TV, radio, social media advertisement $M = \{0, 0.3, 0.7, 1\}$.

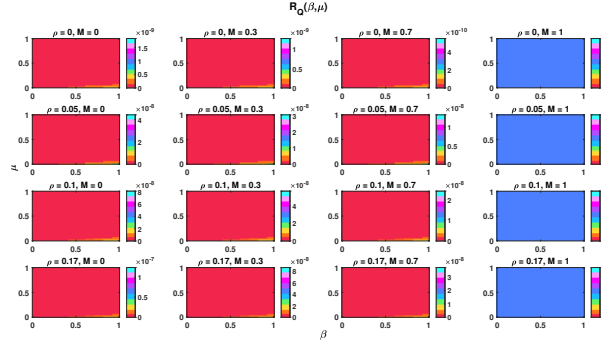


FIGURE 2.30: Simulation of $\mathcal{R}_Q$, of model (2.37) w.r.t β and μ with variation of treatment of smokers $\rho = \{0, 0.05, 0.1, 0.17\}$ and TV, radio, social media advertisement $M = \{0, 0.3, 0.7, 1\}$.

Efficiency of TV, Radio, and Social media advertisement with treatment on smoking-related deaths

Deaths due to SRD are a serious health issue in this age. Different research studies, literature reviews, and WHO data also give an overview of the total number of deaths per year. Numerous scholars have studied this issue using the model of smoking dynamics. But we are here to study this issue deeply and try to simulate some numerical results to control the death rate due to smoking-related disease with the utilization of media awareness and treatment of smokers to manage the transmission of smoking, which can be a good tool to slow the mortality rate due to smoking-related disease. In this section, we present the death compartment D of model (2.37) to analyze how effective media use and proper treatment can heal this serious health concern. We use the TV, radio, and social media advertisements for the awareness programs and camping without applying the treatment on smokers in Fig 2.31(a) shows that when no media use $M = 0$, the number of deaths due to smoking-related disease increases initially and after reaching a peak moves down and stabilizes after 35 days. When we start using all media $M = 0.25$, we see a small downfall in the death smoker D curve, and it stabilizes after 30 days. To keep this behavior in mind, we increase M from 0.25 to 0.75 and experience a sudden decrease in the death smoker population, which is stable before 30 days. This sudden decrease is due to the lack of transmission of smoking, which happens due to the success rate of media campaigns, due to which people properly utilize the PM and avoid participating in smoking parties, special gatherings, etc. Similarly, if we use the full strength of TV, radio, and social media advertisement for the awareness programs and campaign $M = 1$, there is a quick downfall in the death of the smoker population, approaching zero before 30 days. This behavior can be experienced in 3D (see Fig 2.31(b)), predicting that media campaigns through TV, radio, and social media advertisement can play a critical role in overcoming the mortality rate due to smoking-related disease. In Fig 2.31(c), we use only the treatment as initial persecution for the smokers who are badly addicted to smoking and do not implement media awareness programs. When there is no treatment, $\rho = 0$, deaths of smokers initially start increasing and reach a stable state after 35 days, showing a huge amount of individual deaths. When we initially start using the treatment of smokers $\rho = 0.02$, the death rate decreases as compared to earlier. By increasing the ρ value from 0.02 to 0.1, we see a smooth downfall in the number of deaths due to SRD, and the death smokers curve at $\rho = 0.1$ reaches its stable state after

30 days. This smooth decrease in the death rate due to smoking shows the high success rate of treatment given to smokers who are affected by smoking-related diseases and recover after completing treatment. Due to this reason, the death rate from smoking-related diseases decreases instantly. When we use the full strength of treatment $\rho = 0.17$, then we see how suddenly the death of smokers curve almost approaches zero (but not exactly zero). Also, the 3D view of treatment implementation is shown in Fig 2.31(d). One thing we note is that during the treatment process, the death smoker curve approaches its stable states as compared to media use. The combined impact of TV, radio, and social media advertisement for the awareness programs and campaigning with the use of treatment on smokers shown in Fig 2.31 (e) at $M = \{0, 0.2, 0.4, 0.6, 0.8, 1\}$ and $\rho = \{0, 0.02, 0.05, 0.1, 0.14, 0.17\}$. Numerical simulation at $M = 0$, $\rho = \{0, 0.02, 0.05, 0.1, 0.14, 0.17\}$ (variation of treatment and no media use) and $M = 1$, $\rho = \{0, 0.02, 0.05, 0.1, 0.14, 0.17\}$ (full strength of treatment and media use) show a clear difference in the deaths of smokers due to smoking diseases. $M = 0$ shows more distance in the death smokers curves at different values of ρ with a gradual decrease in death rate. when $M = 1$ with full treatment of smokers, it shows the smoker population is zero, and predicts the successful use of media with treatment to eliminate the smoking death issue. Next, we examine the variation of $\mathcal{R}_0$ due to effective parameters because $\mathcal{R}_0$ effectively describes the probability of persistence of death due to smoking dynamics. Death rate of smokers due to smoking-related disease γ and natural mortality rate μ variation on $\mathcal{R}_0$ shown in Fig 2.32. We check the combine impact at $M = \{0, 0.3, 0.7, 1\}$ and $\rho = \{0, 0.05, 0.1, 0.17\}$. Diagonal subplots when no treatment and no media use, the value of $\mathcal{R}_0$ is 12×10^{-3} , When $\rho = 0.05$, $M = 0.3$ value of $\mathcal{R}_0$ is 8×10^{-3} , keep going when $\rho = 0.1$, $M = 0.7$ value of $\mathcal{R}_0$ is 3×10^{-3} . Similarly, with the full strength of media use and treatment of smokers, the value of $\mathcal{R}_0$ is 0. This shows how fast deaths due to smoking dynamics vanish when we collectively implement the treatment of smokers and utilize the full power of TV, radio, social media advertisements, and campaigning. But one thing we note is that γ has more impact on $\mathcal{R}_0$ as compared to μ . One can also examine the variation of γ and β on $\mathcal{R}_0$ in Fig 2.33 to evaluate that decrease in transmission of smoking β also causes a sudden decrease in the death rate due to smoking γ . We concluded the discussion with these sentences that numerical results accurately show that proper use of all media forms and treatment for badly affected smokers can become a novel thing to reduce the growing rate of deaths due to smoking-related diseases.

2.6 Comparison of models

The worst global health issue of this age is the smoking dynamics, which are the routines and actions that are related to persistent inhaling of tobacco and cigarettes. These dynamics encompass beginning, growth, severity, duration, quitting, and relapse patterns of smoking. Youths often start smoking due to influences including peer pressure, social surroundings, and anxiety alleviation. Once developed, smoking seems to occur in a predictable pattern, with nicotine addiction being crucial in upkeep consumption. Adaptation and dependence may cause a gradual rise in smoking patterns and severity. Different people attempt different things to stop using tobacco and cigarettes. Some are successful in the chase, but others relapse due to societal pressures, cognitive causes, and physical desires. Comprehending smoking behaviors is crucial for devising efficacious initiatives and prevention strategies aimed at curbing smoking and enhancing chronic health.

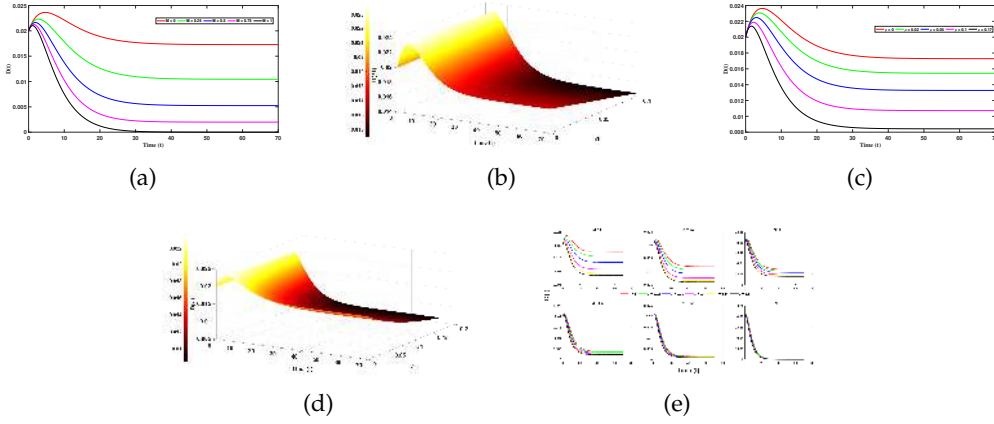


FIGURE 2.31: Numerical simulation of model (2.37) with variation of treatment of smokers (ρ) and TV, radio, social media advertisement (M): **(a)** shows the variation of $\rho = \{0, 0.02, 0.05, 0.1, 0.17\}$ on smoker S while no TV, radio, social media advertisement (M), **(c)** shows the variation of $M = \{0, 0.25, 0.5, 0.75, 1\}$ on smoker S while no treatment (ρ) of smokers, **(b)** and **(d)** presents the 3D views by variation of $\rho = 0 : 0.001 : 0.17$ and $M = 0 : 0.001 : 1$ respectively. **(e)** smoker compartments with variation of treatment of smokers (ρ) and TV, radio, social media advertisement (M) at $M = \{0, 0.2, 0.4, 0.6, 0.8, 1\}$ and $\rho = \{0, 0.02, 0.05, 0.1, 0.14, 0.17\}$.

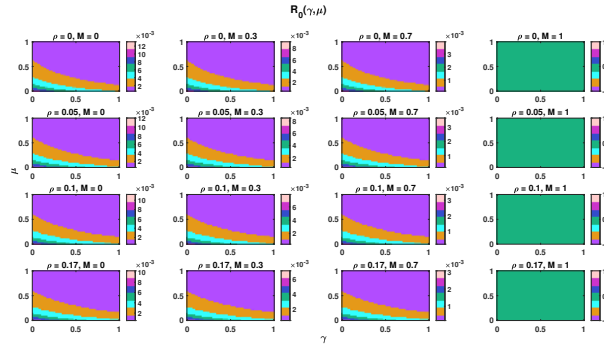


FIGURE 2.32: Simulation of $\mathcal{R}_0$ of model (2.37): $\mathcal{R}_0$ of model w.r.t γ and μ with variation of treatment of smokers $\rho = \{0, 0.05, 0.1, 0.17\}$ and TV, radio, social media advertisement $M = \{0, 0.3, 0.7, 1\}$.

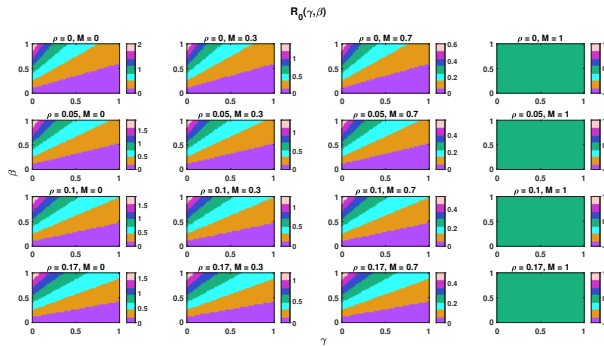


FIGURE 2.33: Simulation of $\mathcal{R}_0$ of model (2.37): $\mathcal{R}_0$ of model w.r.t β and γ with variation of treatment of smokers $\rho = \{0, 0.05, 0.1, 0.17\}$ and TV, radio, social media advertisement $M = \{0, 0.3, 0.7, 1\}$.

Motivated by these concerns about smoking dynamics, in this manuscript, we try to formulate multiple deterministic models to control the smoking dynamics, and deaths due to SRD by utilizing clinical therapy as treatment of smokers to manage their smoking behavior along with the proper treatment of individuals who are badly affected by smoking related serious disease like heart problems, cancers, stomach issue to minimize the mortality rate due to smoking and getting benefits by the expertise of media which is fastly growing, quick and powerful tool of this age to share information all over the world to change the behavior of the people about the PM applied through government policymakers by media awareness programs, advertisements, and camping in such way people start following them. The total population is divided into potential smokers, recovered, quitters, and death smokers.

In the first model (5.1), we developed a simple smoking model to study the smoking dynamics with various compartments, including the dead smoker, which is of great interest to us. Later on, we try to minimize the number of smokers by applying the treatment of smokers as a control factor and to slow down the mortality rate due to smoking-related deaths. This model's goal is only to check the impact of proper treatment during the smoking dynamics. The second model (2.33) is about the utilization of media by advertising daily news of addicted smokers as a bilinear incidence rate. This thing makes changes in people's opinion about the smoking dynamics due to daily news, which will produce fear in people about the bad effects of smoking, and people start following some self-care. We also continue to implement treatment for smokers. In this way, we study the combined impact of overcoming the smoking dynamics. The goal of this model is to computationally analyze how much daily advertising of several smokers and treatment can change people's thinking about this global issue. Our last model (2.37) predicts the effectiveness of proper and efficient use of TV, radio, and all social media platforms (SMP) for advertisement and live streaming the awareness programs about the PM to get rid of smoking and also implementing successfully treatment to smokers who are addicted of smoking and try to complete the clinical trial of the smoker who is badly affected by SRD. The motivation behind this model is to examine how efficiently media, most importantly social media, can become an effective tool to overcome the transmission of smoking in the population, along with the continuous treatment of smokers.

A comparative view of the smoker compartment of all models is shown in Fig 2.34. Model (5.1) graphical representation indicates that when the treatment of smokers ρ increases, then smokers S gradually decrease with time seen in Fig 2.34(a). Actually, when we try to replace the nicotine with gum patches, etc, which is a kind of drug used in cigarettes and tobacco that helps smokers reduce detox symptoms and appetites. Through the integration of behavioral therapies, medication support, and psychological counseling, these treatments provide a nurturing atmosphere that promotes cessation. Due to this reason, smokers S individuals control the use of tobacco and cigarettes. Individuals who have recovered from the treatment move to the recovered compartment, and their interaction with potential smokers P decreases, which becomes a stop to the increase in smoking transmission. This comprehensive plan not only assists current smokers in trimming back on cigarettes but also provides the groundwork for future quitting and enhanced wellness.

Computation of model (2.33) smoker visualize in Fig 2.34(b) with the variation of treatment parameter ρ and the weighted shared information about addicted smokers m . When there is no media use, $m = 0$, smokers S decrease gradually with increasing the treatment. When we increase the weight of shared information about addicted smokers to 50%, the change in color from dark to light blue happens earlier and

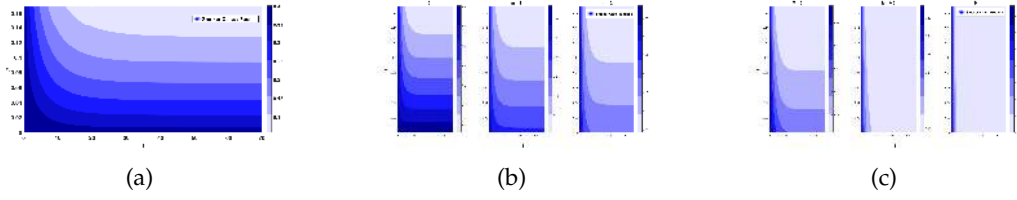


FIGURE 2.34: Numerical simulation of model (5.1), (2.33), (2.37) smoker compartment respectively with variation of treatment of smokers (ρ), TV, radio, social media advertisement (M) and daily media advertisement of number of smokers (m).

with more gradient, showing how quickly the smoker decreases. Similarly, when the weight of shared information about addicted smokers is 100%, transmission of smoking sharply decreases as compared to previous data. To check the complete impact of TV, radio, and social media advertisements for awareness programs and treatment of smokers in a model (2.37) seen in Fig 2.34(c). A clear view indicates that when there is no TV, radio, or social media advertisement, $M = 0$, then we see a sudden decrease in smokers. If we increase TV, radio, social media advertisement M to 50%, then no slope of strip seen indicates a small population of smokers, while full strength of TV, radio, social media advertisement M 100% shows no smoker means the smoking dynamics die out. When we use full media coverage by live stream, radio, and all types of SMP then people become more informative and motivated to adopt the PM by the health care departments and self prevention strategies including establishing a definite date to stop smoking, become motivated, look for social support, stay away from smoking activates, take NRT, get moving, and Keep your hands and mouth engaged. Drink plenty of water, and eat well. Keep in mind the health advantages, select a pastime or diversion, and get ready for removal indicators of smoking. Social media, radio, and television are effective means of advertising quit programs, raising awareness, and providing support to people who want to stop smoking. Massive advertisements for public health are offered via TV and radio, but social media shines at enticing consumers with tailored content, social networks, and interactive components. Together, these platforms spread the word about the dangers of smoking, give people access to goods, and inspire people with personal stories, professional guidance, and focused outreach. As such, they are essential for supporting smokers in their efforts to stop and avoid relapsing.

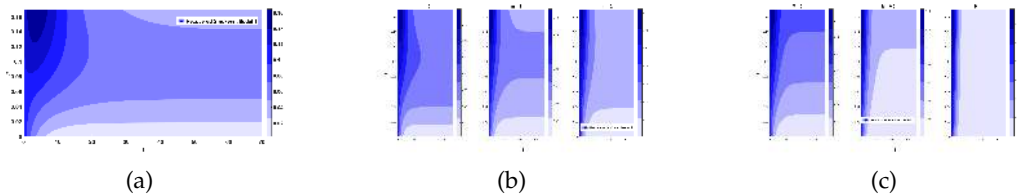


FIGURE 2.35: Numerical simulation of model (5.1), (2.33), (2.37) recovered compartment respectively with variation of treatment of smokers (ρ), TV, radio, social media advertisement (M) and daily media advertisement of number of smokers (m).

Relapse is a crucial component of the dynamics of smoking and is frequently brought on by external factors, mental disorders, and physiological cravings. To guarantee that smokers who have recovered or who have stopped will continue to

be smoke-free, effective programs must include ongoing assistance, means of coping, and avoiding relapse. In all models, we observe the relapse in recovered R and quitter smokers Q due to which these individuals do not show long-term behavior of quitting smoking activities and again become potential smokers, as seen in Fig 2.35-2.36. When we only apply the treatment to smokers, then the recovery rate increases, due to which the recovered class gradually increases (see Fig 2.35(a)). When we increase the weight of information shared by media from 0, 50%, and 100%, respectively, recovered smokers suddenly increase and go to an equilibrium state. Similarly, without TV, radio, all social media platform advertisement $M = 0$ and treatment variate then we see a gradual increase in recovered smokers, when TV, radio, all social media platform advertisement M is 50% then recovered smoker suddenly show equilibrium state after increasing, also increasing all social media platform advertisement M is 100% all smoker within no days finished. One thing that we note is that recovered smokers suddenly move to an equilibrium state, which is characterized by relapse phenomena by which recovered smokers again become potential smokers. However, the ratio of relapse in quitter smokers is much less than compared of recovered smokers because there are some occasions smokers and other self-aware people who initially moved to the quitter class can be examined in Fig 2.36. Some of the reasons for relapse in smoking dynamics are given:

- **Appetites for Nicotine:** Addictive properties of nicotine cause cravings that last long after quitting and, if left unchecked, can result in relapse.
- **Mental Triggers:** Since smoking may have been a coping strategy, anxiety, strain, or emotional turmoil are frequently identified as triggers for recurrence.
- **Behavioral Trends:** The impulse to smoke might resurface when engaging in regular or typical behaviors linked to smoking, such as socializing or consuming coffee.
- **Environmental Calls:** Both recovered, and quitter smokers are more likely to relapse when they are in frequent smoking situations involving other smokers.

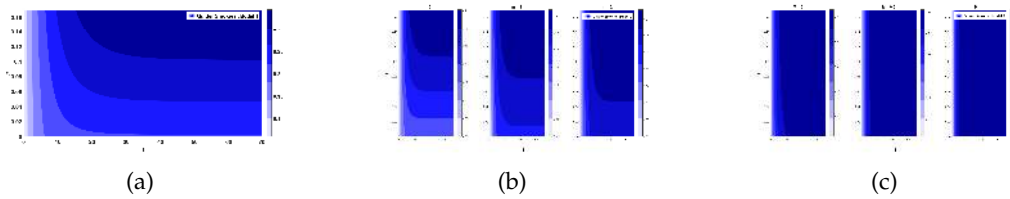


FIGURE 2.36: Numerical simulation of model (5.1), (2.33), (2.37) quitter compartment respectively with variation of treatment of smokers (ρ), TV, radio, social media advertisement (M) and daily media advertisement of number of smokers (m).

Now we discuss the mortality rate due to SRD in all models. One thing is clear from Fig 2.37 that the mortality rate due to smoking disease decreases gradually, but in model (2.37), media and treatment show more effectiveness and eliminate the death dynamics due to smoking diseases. We conclude our discussion with results that combine the impact of TV, radio, social media advertisement, and treatment to control smoking dynamics (model (2.37)) results are more effective to guarantee in utilizing all mainstream and SMP for effective media campaign by some know slogans 'Choose Life Not Smoke', 'Smoking Cause Cancer', 'Quit Smoking, Save

Family', 'Smoking killing you Every Day'. Make a social network on Facebook, Instagram, X, etc, to volunteer for work and provide people with effective information about smoking dynamics. All the results may vary by different biological parameters taken by any individual.

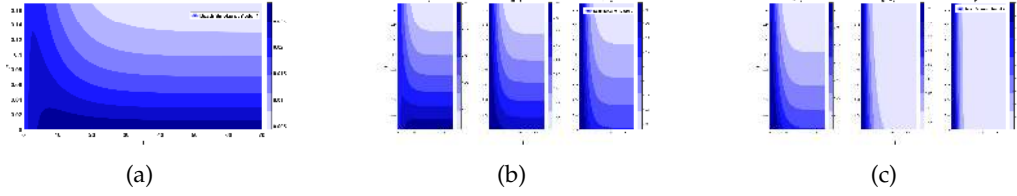


FIGURE 2.37: Numerical simulation of model (5.1), (2.33), (2.37) death compartment respectively with variation of treatment of smokers (ρ), TV, radio, social media advertisement (M) and daily media advertisement of number of smokers (m).

2.6.1 Future work

In this manuscript, we developed deterministic models for the prediction of smoking dynamics. In smoking behavior modeling, addiction may rely on smoking history as well as current nicotine level, which we can not examine without fractional derivatives. Fractional derivative modeling is better at capturing than traditional models. So we are working on the implementation of the Caputo fractional derivative to study more deeply memory effects in these models. We will establish some known theorems and computational results for their fractional order models using by Caputo fractional derivative. A real data set can be used to validate the model results.

2.7 Conclusion

The present manuscript describes the development and analysis of deterministic compartmental mathematical models designed to address the worldwide smoking dynamics. We began our modeling process by investigating the biological features of diseases attributed to smoking, which gave us an overview of how to handle the modeling task. The first model came into account only the implementation of treatment of smokers, the second formulated the impact of daily basis media coverage of smokers along with proper treatment of smokers and the last one is about the combined impact of TV, Radio, and SMP advertisement, campaigns and awareness programs coupled with treatment to addicted smokers are the system of nonlinear differential equations consists of five compartment including Potential smokers P , Smoker S , Recovered smokers R , Quitter smokers Q , and Death smokers D . We confirmed the well-posedness of the models and estimated the threshold parameter that drives the smoking dynamics. Several of the outcomes drawn from our numerical and qualitative analyses are emphasized:

- The equilibrium points with no smoking are both locally and globally asymptotically stable when the reproduction number is less than 1. This implies that if each smoker fails to initiate secondary smoking, smoking may be completely

eradicated from the community. Smoking will eventually become extinct in society over time as the requirement is fulfilled. Equilibrium points with smoking are also both locally and globally asymptotically stable when the reproduction number is greater than 1, which indicates that if each smoker produced secondary smokers, then smoking dynamics persist in the community over time.

- All solutions remain positive in the feasible region by positivity and boundedness. Under the specific condition, a unique solution of the model exists, which shows the evaluation of smokers over time by application of uniqueness and existence theorems.
- Sensitivity analysis of all model reproduction numbers shows the significance parameters indicating that increasing the transmission of smoking and mortality rate due to SRD increases the reproduction number, leading to more prevalence of smoking dynamics in the population. The reproduction number of model (5.1) shows that to die out smoking we must increase the treatment parameter ρ to decrease the transmission of smoking β_1 and increase the recovery rate of smokers α . Following this way, model (2.33) predicts the same behavior to overcome the smoking dynamics. Similarly model (2.37) Increasing the media coverage M and treatment ρ can control the baseline transmission of smoking and infection probability in recovered and quitter smokers, guarantee the die-out of smoking dynamics.
- In model (5.1) without treatment, smokers' evaluation over time does not show any fluctuation over time. However, with the utilization of treatment for smokers, we observe a sudden decrease in the evaluation of smokers during the smoking dynamics. This process continues for the long term, leading to a decrease in the spread of smoking dynamics in the population.
- In model (2.33) with the effective media coverage of daily smokers and proper treatment to smokers, ρ can decrease the transmission of smoking in the population. Due to the increase of media coverage m from 0, 50%, and 100%, people start to follow the information about this dynamics and increase self PM and increase the success of clinical trials increase the recovery rate along with the decrease of mortality rate due to smoking disease leading to dying out the smoking dynamics from a population with 100% strength of ρ & m over time.
- In model (2.37) with TV, Radio, Social Media Advertisement, campaign, trending slogan, making social networking and effective treatment to smokers ρ suddenly decrease the transmission of smoking in the population as compared to previous models. Due to the increase of TV, Radio, Social Media Advertisements, campaigns, trending slogans, M from 0, 25%, 50%, 75% and 100% people have started to change their behavior and attitudes toward this worst smoking dynamics and strictly follow the persecution due to quickly reach of information about this dynamics through social media use along an increase in the success rate of treatment to addicted smokers increase recovery rate along with the decrease of mortality rate due to smoking disease leading to dying out the smoking dynamics from the population over time. Utilizing the 100% of M and ρ vanished the smoker class, due to which the reproduction number is zero, proving the die out of smoking dynamics. But, identically, it's impossible due to biological, physiological, and demographic challenges. So the effective results of smoker evaluation over time are between $50\% < M < 70\%$.

- In this modeling, our main goal is to check the variation of death due to smoking-related diseases over time. From the manuscript data, we came to the point that efficient and helpful information with proper treatment effectively decreases this issue. model (2.37) show this behavior in excellent way.

Drawing on the outcomes of our investigation, we suggest stepping up media awareness campaigns to combat the propagation and transmission of smoking. It is important to use all available media platforms (including newspapers, Radio, television, posters, pamphlets, and public events) to spread education about smoking and its negative consequences to eradicate smoking among individuals. The authors have not simulated the population dynamics of smoking in this work using actual data. On the other hand, data-driven analysis plays a crucial role in forecasting the complexity of smoking dynamics.

Chapter 3

Behavioral dynamics focusing on the interplay of social media addiction and depression with memory effect

Social media platforms significantly impact daily life, allowing information sharing and connections, but raising concerns about mental health issues like addiction and depression. To investigate this relationship, a fractional order model of social media addiction and depression has been developed using the Caputo derivative. The study calculates a basic reproduction number to predict the disease's presence and evaluates local stability using Routh-Hurwitz criteria. Sensitivity analysis is performed with partial rank correlation coefficients, and the model's solution reliability is assessed, including Ulam's stability analysis. Various numerical techniques are employed for simulation and error analysis, with graphical results demonstrating the effectiveness of the theoretical approach based on fractional order derivatives. This chapter is based on the studies [15, 14].

3.1 Introduction

The documentation of information in the modern era has undergone a substantial change, largely due to the exponential growth of internet-based social media platforms. Consequently, interpersonal communication has undergone a significant transformation [134]. The psychoactive influence of networking communication has increased as a result of its increasing popularity. Extensive research has associated compulsive use of social media platforms with detrimental outcomes, including reduced efficiency, negative social interactions, and decreased life satisfaction. Information technology has significantly reshaped the nature of social communication in the past few decades, with the rapid growth of internet-based social media platforms playing a pivotal role [299, 274, 132, 300]. Social media is a crucial technology that offers individuals valuable abilities, including but not limited to knowledge accessibility, addressing issues, entrepreneurship, self-motivated education, and more when utilized effectively [100, 259, 260]. Improper utilization of social media has adverse consequences, and one of the most significant outcomes is social media addiction [261, 205]. The emotional, relational, and performance-related problems associated with social media addiction (SMA) are a cause for concern, particularly given the rising popularity of social networking sites and the limited time people spend on

them [107, 296]. The misuse of social networking sites is on the rise, and this is exemplified by the alarming statistics of 68% and 73% of American adolescents using Facebook, respectively [263]. Overusing social media is linked to poor performance evaluation, difficult interpersonal connections, poor sleep, lower levels of pleasure, and hostile, anxious, and depressed moods [152, 119, 208, 138]. Similar to addictions to drugs, liquor, and gambling, SMA may initially seem not exceptional, but it is widespread and is only getting worse, and has to be addressed. The phrase “social media addiction” is frequently used to characterize those who participate in online communication apps like Instagram, Twitter, Facebook, YouTube, and others obsessively [74, 145]. In the twenty-first century, social media addiction is a rising issue. As a result, there has been an escalation of research studies on this topic conducted in multiple countries [97, 167].

Social relationships have been impacted by the transition away from in-person encounters to virtual ones brought on by social media. Online, people can act aggressively in ways they would not in real life. Aggression and SMA have a connection on a global and national scale. Depression, abusive behavior, and mental decline can be caused by social media addiction [135]. Spending too much time on social media has been linked with depression, social distance, and FOMO (fear of missing out), all of which have been shown to have a bad influence on one’s mental health [35]. More research is required to establish the correlation between addiction and depression on social media (SMAD). The study looks at the relation between SMA, anxiety, and depressive symptoms in Turkish, as can be shown in [88]. The World Health Organization (WHO) published a paper in 2019 that addressed the possible drawbacks of excessive social media use. According to studies included in the paper, using social media excessively might cause depression, stress, and other mental health problems [298]. Many reports and studies indicate that social media addiction leads to depression [146, 183]. The number of American women who fulfill the criteria for clinical depression is presently over seven million [215]. Even after considering gender variations in self-reporting behavior, a reliable study indicates that women are nearly twice as likely as men to suffer from clinical or subclinical depression [219]. Social media addiction and depression must be treated to stop additional negative effects. Both social media addiction and depression have treatments available, including clinical depression treatment, control measures like advertisements and educational materials, and therapeutic techniques like turning off alerts, putting time restrictions in place, cutting back on internet use, and never using a smartphone before bed.

Mathematical modeling is applied to study the prevention of infectious disease dynamics and plays an important scientific contribution [178, 177, 137]. The infectious disease dynamics model has been used by many researchers to study addictions to gaming, alcohol, drugs, and social media, depression, and other issues [136, 151, 12, 96]. Here, we proposed a deterministic model of social media addiction and depression (SMAD) to examine the effects of social media addiction on the mental health of people that leads to depression of any age. The social media-addicted population can be depressed by social comparison, increasing feelings of loneliness, harassment, bad news, and comments on social apps, decreasing motivation, increasing stress levels, decreasing social interaction in real life, relationship problems, etc. These psychological effects developed gradually over time, influenced by the user’s past exposure, behavior patterns, and emotional states. Due to the non-local phenomena, the current addiction or depression state is shaped by prior conditions. Multiple factors like screen time, pre-influence, stress, anxiety, and dopamine-driven reward process gradually influence human behavior. Fractional-order models have

been utilized in various fields like epidemiology, engineering, and social science to address real-life problems using various fractional derivatives [114, 239, 16], which particularly capture the non-locality through the memory effect, which indicates that the state depends on position, time, and prior conditions. The Caputo fractional derivative provides more significant solutions for fractional differential equations by considering the initial state, memory, and hereditary effect, enhancing simulation flexibility. Further insight into the fractions operators can be found in [248, 23, 156, 37, 3]. To the best of our knowledge, no prior study has investigated the interplay of these physiological states using fractional derivatives. The main novelty of this work lies in utilizing the Caputo fractional derivative to develop a fractional-order model that explores this phenomenon. We fill this research gap by extending the deterministic model through introducing a fractional order model with Caputo derivative of order $\aleph$. This framework enables the model to capture the memory and hereditary influence, which is common in human behavior processes.

The chapter outlines its organization, beginning with a deterministic model discussed in Sec 3.2. It covers mathematical analysis, including steady states and bifurcation in Sec 3.3, followed by sensitivity analysis in Sec 3.4. Dynamic analysis is further explored in Sec 3.5, while Sec 3.6 introduces fractional derivatives. Sec 3.7 focuses on the Caputo fractional order model regarding Social Media Addiction and Depression, emphasizing steady states and stability analysis. The mathematical validity of the model is supported in Sec 3.8, with solution existence and uniqueness properties detailed. The impact of deviations is examined in Sec 3.9 through Hyers–Ulam stability analysis. Numerical simulations in Sec 3.10 validate the model, showing addiction and depression dynamics across parameters. Sec 3.11 analyzes sensitive parameters pertinent to policy-making. Sec 3.12 evaluates simulation results and error analysis, leading to conclusions in Sec 3.13, which propose future directions in data calibration and optimal control.

3.2 Social media addiction and depression model formulation

In this research work, a fractional-order model of social media addiction and depression is developed. The model consists on six compartments of the population namely, Susceptible population (S) that are not addicted yet, Exposed individuals (E) are the people who use social media less frequently but are not addicted yet, Addicted individuals (I_1) are those people who are addicted to social media and spent most of their time on it, Depressed individuals (I_2) are the people those are in depression because of the SMA, Recovered individuals (R) the people who recovered from addiction, and Quitter individuals (Q) are those who give up and permanently stop using social media. The parameters involved in the system are presented in Table 3.1. The following epidemiological presumptions were made when developing the model.

- All the new immigrants join the susceptible at a rate Λ . The entire population interacts uniformly (homogeneous mixed). The SMA spreads due to pre-influence when susceptible individuals interact with addicted individuals.
- SMA outbreak in a closed environment where addiction risk is independent of race, gender, or wealth. There is natural mortality in each class.

- Susceptible moved to exposed individuals who interact with the addicted people shows the cycle of addiction transmission. These addicted individuals may recover or relapse to susceptibility during or after recovery.
- Risk of depression in SMA is strongly influenced by the pre-pressure of media, but this effect may be moderated by personal issues like socialization, etc.
- Depressed individuals may recover due to successful treatment, whereas the death rate due to depression, also considered here, includes suicide. Individuals who have recovered try to quit social media use completely.
- Immunity from addiction is temporary, as recovered individuals may relapse to susceptibility, and transmission from addiction to a depressed individual is instantaneous.

We proposed the social media addiction depression (SMAD) mathematical model here.

$$\begin{cases} \frac{dS}{dt} = \Lambda + \zeta \lambda R - \phi \chi I_1 S - (\beta + \tau) S, \\ \frac{dE}{dt} = \phi \chi I_1 S - (\zeta + \tau) E, \\ \frac{dI_1}{dt} = \Phi \zeta E - (\tau + \psi + \alpha) I_1, \\ \frac{dI_2}{dt} = \alpha I_1 + \psi (1 - \omega) I_1 - (v + \rho + \tau) I_2, \\ \frac{dR}{dt} = (1 - \Phi) \zeta E + v I_2 + \psi \omega I_1 - (\tau + \lambda) R, \\ \frac{dQ}{dt} = \beta S + (1 - \zeta) \lambda R - \tau Q. \end{cases} \quad (3.1)$$

The following are the initial conditions (ICs) at time $t=0$:

$$S(0) \geq 0, E(0) \geq 0, I_1(0) \geq 0, I_2(0) \geq 0, R(0) \geq 0, Q(0) \geq 0,$$

In this model (5.1), the total number of individuals (population) is denoted by N and is given by

$$N = S + E + I_1 + I_2 + R + Q$$

The recruiting of susceptible people into the population happens at a rate of Λ . These individuals are influenced by their peer pressure to use social media at a contact rate of ϕ from addicted individuals, with the possible transmission of χ , and join the exposed class. Some susceptibles transfer to permanent quitters at a rate of β . The exposed population suffers from SMA and transitions to the addicted class at a rate of $\Phi \zeta$, whereas the rest recovered by treatments at a rate $(1 - \Phi) \zeta$. The addictive individuals become depressed at a rate α , and those who leave treatment or need a second dose at a rate $(1 - \omega) \psi$ also become depressed. The depressed individuals either moved to the recovered class through education and/or treatment at a rate of v , or they passed away from depression at a rate of ρ . The probability rate of treatment to be successful in becoming recovered is $\omega \psi$. The recovered individuals either permanently quit using social media at a rate of $(1 - \zeta) \lambda$ or they start to be susceptible again to the SMAD at a rate of $\zeta \lambda$. The average death rate for the entire population is τ .

TABLE 3.1: Values of parameters involved in the proposed SMAD model (5.1).

Symbol	Parameter description	Value	Source
Λ	Recruitment rate of susceptible individuals	0.5	Assumed
ζ	Proportion of individuals in the recovered class susceptible to SMAD	0.35	[168]
λ	Rate at which individuals leave the recovered class	0.4	[12]
τ	Natural death rate	0.05–0.25	[12, 168]
α	Rate at which depression is induced by media impact	0.3–0.5	Approximate
β	Rate at which susceptible individuals avoid or stop using social media	0.01	Assumed
χ	Contact rate between susceptible and addicted individuals	0.25	[12]
v	Rate at which depressed individuals recover through treatment	0.7	[136]
ς	Rate at which individuals leave the exposed class	0.25	[136]
ω	Probability of successful treatment	0.8	[96]
ψ	Rate at which individuals leave treatment	0.0027	[96]
Φ	Proportion of exposed individuals who become addicted	0.7	[136]
ρ	Rate of suicide induced by depression	0.01	Assumed
ϕ	Transmission rate of addiction to the susceptible population	0.1–0.8	[136]

3.3 Mathematical analysis

The equilibrium points of the SMAD model are determined, showing that two equilibrium points are reached in the feasible region Δ after the model system. These steady states were derived using the Maple software. DFE of the model is given as

$$E_{q0} = (S^0, E^0, I_1^0, I_2^0, R^0, Q^0) = \left(\frac{\Lambda}{(\beta + \tau)}, 0, 0, 0, 0, \frac{\beta \Lambda}{(\beta + \tau)\tau} \right).$$

It will occur when $R_0 < 1$, which means there is no infection. This indicates that only susceptible individuals and those who permanently quit will remain, while others will vanish. The EEP of the model system is

$$E_{q1} = (S^1, E^1, I_1^1, I_2^1, R^1, Q^1).$$

who corresponding values are given in [15]. From biological considerations, we study model (5.1) in the closed set

$$\Delta = \left\{ (S, E, I_1, I_2, R, Q) \in \mathbb{R}_+^6 : 0 < N \leq \frac{\Lambda}{\tau} \right\}.$$

where $\mathbb{R}_+^6$ shows the lower-dimensional faces of the non-negative cone. Δ is evidently positively invariant with respect to model (5.1).

Theorem 3.1. *The Δ is positively invariant for model (5.1).*

Proof. Assume the total population

$$N = S + E + I_1 + I_2 + R + Q,$$

we have

$$\frac{dN}{dt} \leq \Lambda - \tau N - \rho D,$$

which yields that

$$\frac{dN}{dt} \leq \Lambda - \tau N.$$

Hence, it suggests that $\frac{dN}{dt} < 0$ as often as $N > \frac{\Lambda}{\tau}$. Thus, we have $\frac{dN}{dt}$ is bounded by $\Lambda - \tau$. Using the method of integrating factor on the inequality, we get

$$\frac{d}{dt}(e^{\tau t} N) \leq e^{\tau t} \Lambda. \quad (3.2)$$

Thus with IC's, integrating (3.2), we write

$$N(t) \leq N(0) e^{-\tau t} + \frac{\Lambda}{\tau} (1 - e^{-\tau t}).$$

Suppose $t \rightarrow \infty$, we obtain

$$N \leq \frac{\Lambda}{\tau}.$$

As a result, Δ is positively invariant of the model (5.1), meaning that none of its boundaries are crossed by any solution path. This demonstrates that the developed model is applicable from a mathematical and epidemiological perspective. The model is regarded as being within the range of biological feasibility. It is necessary to demonstrate that every phase trajectory that began anywhere in the feasible region Δ of the phase space ultimately enters Δ and stays there. It is possible to achieve this by demonstrating the system's global attraction and positively invariant set. $\square$

We move to define the non-negativity of the model's solution (5.1) by calculating the positivity of the solution. It is important to establish that all of the state variables in the equations of the model (5.1) are non-negative $\forall t$ for it to have epidemiological significance. To prove the positivity of the model's solution, we considered the following results.

Theorem 3.2. *Considering the ICs presented in (5.1), the solution of the SMAD model(5.1) is positive for all $t > 0$.*

Proof. By using the susceptible class in the system of equations (5.1), we assumed that

$$\frac{dS}{dt} = \Lambda + \zeta \lambda R - \phi \chi I_1 S - (\beta + \tau) S \leq \Lambda - (\beta + \tau) S.$$

By taking I.F = $e^{(\beta+\tau)t}$, we get

$$\begin{aligned} e^{(\beta+\tau)t} \frac{dS}{dt} + (\beta + \tau) S e^{(\beta+\tau)t} &\leq \Lambda e^{(\beta+\tau)t}. \\ S e^{(\beta+\tau)t} &\leq \frac{\Lambda e^{(\beta+\tau)t}}{\beta + \tau} + k. \end{aligned} \quad (3.3)$$

At $t = 0$, we have

$$S(0) \leq \frac{\Lambda}{\beta + \tau} + k.$$

Using (3.3), we define

$$S \leq S(0) e^{-(\beta+\tau)t} + \frac{\Lambda}{\beta+\tau} \left(1 - e^{-(\beta+\tau)t}\right). \quad (3.4)$$

When $t \rightarrow \infty$, we get

$$\frac{\Lambda}{\beta+\tau} \geq S(t) \geq 0. \quad (3.5)$$

By the same procedure, we can show that

$$E \geq 0, I_1 \geq 0, I_2 \geq 0, R \geq 0, Q \geq 0.$$

This indicates that the solution of model (5.1) is positive. $\square$

To describe the mathematical property that the obtained solutions of the model remain bounded in a feasible region Δ , we show the following results.

Theorem 3.3. *All the solutions of the model (5.1) are bounded.*

Proof. By considering total population N , we have

$$\frac{dN}{dt} = \Lambda - \tau N - \rho I_2. \quad (3.6)$$

Suppose, for any initial condition $N(0) \leq \frac{\Lambda}{\tau}$, where $N(0) = S(0) + E(0) + I_1(0) + D(0)R(0) + Q(0)$, we get

$$N \leq \frac{\Lambda}{\tau} \quad \text{for all } t \geq 0.$$

From (3.6), we have

$$\frac{dN}{dt} \leq \Lambda - \tau N.$$

Using Gronwall's inequality, we get

$$N \leq \frac{\Lambda}{\tau} + \left(N(0) - \frac{\Lambda}{\tau}\right) e^{-\tau t}.$$

Hence $N \leq \frac{\Lambda}{\tau}$, for all $t \geq 0$, whenever $N(0) \leq \frac{\Lambda}{\tau}$.

Clearly,

$$\liminf_{t \rightarrow \infty} N \leq \limsup_{t \rightarrow \infty} N \leq \frac{\Lambda}{\tau} > 0.$$

This indicates that the solution of model (5.1) is bounded in the feasible region Δ . This shows that the feasible area Δ contains the positive solution to the model (5.1). $\square$

3.3.1 Threshold parameter

The disease's propagation is measured using the basic reproduction number $\mathbf{R}_0$ which refers to the infected population that can be generated by an infected individual during their infectious period. $\mathbf{R}_0$ of the model plays a vital role in analyzing the stability of steady states. If $\mathbf{R}_0 < 1$, there won't be an epidemic in the population, and if $\mathbf{R}_0 > 1$, the epidemic will continue in the population. The next generation matrix (NGM) technique is applied to calculate the $\mathbf{R}_0$ and according to the approach,

system (5.1) can be expressed as :

$$\begin{cases} \frac{dE}{dt} = \phi \chi S I_1 - \varsigma E - \tau E, \\ \frac{dI_1}{dt} = \Phi \varsigma E - \tau I_1 - \psi I_1 - \alpha I_1, \\ \frac{dI_2}{dt} = \alpha I_1 + \psi (1 - \omega) I_1 - (v + \rho + \tau) I_2, \\ \frac{dR}{dt} = (1 - \Phi) \varsigma E + v I_2 + \psi \omega I_1 - (\tau + \lambda) R. \end{cases} \quad (3.7)$$

Then, we get

$$\mathbf{F} = \begin{pmatrix} \phi \chi S I_1 \\ 0 \\ 0 \\ 0 \end{pmatrix}, \quad \mathbf{V} = \begin{pmatrix} (\varsigma + \tau) E \\ -\Phi \varsigma E + (\tau + \psi + \alpha) I_1 \\ -\alpha I_1 - \psi (1 - \omega) I_1 + (v + \rho + \tau) I_2 \\ -(1 - \Phi) \varsigma E - v I_2 - \psi \omega I_1 + (\tau + \lambda) R \end{pmatrix}$$

The Jacobi matrices of $\mathbf{F}$ and $\mathbf{V}$ at SMADDFE, represented by $\mathcal{F}$ and $\mathcal{V}$ are provided below:

$$\mathcal{F} = \begin{pmatrix} 0 & \frac{\Lambda \phi \chi}{\beta + \tau} & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix},$$

$$\mathcal{V} = \begin{pmatrix} \varsigma + \tau & 0 & 0 & 0 \\ -\varsigma \Phi & \tau + \psi + \alpha & 0 & 0 \\ 0 & -\alpha - \psi (1 - \omega) & v + \rho + \tau & 0 \\ -(1 - \Phi) \varsigma & -\psi \omega & -v & \tau + \lambda \end{pmatrix}$$

The product matrix $\mathcal{F} \mathcal{V}^{-1}$ contains the largest eigenvalue, which is the model's next generation matrix and is shown by

$$\mathbf{R}_0 = \frac{\Lambda \varsigma \phi \chi \Phi}{(\alpha + \psi + \tau) (\beta + \tau) (\varsigma + \tau)}.$$

Thus, by epidemiological properties, we conclude that

- if $\mathbf{R}_0 < 1$, the occurrence of the disease will decrease.
- if $\mathbf{R}_0 = 1$, the disease occurrence will be constant.
- if $\mathbf{R}_0 > 1$ the occurrence of the disease will increase.

In view of these remarks on our model, we have

- if $\Lambda \varsigma \phi \chi \Phi < (\alpha + \psi + \tau) (\beta + \tau) (\varsigma + \tau)$, the occurrence of the addiction and depression will decrease.
- if $\Lambda \varsigma \phi \chi \Phi = (\alpha + \psi + \tau) (\beta + \tau) (\varsigma + \tau)$, the addiction and depression occurrence will be constant.
- if $\Lambda \varsigma \phi \chi \Phi > (\alpha + \psi + \tau) (\beta + \tau) (\varsigma + \tau)$ the occurrence of the addiction and depression will increase.

The short-term breakouts of the disease are caused by the stability of disease-free equilibrium points. Over a longer time, its dynamics are characterized by stability at the endemic equilibrium sites. We discovered that persistent behavior influences critical epidemiological results, including whether an outbreak of a disease would remain in an endemic condition or if the disease would disperse. Results of stability analysis of model steady states can be seen in [15].

3.3.2 Forward bifurcation analysis of model

Theorem 3.4. *If $R_0 > 1$, then the EEP E_{q1} of model (5.1) is locally asymptotically stable in feasible region Δ and the model (5.1) shows forward bifurcation at $R_0 = 1$*

Proof. To use the approach, the model system (5.1) is simplified, and the following variables are changed. Let

$$S = y_1, E = y_2, I_1 = y_3, I_2 = y_4, R = y_5, Q = y_6$$

so that

$$N = y_1 + y_2 + y_3 + y_4 + y_5 + y_6$$

Moreover, the model (5.1) becomes $\frac{dY}{dt} = (g_1, g_2, g_3, g_4, g_5, g_6)^T$ when we apply the vector notation $Y = (y_1, y_2, y_3, y_4, y_5, y_6)^T$.

$$\begin{cases} \frac{dy_1}{dt} = g_1 = \Lambda + \zeta \lambda y_5 - \phi \chi y_3 y_1 - (\beta + \tau) y_1, \\ \frac{dy_2}{dt} = g_2 = \phi \chi y_3 y_1 - (\varsigma + \tau) y_2, \\ \frac{dy_3}{dt} = g_3 = \Phi \varsigma y_2 - (\tau + \psi + \alpha) y_3, \\ \frac{dy_4}{dt} = g_4 = \alpha y_3 + \psi (1 - \omega) y_3 - (v + \rho + \tau) y_4, \\ \frac{dy_5}{dt} = g_5 = (1 - \Phi) \varsigma y_2 + v y_4 + \psi \omega y_3 - (\tau + \lambda) y_5, \\ \frac{dy_6}{dt} = g_6 = \beta y_1 + (1 - \zeta) \lambda y_5 - \tau y_6. \end{cases} \quad (3.8)$$

The Jacobin of the model (3.8) is given as

$$J = \begin{pmatrix} K_1 & 0 & -\phi \chi S & 0 & \zeta \lambda & 0 \\ \phi \chi I_1 & -\varsigma - \tau & \phi \chi S & 0 & 0 & 0 \\ 0 & \Phi \varsigma & K_2 & 0 & 0 & 0 \\ 0 & 0 & K_3 & -v - \rho - \tau & 0 & 0 \\ 0 & (1 - \Phi) \varsigma & \omega \psi & v & -\tau - \lambda & 0 \\ \beta & 0 & 0 & 0 & K_4 & -\tau \end{pmatrix} \quad (3.9)$$

where

$$\begin{aligned} K_1 &= -\phi \chi I_1 - \beta - \tau, & K_2 &= -\tau - \alpha - \psi, & K_3 &= \alpha + \psi (1 - \omega), \\ K_4 &= (1 - \zeta) \lambda. \end{aligned}$$

Evaluating (3.9) at the DFE $E_{q0} = (S^0 = y_1^0, E^0 = y_2^0, I_1^0 = y_3^0, I_2^0 = y_4^0, R^0 = y_5^0, Q^0 = y_6^0)$, we get

$$J(E_{q0}) = \begin{pmatrix} -\beta - \tau & 0 & -\frac{\phi \chi \Lambda}{\beta + \tau} & 0 & \zeta \lambda & 0 \\ 0 & -\zeta - \tau & \frac{\phi \chi \Lambda}{\beta + \tau} & 0 & 0 & 0 \\ 0 & \Phi \zeta & K_2 & 0 & 0 & 0 \\ 0 & 0 & K_3 & -v - \rho - \tau & 0 & 0 \\ 0 & -(-1 + \Phi) \zeta & \omega \psi & v & -\tau - \lambda & 0 \\ \beta & 0 & 0 & 0 & K_4 & -\tau \end{pmatrix} \quad (3.10)$$

The threshold parameter R_0 of SMAD is specified as

$$R_0 = \frac{\Lambda \zeta \phi \chi \Phi}{(\alpha + \psi + \tau)(\beta + \tau)(\zeta + \tau)}$$

We use ϕ as a bifurcation parameters implies that $R_0 = 1$ iff

$$\phi = \phi^* = \frac{(\alpha + \psi + \tau)(\beta + \tau)(\zeta + \tau)}{\Lambda \zeta \chi \Phi}$$

There is a clear zero eigenvalue in the linearized system of the modified model system (3.8) with $\phi = \phi^*$ as the bifurcation parameter.

$$J(\phi^*) = \begin{pmatrix} -\beta - \tau & 0 & -\frac{(\zeta + \tau)(\tau + \alpha + \psi)}{\Phi \zeta} & 0 & \zeta \lambda & 0 \\ 0 & -\zeta - \tau & \frac{(\zeta + \tau)(\tau + \alpha + \psi)}{\Phi \zeta} & 0 & 0 & 0 \\ 0 & \Phi \zeta & K_2 & 0 & 0 & 0 \\ 0 & 0 & K_3 & -v - \rho - \tau & 0 & 0 \\ 0 & -(-1 + \Phi) \zeta & \psi \omega & v & -\tau - \lambda & 0 \\ \beta & 0 & 0 & 0 & K_4 & -\tau \end{pmatrix} \quad (3.11)$$

The eigenvalues are

$$0, -\tau, -(\tau + \lambda), -(\beta + \tau), -(\alpha + \zeta + 2\tau + \psi), -(v + \rho + \tau).$$

Next, we find the left and right eigenvector V, W respectively, both of which are associated with the zero eigenvalues of the Jacobin of (3.11) at (represented by J_{ϕ^*}) selected such that $J(E_{q0}) \times W = 0$ and $V \times J(E_{q0}) = 0$ with $V.W = 1$, where

$$W = [w_1, w_2, w_3, w_4, w_5, w_6] \quad V = [v_1, v_2, v_3, v_4, v_5, v_6]$$

$$J(E_{q0}) \times W_i = 0$$

where

$$\begin{aligned}
 W_i &= \begin{pmatrix} w_1 \\ w_2 \\ w_3 \\ w_4 \\ w_5 \\ w_6 \end{pmatrix} \\
 &\begin{pmatrix} -\beta - \tau & 0 & -\frac{\phi \chi \Lambda}{\beta + \tau} & 0 & \zeta \lambda & 0 \\ 0 & -\varsigma - \tau & \frac{\phi \chi \Lambda}{\beta + \tau} & 0 & 0 & 0 \\ 0 & \Phi \varsigma & -\tau - \alpha - \psi & 0 & 0 & 0 \\ 0 & 0 & -\omega \psi + \alpha + \psi & -v - \rho - \tau & 0 & 0 \\ 0 & -(-1 + \Phi) \varsigma & \omega \psi & v & -\tau - \lambda & 0 \\ \beta & 0 & 0 & 0 & -(-1 + \zeta) \lambda & -\tau \end{pmatrix} \times W_i \\
 &= \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}
 \end{aligned}$$

Thus, we have

$$\begin{cases} (-\beta - \tau)w_1 - \frac{\phi \chi \Lambda}{\beta + \tau}w_3 + \zeta \lambda w_5 & = 0, \\ (-\varsigma - \tau)w_2 + \frac{\phi \chi \Lambda}{\beta + \tau}w_3 & = 0, \\ \Phi \varsigma w_2 + (-\tau - \alpha - \psi)w_3 & = 0, \\ (-\omega \psi + \alpha + \psi)w_3 + (-v - \rho - \tau)w_4 & = 0, \\ (1 - \Phi) \varsigma w_2 + \omega \psi w_3 + v w_4 + (-\tau - \lambda)w_5 & = 0 \\ \beta w_1 + (1 - \zeta) \lambda w_5 - \tau w_6 & = 0. \end{cases} \quad (3.12)$$

Solving (3.12), we write

$$\begin{aligned}
w_1 &= (-(((\zeta(-1+\Phi)\tau^3 - ((-\beta-\rho+(\omega-1)\psi-\alpha-v)\Phi + \psi+\rho+\beta+\alpha+v)\zeta\tau^2 \\
&\quad + ((((-\omega+1)\psi+\rho+\alpha+v)\beta - ((\omega-1)\psi-\alpha)\rho)\Phi + (-\psi-\rho-\alpha-v)\beta - \\
&\quad (v+\rho)(\psi+\alpha))\zeta + \phi\chi\Lambda\Phi)\tau - \beta(\rho((\omega-1)\psi-\alpha)\Phi + (v+\rho)(\psi+\alpha))\zeta \\
&\quad + \chi\Lambda\Phi\phi(v+\rho))\lambda + \tau\chi\Lambda\Phi\phi(v+\rho+\tau)\zeta)/((\beta+\tau)^2(\tau+\alpha+\psi) \\
&\quad (v+\rho+\tau)(\tau+\lambda)))w_2, \\
w_2 &> 0 \quad (\text{can take any value}), \\
w_3 &= \frac{\Phi\zeta w_2}{\tau+\alpha+\psi}, \\
w_4 &= \frac{(-\omega\psi+\alpha+\psi)\Phi\zeta w_2}{(\tau+\alpha+\psi)(v+\rho+\tau)}, \\
w_5 &= -((-1+\Phi)\tau^2 + (((-\omega+1)\psi+\rho+\alpha+v)\Phi - \psi-\rho-\alpha-v)\tau \\
&\quad - \rho((\omega-1)\psi-\alpha)\Phi - (v+\rho)(\psi+\alpha))\zeta w_2/((\tau+\alpha+\psi)(v+\rho+\tau)(\tau+\lambda)) \\
w_6 &= (((((-1+\Phi)(-1+\zeta)\tau^4 + ((-1+\Phi)(\zeta-2)\beta - ((-\rho+(\omega-1)\psi-\alpha-v)\Phi + \psi+\rho+\alpha+v)(\zeta-2)\beta - (-1+\zeta)(\rho((\omega-1)\psi-\alpha)\Phi + (v+\rho)(\psi+\alpha)))\tau^2 - \\
&\quad ((((-\omega+1)\psi+\rho+\alpha+v)\Phi - \psi-\rho-\alpha-v)\beta + (((\omega-1)\psi-\alpha)(\zeta-2)\rho + \phi\chi\Lambda\Phi + (v+\rho)(\psi+\alpha)(\zeta-2))\beta\tau - ((-\rho((\omega-1)\psi-\alpha)\Phi - (v+\rho)(\psi+\alpha))\beta + \chi\Lambda\Phi\phi(v+\rho))\beta)\lambda - \tau\chi\Lambda\Phi\phi\beta(v+\rho+\tau)\zeta)/((\beta+\tau)^2(\tau+\alpha+\psi)(v+\rho+\tau)(\tau+\lambda)\tau))w_2.
\end{aligned} \tag{3.13}$$

Similarly, calculating the left eigenvector $V = [v_1, v_2, v_3, v_4, v_5, v_6]^T$ with $V \times J(E_{q0}) = 0$, gives

$$V_i \times J(E_{q0}) = 0,$$

where

$$\begin{aligned}
V_i &= (v_1 \ v_2 \ v_3 \ v_4 \ v_5 \ v_6) \\
V_i \times &\begin{pmatrix} -\beta-\tau & 0 & -\frac{\phi\chi\Lambda}{\beta+\tau} & 0 & \zeta\lambda & 0 \\ 0 & -\zeta-\tau & \frac{\phi\chi\Lambda}{\beta+\tau} & 0 & 0 & 0 \\ 0 & \Phi\zeta & -\tau-\alpha-\psi & 0 & 0 & 0 \\ 0 & 0 & K_1 & K_2 & 0 & 0 \\ 0 & -(-1+\Phi)\zeta & \omega\psi & v & -\tau-\lambda & 0 \\ \beta & 0 & 0 & 0 & K_3 & -\tau \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}.
\end{aligned}$$

where

$$K_1 = -\omega\psi + \alpha + \psi, \quad K_2 = -v - \rho - \tau, \quad K_3 = -(-1 + \zeta)\lambda.$$

Thus, we have

$$\begin{cases} -(\beta + \tau)v_1 + \beta v_6 & = 0 \\ -(\zeta + \tau)v_2 + \Phi \zeta v_3 + (1 - \Phi)\zeta v_5 & = 0 \\ -\frac{\phi \chi \Lambda}{\beta + \tau}v_1 + \frac{\phi \chi \Lambda}{\beta + \tau}v_2 - (\tau + \alpha + \Psi)v_3 + (\omega \psi + \alpha + \psi)v_4 + \omega \psi v_5 & = 0 \\ -(v + \rho + \tau)v_4 + v v_5 & = 0 \\ \zeta \lambda v_1 - (\tau + \lambda)v_5 + (1 - \zeta)\lambda v_6 & = 0 \\ -\tau v_6 & = 0 \end{cases} \quad (3.14)$$

Solving (3.14), we conclude

$$\begin{aligned} v_1 &= 0, \\ v_2 &> 0 \quad (\text{can take any value}), \\ v_3 &= \frac{\zeta + \tau}{\Phi \zeta} v_2, \\ v_4 &= 0, \\ v_5 &= 0, \\ v_6 &= 0. \end{aligned} \quad (3.15)$$

The stability of model (5.1) around DFE is entirely determined by a and b, defined as

$$a = \sum_{k,i,j=1}^{n=6} v_k w_i w_j \frac{\partial^2 g_x(0,0)}{\partial y_i \partial y_j} \quad (3.16)$$

$$b = \sum_{k,i=1}^{n=6} v_k w_i \frac{\partial^2 g_x(0,0)}{\partial y_i \partial \phi^*} \quad (3.17)$$

Thus, the only non-zero terms in the sums for a and b are those that correspond to $k = 2, 3$. So from the system (3.8), we have

$$g_2 = \phi \chi y_3 y_1 - (\zeta + \tau) y_2 \quad (3.18)$$

$$g_3 = \Phi \zeta y_2 - (\tau + \psi + \alpha) y_3. \quad (3.19)$$

All the second derivatives of (3.18) are zeros. The only remaining equation is (3.19), which has non-zero derivatives as

$$\begin{aligned} \frac{\partial^2 g_2}{\partial y_1 \partial y_3} &= \phi^* \chi \\ \frac{\partial^2 g_2}{\partial y_3 \partial y_1} &= \phi^* \chi \\ \frac{\partial^2 g_2}{\partial y_3 \partial \phi} &= y_1^* \chi \end{aligned}$$

Now through applying (3.16), we have

$$\begin{aligned} a &= v_2 w_1 w_3 \left(\frac{\partial^2 g_2}{\partial y_1 \partial y_3} + \frac{\partial^2 g_2}{\partial y_3 \partial y_1} \right) \\ a &= 2v_2 w_1 w_3 \phi^* \chi < 0 \end{aligned} \quad (3.20)$$

For the value of b , use (3.17) leads to

$$b = v_2 w_3 x_1^* \chi > 0 \quad (3.21)$$

Since the unique endemic equilibrium point is locally asymptotically stable for $\mathbf{R}_0 > 1$ and model (5.1) displays forward bifurcation at $\mathbf{R}_0 = 1$, the DFE and EEP cannot coexist at $\mathbf{R}_0 < 1$. $\square$

3.4 Sensitivity analysis of model

We performed a sensitivity analysis to show the influence of every parameter on the SMAD transmission. We applied the normalized sensitivity index concept to conduct this analysis.

Definition 3.5. The normalized forward sensitivity index (NFSI) of a variable, threshold parameter $\mathbf{R}_0$, that depends differentially on a parameter A is defined as:

$$\Lambda_p^{\mathbf{R}_0} = \frac{\partial \mathbf{R}_0}{\partial I_1} \times \frac{A}{\mathbf{R}_0}$$

where s denotes all parameters and $\mathbf{R}_0$ is the basic reproduction number

$$\mathbf{R}_0 = \frac{\Lambda \varsigma \phi \chi \Phi}{(\alpha + \psi + \tau)(\beta + \tau)(\varsigma + \tau)}.$$

For calculating the sensitivity index of $\mathbf{R}_0$ to the parameters of the model:

- For the parameter ς

$$\begin{aligned} \Lambda_{\varsigma}^{\mathbf{R}_0} &= \frac{\partial \mathbf{R}_0}{\partial \varsigma} \times \frac{\varsigma}{\mathbf{R}_0} \\ \Lambda_{\varsigma}^{\mathbf{R}_0} &= \frac{\Lambda \Phi \chi \phi \tau}{(\beta + \tau)(\tau + \varsigma)^2(\tau + \alpha + \psi)} \times \frac{(\beta + \tau)(\tau + \varsigma)(\tau + \alpha + \psi)}{\Lambda \Phi \chi \phi} \\ \Lambda_{\varsigma}^{\mathbf{R}_0} &= \frac{\tau}{\tau + \varsigma} > 0 \end{aligned}$$

- For the parameter ϕ

$$\begin{aligned} \Lambda_{\phi}^{\mathbf{R}_0} &= \frac{\partial \mathbf{R}_0}{\partial \phi} \times \frac{\phi}{\mathbf{R}_0} \\ \Lambda_{\phi}^{\mathbf{R}_0} &= \frac{\Lambda \varsigma \Phi \chi}{(\beta + \tau)(\tau + \varsigma)(\tau + \alpha + \psi)} \times \frac{(\beta + \tau)(\tau + \varsigma)(\tau + \alpha + \psi)}{\Lambda \varsigma \Phi \chi} \\ \Lambda_{\phi}^{\mathbf{R}_0} &= 1 > 0 \end{aligned}$$

- For the parameter χ

$$\begin{aligned} \Lambda_{\chi}^{\mathbf{R}_0} &= \frac{\partial \mathbf{R}_0}{\partial \chi} \times \frac{\chi}{\mathbf{R}_0} \\ \Lambda_{\chi}^{\mathbf{R}_0} &= \frac{\Lambda \varsigma \Phi \phi}{(\beta + \tau)(\tau + \varsigma)(\tau + \alpha + \psi)} \times \frac{(\beta + \tau)(\tau + \varsigma)(\tau + \alpha + \psi)}{\Lambda \varsigma \Phi \phi} \\ \Lambda_{\chi}^{\mathbf{R}_0} &= 1 > 0 \end{aligned}$$

- For the parameter Φ

$$\begin{aligned}\Lambda_{\Phi}^{\mathbf{R}_0} &= \frac{\partial \mathbf{R}_0}{\partial \Phi} \times \frac{\Phi}{\mathbf{R}_0} \\ \Lambda_{\Phi}^{\mathbf{R}_0} &= \frac{\Lambda \varsigma \chi \phi}{(\beta + \tau)(\tau + \varsigma)(\tau + \alpha + \psi)} \times \frac{(\beta + \tau)(\tau + \varsigma)(\tau + \alpha + \psi)}{\Lambda \varsigma \chi \phi} \\ \Lambda_{\Phi}^{\mathbf{R}_0} &= 1 > 0\end{aligned}$$

- For the parameter β

$$\begin{aligned}\Lambda_{\beta}^{\mathbf{R}_0} &= \frac{\partial \mathbf{R}_0}{\partial \beta} \times \frac{\beta}{\mathbf{R}_0} \\ \Lambda_{\beta}^{\mathbf{R}_0} &= -\frac{\Lambda \varsigma \Phi \chi \phi}{(\beta + \tau)^2(\tau + \varsigma)(\tau + \alpha + \psi)} \times \frac{\beta(\beta + \tau)(\tau + \varsigma)(\tau + \alpha + \psi)}{\Lambda \varsigma \Phi \chi \phi} \\ \Lambda_{\beta}^{\mathbf{R}_0} &= -\frac{\beta}{\beta + \tau} < 0\end{aligned}$$

- For the parameter τ

$$\begin{aligned}\Lambda_{\tau}^{\mathbf{R}_0} &= \frac{\partial \mathbf{R}_0}{\partial \tau} \times \frac{\tau}{\mathbf{R}_0} \\ \Lambda_{\tau}^{\mathbf{R}_0} &= -3\phi\chi\Phi\varsigma\Lambda \frac{\tau^2 + \frac{2}{3}(\psi + \beta + \alpha + \varsigma)\tau + \frac{1}{3}\varsigma(\psi + \beta + \alpha) + \frac{1}{3}\beta(\psi + \alpha)}{(\beta + \tau)^2(\tau + \varsigma)^2(\tau + \alpha + \psi)^2} \\ &\quad \times \frac{\tau(\beta + \tau)(\tau + \varsigma)(\tau + \alpha + \psi)}{\Lambda \varsigma \Phi \chi \phi} \\ \Lambda_{\tau}^{\mathbf{R}_0} &= -3\tau \frac{\tau^2 + \frac{2}{3}(\psi + \beta + \alpha + \varsigma)\tau + \frac{1}{3}\varsigma(\psi + \beta + \alpha) + \frac{1}{3}\beta(\psi + \alpha)}{(\tau + \alpha + \psi)(\beta + \tau)(\tau + \varsigma)} < 0\end{aligned}$$

- For the parameter α

$$\begin{aligned}\Lambda_{\alpha}^{\mathbf{R}_0} &= \frac{\partial \mathbf{R}_0}{\partial \alpha} \times \frac{\alpha}{\mathbf{R}_0} \\ \Lambda_{\alpha}^{\mathbf{R}_0} &= -\frac{\Lambda \varsigma \Phi \chi \phi}{(\beta + \tau)(\tau + \varsigma)(\tau + \alpha + \psi)^2} \times \frac{\alpha(\beta + \tau)(\tau + \varsigma)(\tau + \alpha + \psi)}{\Lambda \varsigma \Phi \chi \phi} \\ \Lambda_{\alpha}^{\mathbf{R}_0} &= -\frac{\alpha}{\tau + \alpha + \psi} < 0\end{aligned}$$

- For the parameter ψ

$$\begin{aligned}\Lambda_{\psi}^{\mathbf{R}_0} &= \frac{\partial \mathbf{R}_0}{\partial \psi} \times \frac{\psi}{\mathbf{R}_0} \\ \Lambda_{\psi}^{\mathbf{R}_0} &= -\frac{\Lambda \varsigma \Phi \chi \phi}{(\beta + \tau)(\tau + \varsigma)(\tau + \alpha + \psi)^2} \times \frac{\psi(\beta + \tau)(\tau + \varsigma)(\tau + \alpha + \psi)}{\Lambda \varsigma \Phi \chi \phi} \\ \Lambda_{\psi}^{\mathbf{R}_0} &= -\frac{\psi}{\tau + \alpha + \psi} < 0\end{aligned}$$

If any of the variables have positive indices, it means that they have a significant influence on the spread of the disease throughout the population. Additionally, when parameter values increase, individuals whose sensitivity indices are negative have an impact on reducing disease burden in the population. As a result, stakeholders and policy makers should concentrate on decreasing positive indices and raising negative indices parameters. The impact of different parameters is shown in Fig 3.1. The graph in Fig 3.1(a) shows how the value of the R_0 changes when the number of people who leave the exposed class B and the pace at which addiction spreads to the vulnerable population ϕ fluctuate. Here, we see that ς raises the value of R_0 as ϕ increases, indicating that the disease can be eradicated by lowering ς and ϕ . Fig 3.1(a) also shows that ϕ has a stronger effect on R_0 than ς . In Fig 3.1(b), the plot depicts how the value of R_0 changes when the susceptible who avoid and/or stop using social media β and the transmission rate of addiction to the susceptible population ϕ vary concurrently. Here we analyses that ϕ increases the value of R_0 and β decreases the value of R_0 , which indicates that the disease can be extinct by reducing the value of ϕ and by increasing the value of β . From Fig 3.1(b), it can also be observed that ϕ has more influence than beta on R_0 . Also, the graph in Fig 3.1(e) shows how the value of the R_0 evolves when the contact rate of susceptible with the addicted population χ and the rate at which depression is caused by media effect α vary simultaneously. Here, we determine that χ increases the R_0 value while alpha reduces it, indicating that the disease may be wiped out by decreasing χ and raising α . It is also clear from Fig 3.1(e) that χ has a stronger impact on R_0 than α . Similarly, the graph in Fig 3.1(j) shows how the value of R_0 varies when the natural mortality rate τ and the proportion of individuals exposed who subsequently develop addiction Φ are taken into account. Here, we see that τ reduces the value of R_0 but Φ increases it, indicating that the disease may be cured by decreasing Φ . Fig 3.1(j) additionally indicates that Φ has a stronger impact on R_0 than τ .

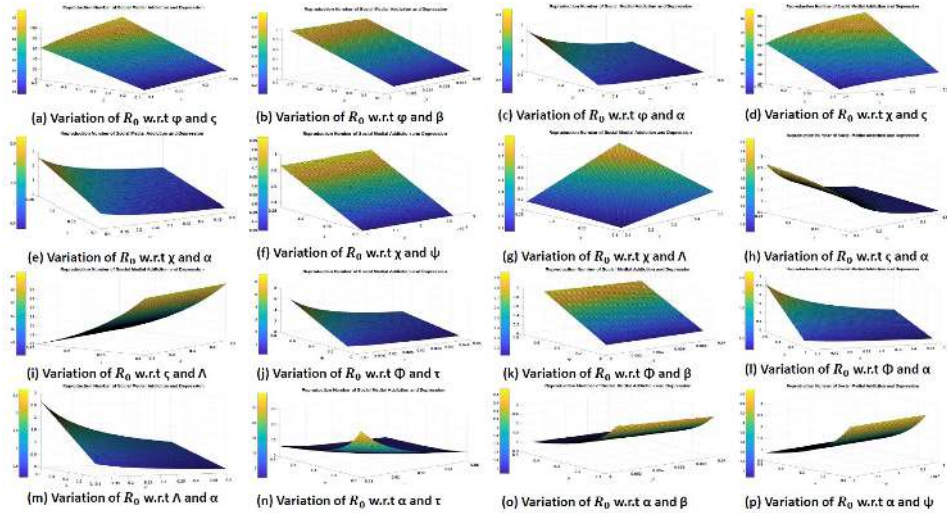


FIGURE 3.1: Variation of different parameters over R_0 .

3.4.1 Global sensitivity analysis by Partial Rank Correlation Coefficient

Sensitivity analysis is used to identify key parameters that influence the model's predictions and could potentially reduce the spread of the disease. To accomplish this, the Latin Hypercube Sampling (LHS) method is used here to estimate the PRCC,

which is a globally sensitive analysis method. The level of correlation between the model output and input variables is evaluated using PRCC. It measures the linear dependency between two variables after taking into account the linear impacts of other variables. The PRCC analysis is used here to examine the sensitivity of the model parameters on the output variables of social media addiction and depression. It measures how much the variation in the output variables is explained by the variation in the input parameters while controlling for the effects of other input parameters. The range of PRCC values is $[-1, 1]$, where a value of 1 specifies a perfect positive correlation, -1 specifies a perfect negative correlation, and 0 indicates no correlation between the variables.

For each output variable of interest, approximately 5,000 simulations were conducted, with each model input parameter allocated a uniform distribution from its baseline value. Now, we take into consideration that the model outputs are the number of susceptible individuals, exposed individuals, addicted individuals, depressed individuals, recovered Individuals, permanent quitter individuals, and R_0 . Fig 3.2 indicates the sensitivity indexes of parameters of R_0 . Fig 3.3 refers to the correlation between the parameters of R_0 and all model classes. It also indicates the P-values. By performing PRCC analysis, we aimed to identify the most influential parameters in the SMAD model and to determine the direction and strength of their influence on the output variables. From Fig 3.3(c-d), we analyse that for addicted individuals the parameters $\Lambda, \tau, \phi, \psi, \Phi$ have positive PRCC values and $\alpha, \beta, \chi, \varsigma$ have negative PRCC values. Similarly, for depressed individuals, the parameters α, ϕ, ψ have positive PRCC values, and $\Lambda, \tau, \Phi, \beta, \chi, \varsigma$ have negative PRCC values. This analysis indicates that a positive change in those parameters having positive PRCC values will increase the number of infected and depressed individuals. On the other hand, those parameters have negative PRCC values, which shows that increasing these parameters will decrease the number of infected and depressed individuals.

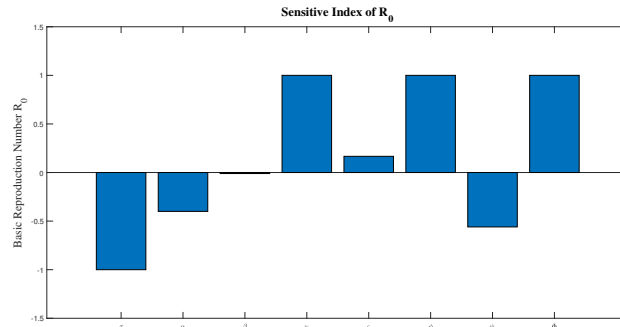


FIGURE 3.2: Sensitivity Index of Reproduction Number R_0

3.5 Dynamic analysis of model

This section carries out a detailed numerical interpretation of the model (5.1) by the RK-4 scheme using Matlab. We analyze numerical experiments for this technique using all the parameter values listed in the Table 3.1. To verify the accuracy of all the solutions of the model (5.1), the Rk-4 scheme was applied using the discretization step size $h = 0.001$ for DFE E_{q0} and EEP E_{q1} . It is observe that numerical scheme converge to true steady states for $1 < R_0 < 1$ and give all positive solutions in feasible region Δ with IC's $(S(0), E(0), I_1(0), I_2(0), R(0), Q(0)) = (100, 1, 5, 2, 0, 10)$.

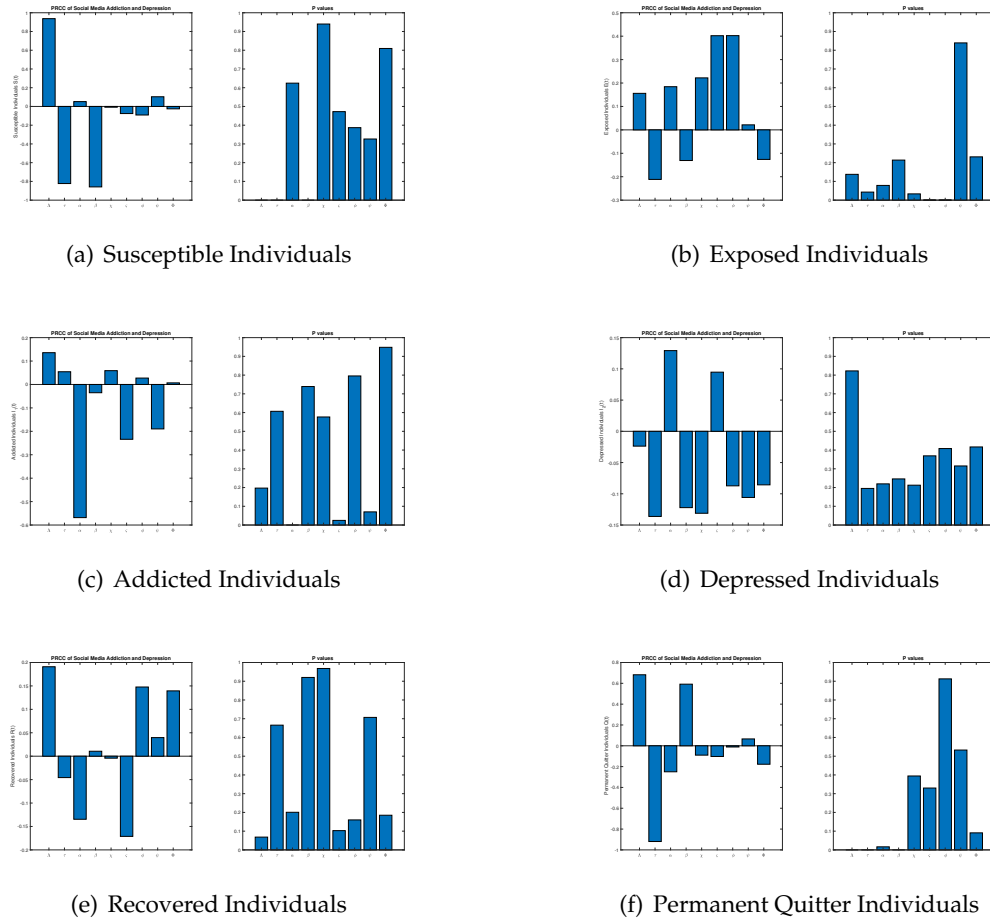


FIGURE 3.3: PRCC values representing the sensitivities of the model outputs.

3.5.1 Case 1: for $R_0 < 1$

Numerical interpenetration for $R_0 < 1$ is discussed here. Based on the numerical results of the SAMD model with RK-4 scheme with $N=1000$ and IC's, if we take $\Lambda = 0.5, \chi = 0.25, \phi = 0.8, \varsigma = 0.25, \Phi = 0.7, \beta = 0.01, \tau = 0.09; \alpha = 0.5, \psi = 0.0027$ then $R_0 = 0.8684 < 1$ obtained and the spread of disease (addiction and depression) stop which can be shown in Fig 3.4. In this case the DFE is $E_{q0}=(5, 0, 0, 0, 0, 0.5556)$. As time passes, we see that the populations of exposed and addicted people quickly expand and then gradually decline to zero. Depression transmission also increases along with the number of addicts. As more people recover from addiction and depression, there are more recovering populations. At the termination of the addict's transmission, the number of depressed people also quits, and the population of those who have recovered from addiction falls to zero. Additionally, the number of permanent quitters fluctuates over time. This is due to the variations in the number of susceptible and recovered populations. As the number of depressed individuals decreases with the decrease of addicted individuals, the permanent quitter increases until a stable equilibrium is reached at 0.5556. This variation is due to some individuals rejoining the exposed class after recovery, and those individuals who leave the treatment of depression or need a second dose moved toward the exposed and/or addicted class. On the other side, infected people (addicted and depressed) decreased, which, along with the decrease in exposed and permanent quitters, once again increased the number of high-quality people. This could be the result of infected persons (addicted and depressed) getting well, or new people joining the susceptible population who are not addicted or depressed and are balanced and stable at 5.

3.5.2 Case 2: for $R_0 > 1$

In this section, we discussed the numerical interpenetration for $R_0 > 1$. The numerical results of the SAMD model with RK-4 scheme with $N=1000$ and ICs are shown in Fig 3.5. If we take $\Lambda = 0.60556, \chi = 0.26104, \phi = 0.89339, \varsigma = 0.22874, \varphi = 0.80142, \beta = 0.010014, \tau = 0.095, \alpha = 0.47155, \psi = 0.002909$ then $R_0 = 1.3372 > 1$ obtained and the EEP is $E_{q1}=(4.3123, 0.5588, 0.1799, 0.1055, 0.2013, 1.0055)$. Fig 3.5 indicates that if $R_0 > 1$, there is a sharp initial increase in exposed and addicted individuals. Once the addiction reaches its peak, the number of exposed and addicted individuals stabilizes at 0.5588 and 0.1799, respectively. Due to the rapid increase in addiction, the depressed individual also increased and gradually decreased and stabilized at 0.1055. Also, as the decrease of exposed individuals decreased along with addicted and depressed individuals, the recovered individuals fluctuated over time and stabilized at 0.2013. With the treatment of addicted and depressed people, permanent quitters also increase and gradually decrease, tending to a stable point of 1.0055. Conversely, as the exposed and permanent quitter increases, the susceptible individual decreases and then stable with a slight increase at 4.3123.

3.6 Fractional derivatives

Starting with the integer-order derivative, consider $f \in \mathcal{C}^n[0, T]$ and $n \in \mathbb{N}$. The derivative of the n -th order is

$$\frac{d^n f(t)}{dt^n} = f^{(n)}(t).$$

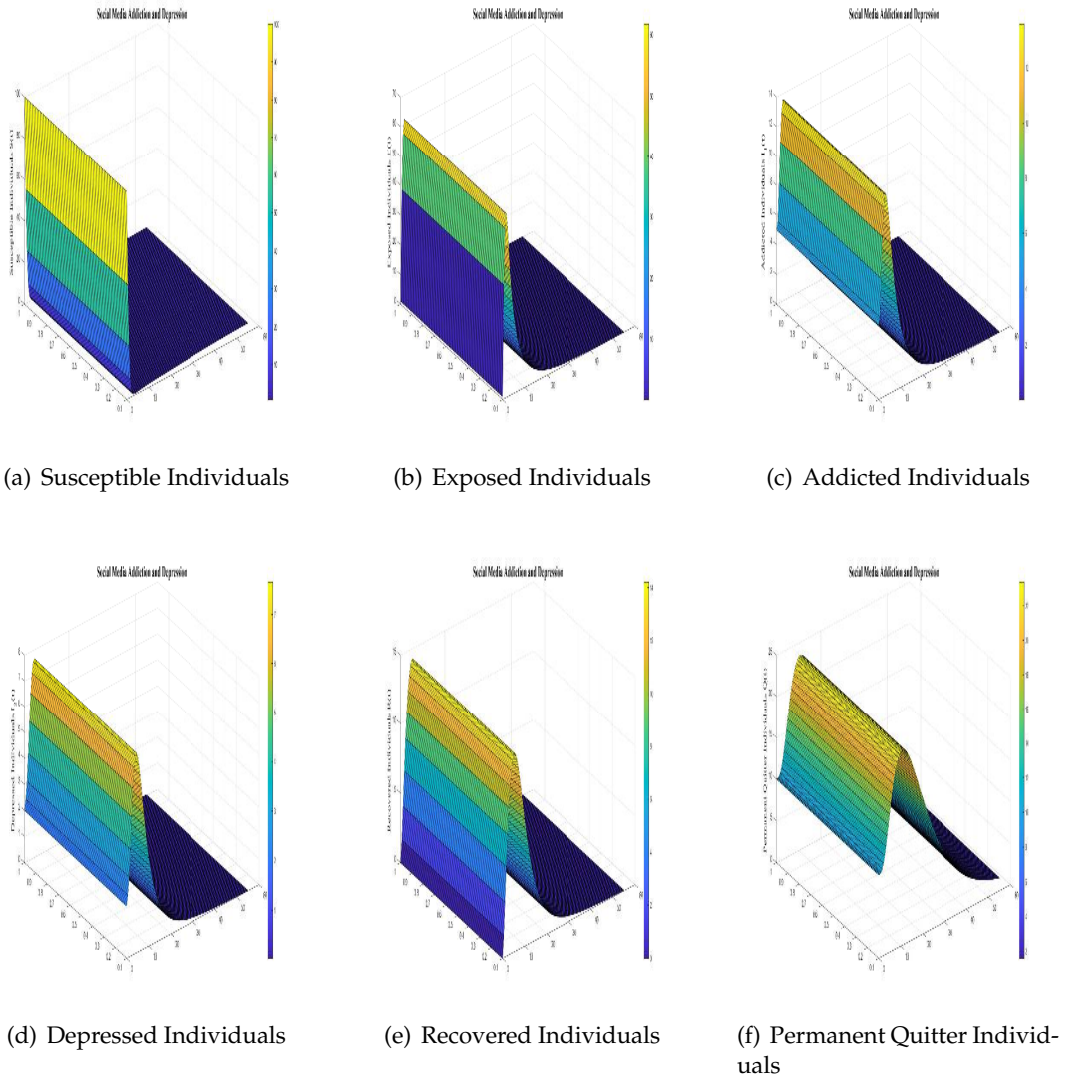


FIGURE 3.4: Surface plots of the solution of all classes of the social media addiction and depression (SMAD) model obtained by the RK-4 scheme for E_{q0} with initial conditions and step size $h = 0.001$. The numerical scheme converges to the true equilibrium point E_{q0} for $R_0 < 1$.

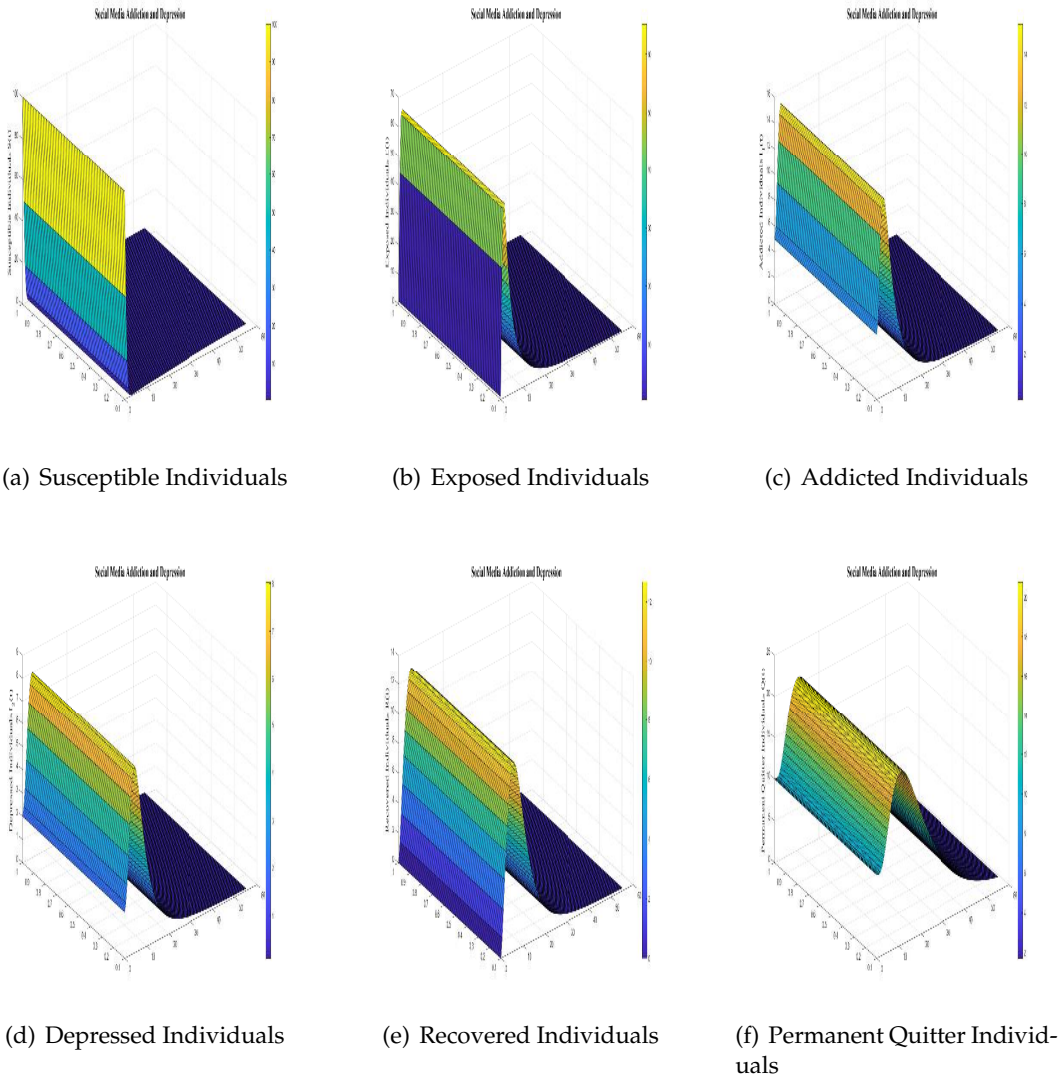


FIGURE 3.5: Surface plots of the solution of all classes of the social media addiction and depression (SMAD) model obtained by the RK-4 scheme for E_{q1} with initial conditions and step size $h = 0.001$. The numerical scheme converges to the true equilibrium point E_{q1} for $R_0 > 1$.

The Riemann-Liouville fractional integral with order $\beta > 0$ is described as

$$(I_t^\beta f)(t) = \frac{1}{\Gamma(\beta)} \int_0^t (t - \tau)^{\beta-1} f(\tau) d\tau.$$

This operator is a generalization of repetitive integer-order integration. To convert the integer derivative to a fractional order α fulfilling $n - 1 < \alpha < n$, write

$$\alpha = n - \beta, \quad \beta = n - \alpha \in (0, 1].$$

Use the fractional integral $I_t^{n-\alpha}$ to compute the integer-order derivative

$${}^C D_t^\alpha f(t) := I_t^{n-\alpha} \left(f^{(n)}(t) \right).$$

This choice preserves classical initial conditions. We express the integral version explicitly with the notion of $I_t^{n-\alpha}$, we get

$$\begin{aligned} {}^C D_t^\alpha f(t) &= \frac{1}{\Gamma(n-\alpha)} \int_0^t (t - \tau)^{n-\alpha-1} f^{(n)}(\tau) d\tau \\ &= \frac{1}{\Gamma(n-\alpha)} \int_0^t \frac{f^{(n)}(\tau)}{(t - \tau)^{\alpha-n+1}} d\tau, \quad n - 1 < \alpha < n. \end{aligned}$$

This is precisely the Caputo fractional derivative. Assume $\alpha \rightarrow n$, then $n - \alpha \rightarrow 0$, and since

$$\lim_{\beta \rightarrow 0^+} I_t^\beta g(t) = g(t),$$

we achieve

$$\lim_{\alpha \rightarrow n} {}^C D_t^\alpha f(t) = f^{(n)}(t),$$

defining that the Caputo derivative is actual modification of the integer-order derivative.

Special case $0 < \alpha < 1$, for $n = 1$,

$${}^C D_t^\alpha f(t) = I_t^{1-\alpha}(f'(t)) = \frac{1}{\Gamma(1-\alpha)} \int_0^t \frac{f'(\tau)}{(t - \tau)^\alpha} d\tau.$$

For this reason, Caputo derivatives are favored in applied PDE/ODE simulations, control structures, and epidemic models. We will define some related concepts about the fractional differential equation, including the Riemann–Liouville fractional (R-LF) integral, Caputo fractional derivative definition with order $\aleph$.

Definition 3.6. The left and Right R-LF integral of a function f is defined as

$$\begin{aligned} {}_a R_t^\aleph f(t) &= \frac{1}{\Gamma(\aleph)} \int_a^t (t - u)^{\aleph-1} f(u) du, \\ {}_t R_b^\aleph f(t) &= \frac{1}{\Gamma(\aleph)} \int_t^b (t - u)^{\aleph-1} f(u) du. \end{aligned}$$

Definition 3.7. The Right and Left Caputo Fractional derivative of order $n - 1 < \aleph < n \in \mathbb{Z}_+$ are define as

$${}_a^C D_t^\aleph g(t) = \frac{(-1)^\aleph}{\Gamma(n - \aleph)} \int_t^b (u - t)^{n-\aleph-1} g^{(n)}(u) du,$$

$${}^C D_b^\aleph g(t) = \frac{(-1)^n}{\Gamma(n-\aleph)} \int_a^t (u-t)^{n-\aleph-1} g^n(u) du.$$

Definition 3.8. The left Caputo derivative with Laplace transformation is denoted and defined as

$$\mathcal{L} \left\{ {}^C D_t^\aleph g(t) \right\} = s^\aleph F(s) - \sum_{n=0}^{k-1} g^n(0) s^{\aleph-n-1}.$$

Definition 3.9. The Laplace transformation of function $t^{v-1} E_{u,v}(\pm \mu t^u)$ is define as

$$\mathcal{L} \left\{ t^{v-1} E_{u,v}(\pm \mu t^u) \right\} = \frac{s^{u-v}}{s^u \mp \mu}.$$

where $E_{u,v}(t)$ is the two-parameter Mittag-Leffler (ML) function with $u, v > 0$.

Definition 3.10. Let $E_{u,v}(Z)$ be the Mittag-Leffler (ML) function. Then

$$E_{u,v}(Z) = Z.E_{u,u+v}(Z) + \frac{1}{\Gamma(v)}.$$

3.7 Model with Caputo derivative

Here, we define a fractional-order model of social media addiction and depression. The model consists on six compartments of the population namely, Susceptible population (S) that are not addicted yet, Exposed individuals (E) are the people who use social media less frequently but are not addicted yet, Addicted individuals (A) are those people who are addicted to social media and spent most of their time on it, Depressed individuals (D) are the people those are in depression because of the SMA, Recovered individuals (R) the people who recovered from addiction, and Quitter individuals (Q) are those who give up and permanently stop using social media. The parameters involved in the system are presented in Table 3.2. The following epidemiological presumptions were made when developing the model. Fig 3.6 represents the flow diagram of the proposed model. The Caputo fractional model of (5.1) is given as:

$$\begin{cases} {}^C D_t^\aleph S(t) = a_1 + a_2 a_3 R(t) - a_{12} a_7 A(t) S(t) - (a_6 + a_4) S(t), \\ {}^C D_t^\aleph E(t) = a_{12} a_7 A(t) S(t) - (a_9 + a_4) E(t), \\ {}^C D_t^\aleph A(t) = a_{14} a_9 E(t) - (a_4 + a_5 + a_{11}) A(t), \\ {}^C D_t^\aleph D(t) = a_5 A(t) + a_{11} (1 - a_{10}) A(t) - (a_8 + a_{13} + a_4) D(t), \\ {}^C D_t^\aleph R(t) = (1 - a_{14}) a_9 E(t) + a_8 D(t) + a_{11} a_{10} A(t) - (a_4 + a_3) R(t), \\ {}^C D_t^\aleph Q(t) = a_6 S(t) + (1 - a_2) a_3 R(t) - a_4 Q(t). \end{cases} \quad (3.22)$$

Initial conditions (ICs) are given as follows

$$S(t=0) \geq 0, E(t=0) \geq 0, A(t=0) \geq 0, D(t=0) \geq 0, R(t=0) \geq 0, Q(t=0) \geq 0.$$

The fractional term is defined by the Caputo fractional derivative as

$${}^C D_t^\aleph p(t) = \frac{1}{\Gamma(n-\aleph)} \int_0^t \frac{p^{(n)}(\xi) d\xi}{(t-\xi)^{\aleph+1-n}}.$$

where $\aleph$ is the order of the derivative, $\Gamma(\cdot, \cdot)$ is the Gamma function, and the kernel $(t - \xi)^{\aleph+1-n}$ describes the non-locality of the fractional derivative with integral taking in $[0, t]$. This formulation emphasizes that the Caputo derivative considers the function's entire history from time 0 to t , making it especially suitable for initial value problems. In this model, $N(t)$ shows the all population and is given by

$$N(t) = S(t) + E(t) + A(t) + D(t) + R(t) + Q(t).$$

The traditional SIR or SEIR model, which focuses on media effects, has been instrumental in controlling disease transmission during epidemics. It highlights the importance of proper media strategies, daily disease updates, and government prevention measures in preventing disease spread. However, a new model has been proposed to examine the relationship between social media addiction and depression, highlighting the behavioral dynamics in communities. The model explains how excessive or negative use of social media can lead to addictive behaviors, causing depression symptoms in adults, often linked to trolling, internet scams, and cyberbullying. The model explicitly models depression triggered by media exposure, highlighting the gradual growth of addiction and depression over time. This mathematical paradigm offers a more biologically and psychologically sound basis for comprehending and forecasting the intricate relationships between depression and social media addiction in contemporary populations. The model is well structured

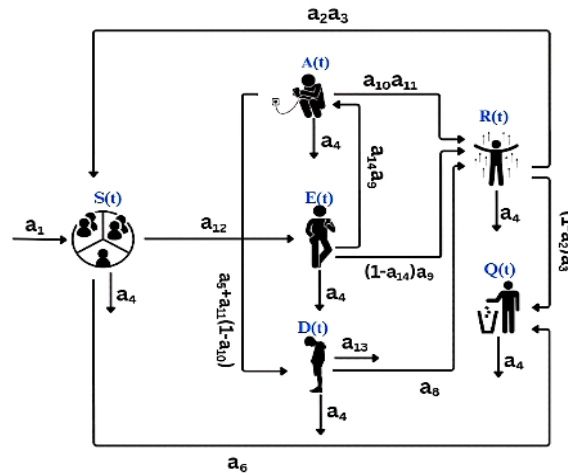


FIGURE 3.6: Compartmental diagram of transmission of social media addiction and depression dynamics

to explore the interaction of these physiological states through the Caputo fraction derivative, but there are some limitations that we discussed here. This model ignores the socio-economic factors, which make the model less realistic. The model assumes uniform mixing, all parameters are fixed, and the time inversion rate ignores the control behavior like social media awareness, campaigns, or education activities, which we will discuss in the future extended model. Real data is not available, but we try to validate the model by considering parameter values from the literature, which may cause uncertainty in the data. Sensitivity analyses are carried out to evaluate the impact of parameter adjustments on results, improving consistency. Due to the literature-driven validation, the model can be used as a first tool for exploring hypotheses and strategy analysis until more specific data becomes accessible.

TABLE 3.2: Parameter values used in the proposed model.

Symbol	Description	Values
a_1	Recruitment rate of susceptible individuals	0.5
a_2	Proportion of recovered individuals susceptible to social media addiction-driven depression	0.35
a_3	Individuals that leave recovered class	0.4
a_4	Natural death rate	0.05–0.25
a_5	Rate at which media influence causes depression	0.3–0.5
a_6	Susceptible people that leave using social media	0.01
a_7	Contact rate causing transition to exposed class	0.25
a_8	Recovery rate from depression after treatment	0.7
a_9	Individuals that leave exposed class	0.25
a_{10}	Probability that treatment is successful	0.8
a_{11}	Rate at which people leave treatment	0.0027
a_{12}	Rate at which addicted individuals become susceptible	0.1–0.8
a_{13}	Induced death rate	0.01
a_{14}	Proportion of exposed moving to addicted class	0.7

3.7.1 Equilibrium points

This section aims to ascertain the steady states of the suggested model.

$$\begin{cases}
 a_1 + a_2 a_3 R(t) - a_{12} a_7 A(t) S(t) - (a_6 + a_4) S(t) & = 0, \\
 a_{12} a_7 A(t) S(t) - (a_9 + a_4) E(t) & = 0, \\
 a_{14} a_9 E(t) - (a_4 + a_5 + a_{11}) A(t) & = 0, \\
 a_5 A(t) + a_{11} (1 - a_{10}) A(t) - (a_8 + a_{13} + a_4) D(t) & = 0, \\
 (1 - a_{14}) a_9 E(t) + a_8 D(t) + a_{11} a_{10} A(t) - (a_4 + a_3) R(t) & = 0, \\
 a_6 S(t) + (1 - a_2) a_3 R(t) - a_4 Q(t) & = 0.
 \end{cases} \quad (3.23)$$

When we solve the system of equations (3.23), then the two different equilibrium points are obtained. The Maple software has been used to obtain the equilibrium points. The first equilibrium point is disease disease-free equilibrium (DFE) point

$$\mathbb{E}_0 = (S_0, E_0, A_0, D_0, R_0, Q_0) = \left(\frac{a_1}{a_6 + a_4}, 0, 0, 0, 0, \frac{a_6 a_1}{(a_6 + a_4) a_4} \right).$$

of the model when $R_0 < 1$, this represents that there is no disease and only the susceptible population exists. The Endemic equilibrium (EEP) point of the system (3.23) is

$$\mathbb{E}_1 = (S_1, E_1, A_1, D_1, R_1, Q_1)$$

$$\begin{aligned}
S_1 &= \frac{a_1 (a_3 + a_4) - A_1 a_2 a_3 (-a_{10} a_{11} a_{14} (a_{13} + a_4 + a_8) + l_4 - l_1)}{a_{14} (a_{13} + a_4 + a_8) (a_3 + a_4) (a_4 + a_6 + A_1 a_{12} a_7)}, \\
E_1 &= \frac{A_1 (a_{11} + a_4 + a_5)}{(a_{14} a_9)}, \\
D_1 &= \frac{A_1 (a_{11} - a_{10} a_{11} + a_5)}{a_{13} + a_4 + a_8}, \\
R_1 &= \frac{\left(a_{10} a_{11} a_{14} (a_{13} + a_4 + a_8) - (a_{14} - 1) (a_{11} + a_4 + a_5) + (a_{11} - a_{10} a_{11} + a_5) a_8 \right) A_1}{a_{14} (a_{13} + a_4 + a_8) (a_3 + a_4)}, \\
Q_1 &= \frac{a_1 a_6 - (A_1 a_3 ((a_2 - 1) a_4 - a_6 + A a_{12} (a_2 - 1) a_7) l_2)}{a_{14} (a_3 + a_4) (a_{13} + a_4 + a_8) ((a_4^2 + a_4 a_6 + A_1 a_4 a_{12} a_7))}, \\
A_1 &= \frac{(a_3 + a_4) (a_{13} + a_4 + a_8) (- (a_4 + a_9) (a_{11} + a_4 + a_5) (a_4 + a_6) + a_1 a_{12} a_{14} a_7 a_9)}{a_{12} a_7 (a_4 (a_3 + a_4) (a_{11} + a_4 + a_5) (a_{13} + a_4 + a_8) + l_3 a_9)}.
\end{aligned} \tag{3.24}$$

where

$$\begin{aligned}
l_1 &= (a_{11} - a_{10} a_{11} + a_5) a_8 a_{14} \\
l_2 &= ((a_{13} + a_4) (a_{11} + (a_{10} - 1) a_{11} a_{14} + a_4 + a_5 - a_{14} (a_4 + a_5)) \\
&\quad + (a_{11} + a_4 - a_{14} a_4 + a_5) a_8) \\
l_3 &= (-a_{11} ((-1 + a_2 + (-1 + a_{10}) a_{14} a_2) a_3 - a_4) (a_{13} + a_4) + (a_{13} + a_4) \\
&\quad (a_3 + (-1 + a_{14}) a_2 a_3 + a_4) (a_4 + a_5) + a_{11} (a_3 - a_2 a_3 + a_4) a_8 + (a_4 (a_4 + a_5) + \\
&\quad a_3 (a_4 + (-1 + a_{14}) a_2 a_4 + a_5 - a_2 a_5)) a_8) \\
l_4 &= (a_{14} - 1) (a_{11} + a_4 + a_5) (a_{13} + a_4 + a_8)
\end{aligned}$$

The model's basic reproduction number (R_0) of the model is significant in analyzing the stability of the proposed model. By next-generation matrix technique we defined the value of R_0 and the system (3.22) as

$$R_0 = \frac{(a_1 a_7 a_9 a_{12} a_{14})}{(a_4 + a_6)(a_4 + a_9)(a_4 + a_5 + a_{11})}. \tag{3.25}$$

3.7.2 Stability Analysis

The stability analysis of the model is discussed in this section using the following results.

Theorem 3.11. *The disease-free equilibrium point (DEF) is asymptotically stable if $R_0 < 1$.*

Proof. Using the disease free equilibrium point $\mathbb{E}_0$ to find the Jacobin matrix of the model (3.22) we obtained the following matrix

$$J = \begin{bmatrix} K_1 & 0 & -a_{12} a_7 S_0 & 0 & a_2 a_3 & 0 \\ a_{12} a_7 A_0 & -a_9 - a_4 & a_{12} a_7 S_0 & 0 & 0 & 0 \\ 0 & a_{14} a_9 & K_2 & 0 & 0 & 0 \\ 0 & 0 & K_3 & K_4 & 0 & 0 \\ 0 & (1 - a_{14}) a_9 & a_{11} a_{10} & -a_8 & -a_4 - a_3 & 0 \\ a_6 & 0 & 0 & 0 & (1 - a_2) a_3 & -a_4 \end{bmatrix}$$

where

$$K_1 = -a_{12}a_7A_0 - a_4 - a_6, \quad K_2 = -a_4 - a_5 - a_{11}, \quad K_3 = a_{11}(1 - a_{10}) + a_5,$$

$$K_4 = -a_8 - a_{13} - a_4.$$

The value of the Jacobian at the DFE point is given below

$$J_0 = \begin{bmatrix} -a_4 - a_6 & 0 & -\frac{a_{12}a_7a_1}{a_6+a_4} & 0 & a_2a_3 & 0 \\ 0 & -a_9 - a_4 & \frac{a_{12}a_7a_1}{a_6+a_4} & 0 & 0 & 0 \\ 0 & a_{14}a_9 & K_2 & 0 & 0 & 0 \\ 0 & 0 & K_3 & K_4 & 0 & 0 \\ 0 & -(-1 + a_{14})a_9 & a_{11}a_{10} & -a_8 & -a_4 - a_3 & 0 \\ a_6 & 0 & 0 & 0 & -(-1 + a_2)a_3 & -a_4 \end{bmatrix}$$

Using Maple, the eigenvalues of the above matrix, by converting to an upper-triangle matrix through row echelon form is are as follows:

$$\lambda_1 = -a_4$$

$$\lambda_2 = -(a_9 + a_4)$$

$$\lambda_3 = -(a_8 + a_{13} + a_4)$$

$$\lambda_4 = -(a_4 + a_6)$$

$$\lambda_5 = -(a_4 + a_3)$$

$$\lambda_6 = \frac{\left\{ \begin{aligned} &a_1a_7a_9a_{12}a_{14} - a_4^2(a_4 + a_5 + a_6 + a_9 + a_{11}) - a_4(a_5a_6 + a_5a_9 + a_6a_9 + a_6a_{11} + a_9a_{11}) \\ &- a_6a_9(a_5 + a_{11}) \end{aligned} \right\}}{(a_4 + a_6)(a_4 + a_9)}.$$

All eigenvalues must be negative for the stability of DFE. Taking λ_6 , we rewrite it as

$$\lambda_6 = \frac{\mathbf{R}_0(a_4 + a_6)(a_4 + a_9)(a_4 + a_5 + a_{11}) - (a_9 + a_4)(a_4 + a_6)(a_4 + a_5 + a_{11})}{(a_4 + a_6)(a_4 + a_9)}$$

$$\lambda_6 = (\mathbf{R}_0 - 1)(a_4 + a_5 + a_{11})$$

Since all eigenvalues are negative. So, one can observe that the disease-free equilibrium point (DFE) is asymptotically stable. $\square$

Theorem 3.12. *The endemic equilibrium point (EEP) is asymptotically stable if $R_0 > 1$.*

Proof. The endemic equilibrium point $\mathbb{E}_1$ of model (3.22) is

$$\mathbb{E}_1 = (S_1, E_1, A_1, D_1, R_1, Q_1).$$

For the Jacobian matrix, we use the system of equations of the model at the first equilibrium point E_1 , and we get the following matrix

$$J = \begin{bmatrix} K_1 & 0 & -a_{12}a_7S_1 & 0 & a_2a_3 & 0 \\ a_{12}a_7A_1 & -a_9 - a_4 & a_{12}a_7S_1 & 0 & 0 & 0 \\ 0 & a_{14}a_9 & K_2 & 0 & 0 & 0 \\ 0 & 0 & K_3 & K_4 & 0 & 0 \\ 0 & (1 - a_{14})a_9 & a_{11}a_{10} & -a_8 & -a_4 - a_3 & 0 \\ a_6 & 0 & 0 & 0 & (1 - a_2)a_3 & -a_4 \end{bmatrix}$$

where

$$K_1 = -a_{12}a_7A_1 - a_4 - a_6, \quad K_2 = -a_4 - a_5 - a_{11}, \quad K_3 = a_{11}(1 - a_{10}) + a_5, \\ K_4 = -a_8 - a_{13} - a_4.$$

The value of the Jacobian at the EEP point is given below

$$J_1 = \begin{bmatrix} M_1 & 0 & M_2 & 0 & a_2a_3 & 0 \\ M_3 & -a_9 - a_4 & M_4 & 0 & 0 & 0 \\ 0 & a_{14}a_9 & M_5 & 0 & 0 & 0 \\ 0 & 0 & M_6 & M_7 & 0 & 0 \\ 0 & M_8 & a_{11}a_{10} & -a_8 & M_9 & 0 \\ a_6 & 0 & 0 & 0 & M_{10} & -a_4 \end{bmatrix}$$

where

$$M_1 = -(a_{12}a_7A_1 + a_4 + a_6), \quad M_2 = -a_{12}a_7S_1, \quad M_3 = a_{12}a_7A_1, \\ M_4 = a_{12}a_7S_1, \quad M_5 = -(a_4 + a_5 + a_{11}), \quad M_6 = a_{11}(1 - a_{10}) + a_5, \\ M_7 = -(a_8 + a_{13} + a_4), \quad M_8 = (1 - a_{14})a_9, \quad M_9 = -(a_4 + a_3) \\ M_{10} = (1 - a_2)a_3.$$

It is clear from the matrix that we have two eigenvalues as follows:

$$\lambda_1 = -a_4, \quad \lambda_2 = M_1 < 0.$$

Now we write the renaming 4×4 matrix to find the remaining eigenvalues.

$$C = \begin{bmatrix} -a_9 - a_4 & M_4 & 0 & 0 \\ a_{14}a_9 & M_5 & 0 & 0 \\ 0 & M_6 & M_7 & 0 \\ M_8 & a_{11}a_{10} & -a_8 & M_9 \end{bmatrix}$$

The characteristic polynomial is given as

$$(\lambda - M_9)(\lambda - M_7)(\lambda^2 + (-M_5 + a_9 + a_4)\lambda + M_5(-a_9 - a_4) - a_{14}a_9M_4). \quad (3.26)$$

The other two eigenvalues are

$$\lambda_3 = M_7 < 0, \quad \lambda_4 = M_9 < 0.$$

From Eq (3.26) we can define the quadratic polynomial as

$$\lambda^2 + A\lambda + B = 0.$$

Where

$$\begin{aligned} A &= +a_9 + a_4 - M_5, \\ B &= -M_5(a_9 + a_4) - a_{14}a_9M_4. \end{aligned}$$

Taking values of A_1 and S_1 from (3.24) shows that by applying Routh-Hurwitz criteria, the two roots are negative because $A > 0, B > 0$ if $M_4 < 0$. Since all eigenvalues are negative. So, the endemic equilibrium point (EEP) is asymptotically stable. $\square$

3.8 Existence and uniqueness of model

The reliability and stability of the proposed model (3.22) are demonstrated by the model's convergence to a single, unique solution. This is demonstrated by the Banach contraction theorem, which mathematically supports the uniqueness of the solution in the context of this study [280, 273]. Some significant measures, such as Lipschitz condition, uniqueness, and existence of the (3.22) will be addressed in this section. The utilization of the Caputo operator on (3.22), with $\aleph$.

$$\left\{ \begin{aligned} {}^C D_t^\aleph S(t) &= \frac{1}{\Gamma(1-\aleph)} \int_0^t (t-u)^{-\aleph} S'(u) du, \\ {}^C D_t^\aleph E(t) &= \frac{1}{\Gamma(1-\aleph)} \int_0^t (t-u)^{-\aleph} E'(u) du, \\ {}^C D_t^\aleph A(t) &= \frac{1}{\Gamma(1-\aleph)} \int_0^t (t-u)^{-\aleph} A'(u) du, \\ {}^C D_t^\aleph D(t) &= \frac{1}{\Gamma(1-\aleph)} \int_0^t (t-u)^{-\aleph} D'(u) du, \\ {}^C D_t^\aleph R(t) &= \frac{1}{\Gamma(1-\aleph)} \int_0^t (t-u)^{-\aleph} R'(u) du, \\ {}^C D_t^\aleph Q(t) &= \frac{1}{\Gamma(1-\aleph)} \int_0^t (t-u)^{-\aleph} Q'(u) du. \end{aligned} \right. \quad (3.27)$$

Firstly, let us assume five kernel values for the simplification.

$$\left\{ \begin{aligned} {}^C D_t^\aleph S(t) &= \Pi_1(t, S), \\ {}^C D_t^\aleph E(t) &= \Pi_2(t, E), \\ {}^C D_t^\aleph A(t) &= \Pi_3(t, A), \\ {}^C D_t^\aleph D(t) &= \Pi_4(t, D), \\ {}^C D_t^\aleph R(t) &= \Pi_5(t, R), \\ {}^C D_t^\aleph Q(t) &= \Pi_6(t, Q). \end{aligned} \right. \quad (3.28)$$

After the implementation of the Caputo operator, we get

$$\left\{ \begin{array}{l} S(t) - S(0) = \Theta(\aleph) \Pi_1(t, S) + \Omega(\aleph) \int_0^t \Pi_1(u, S) du, \\ E(t) - E(0) = \Theta(\aleph) \Pi_2(t, E) + \Omega(\aleph) \int_0^t \Pi_2(u, E) du, \\ A(t) - A(0) = \Theta(\aleph) \Pi_3(t, A) + \Omega(\aleph) \int_0^t \Pi_3(u, A) du, \\ D(t) - D(0) = \Theta(\aleph) \Pi_4(t, D) + \Omega(\aleph) \int_0^t \Pi_4(u, D) du, \\ R(t) - R(0) = \Theta(\aleph) \Pi_5(t, R) + \Omega(\aleph) \int_0^t \Pi_5(u, R) du, \\ Q(t) - Q(0) = \Theta(\aleph) \Pi_6(t, Q) + \Omega(\aleph) \int_0^t \Pi_6(u, Q) du. \end{array} \right. \quad (3.29)$$

Where $\Omega(\aleph)$ and $\Theta(\aleph)$ are the both positive constants. Now we will prove the Lipschitz condition for the Caputo model.

Theorem 3.13. $\Pi_1(t, S), \Pi_2(t, E), \Pi_3(t, A), \Pi_4(t, D), \Pi_5(t, R)$ and $\Pi_6(t, Q)$ are satisfy the Lipschitz condition.

Proof. Let's take S and S^* as the two functions for the first kernel Π_1 . Then the corresponding norm is defined as

$$\begin{aligned} \left\| \Pi_1(t, S) - \Pi_1(t, S^*) \right\| &\leq \left\| \left(a_1 + a_2 a_3 R(t) - a_{12} a_7 A(t) S(t) - (a_6 + a_4) S(t) \right) \right. \\ &\quad \left. - \left(a_1 + a_2 a_3 R(t) - a_{12} a_7 A(t) S^*(t) - (a_6 + a_4) S^*(t) \right) \right\|. \end{aligned} \quad (3.30)$$

$$\left\| \Pi_1(t, S) - \Pi_1(t, S^*) \right\| \leq a_{12} a_7 A(t) \left\| S(t) - S^*(t) \right\| + (a_6 + a_4) \left\| S(t) - S^*(t) \right\|. \quad (3.31)$$

$$\left\| \Pi_1(t, S) - \Pi_1(t, S^*) \right\| \leq \left(a_{12} a_7 A(t) + (a_6 + a_4) \right) \left\| S(t) - S^*(t) \right\|. \quad (3.32)$$

$$\left\| \Pi_1(t, S) - \Pi_1(t, S^*) \right\| \leq \mathcal{K}_1 \left\| S(t) - S^*(t) \right\|. \quad (3.33)$$

Where $\mathcal{K}_1 = a_{12} a_7 A(t) + (a_6 + a_4)$ in which $A(t)$ is bounded and other are non negative parameters.

Similarly, you can easily prove that

$$\left\{ \begin{array}{l} \left\| \Pi_2(t, E) - \Pi_2(t, E^*) \right\| \leq \mathcal{K}_2 \left\| E(t) - E^*(t) \right\|, \\ \left\| \Pi_3(t, A) - \Pi_3(t, A^*) \right\| \leq \mathcal{K}_3 \left\| A(t) - A^*(t) \right\|, \\ \left\| \Pi_4(t, D) - \Pi_4(t, D^*) \right\| \leq \mathcal{K}_4 \left\| D(t) - D^*(t) \right\|, \\ \left\| \Pi_5(t, R) - \Pi_5(t, R^*) \right\| \leq \mathcal{K}_5 \left\| R(t) - R^*(t) \right\|, \\ \left\| \Pi_6(t, Q) - \Pi_6(t, Q^*) \right\| \leq \mathcal{K}_6 \left\| Q(t) - Q^*(t) \right\|. \end{array} \right. \quad (3.34)$$

□

3.8.1 Existence of the solution

We are going to show that the under discussion Caputo model (3.22) has at least one solution in this section. As a result, the following is the recursive formula for (3.28):

$$\left\{ \begin{array}{l} S_n(t) = \Theta(\aleph) \Pi_1(t, S_{n-1}) + \Omega(\aleph) \int_0^t \Pi_1(u, S_{n-1}) du, \\ E_n(t) = \Theta(\aleph) \Pi_2(t, E_{n-1}) + \Omega(\aleph) \int_0^t \Pi_2(u, E_{n-1}) du, \\ A_n(t) = \Theta(\aleph) \Pi_3(t, A_{n-1}) + \Omega(\aleph) \int_0^t \Pi_3(u, A_{n-1}) du, \\ D_n(t) = \Theta(\aleph) \Pi_4(t, D_{n-1}) + \Omega(\aleph) \int_0^t \Pi_4(u, D_{n-1}) du, \\ R_n(t) = \Theta(\aleph) \Pi_5(t, R_{n-1}) + \Omega(\aleph) \int_0^t \Pi_5(u, R_{n-1}) du, \\ Q_n(t) = \Theta(\aleph) \Pi_6(t, Q_{n-1}) + \Omega(\aleph) \int_0^t \Pi_6(u, Q_{n-1}) du. \end{array} \right. \quad (3.35)$$

$$\left\{ \begin{array}{l} \Omega_{1n} = S_n(t) - S_{n-1}(t), \\ \Omega_{2n} = E_n(t) - E_{n-1}(t), \\ \Omega_{3n} = A_n(t) - A_{n-1}(t), \\ \Omega_{4n} = D_n(t) - D_{n-1}(t), \\ \Omega_{5n} = R_n(t) - R_{n-1}(t), \\ \Omega_{6n} = Q_n(t) - Q_{n-1}(t). \end{array} \right. \quad (3.36)$$

where

$$\left\{ \begin{array}{l} S_n(t) - S_{n-1}(t) = \Theta(\aleph) (\Pi_1(t, S_{n-1}) - \Pi_1(t, S_{n-2})) + \\ \quad \Omega(\aleph) \int_0^t (\Pi_1(u, S_{n-1}) - \Pi_1(u, S_{n-2})) du, \\ E_n(t) - E_{n-1}(t) = \Theta(\aleph) (\Pi_2(t, E_{n-1}) - \Pi_2(t, E_{n-2})) + \\ \quad \Omega(\aleph) \int_0^t (\Pi_2(u, E_{n-1}) - \Pi_2(u, E_{n-2})) du, \\ A_n(t) - A_{n-1}(t) = \Theta(\aleph) (\Pi_3(t, A_{n-1}) - \Pi_3(t, A_{n-2})) + \\ \quad \Omega(\aleph) \int_0^t (\Pi_3(u, A_{n-1}) - \Pi_3(u, A_{n-2})) du, \\ D_n(t) - D_{n-1}(t) = \Theta(\aleph) (\Pi_4(t, D_{n-1}) - \Pi_4(t, D_{n-2})) + \\ \quad \Omega(\aleph) \int_0^t (\Pi_4(u, D_{n-1}) - \Pi_4(u, D_{n-2})) du, \\ R_n(t) - R_{n-1}(t) = \Theta(\aleph) (\Pi_5(t, R_{n-1}) - \Pi_5(t, R_{n-2})) + \\ \quad \Omega(\aleph) \int_0^t (\Pi_5(u, R_{n-1}) - \Pi_5(u, R_{n-2})) du, \\ Q_n(t) - Q_{n-1}(t) = \Theta(\aleph) (\Pi_6(t, Q_{n-1}) - \Pi_6(t, Q_{n-2})) + \\ \quad \Omega(\aleph) \int_0^t (\Pi_6(u, Q_{n-1}) - \Pi_6(u, Q_{n-2})) du. \end{array} \right.$$

It is important to note that

$$\left\{ \begin{array}{l} \sum_{i=0}^n \Lambda_{1n} = S_n(t), \\ \sum_{i=0}^n \Lambda_{2n} = E_n(t), \\ \sum_{i=0}^n \Lambda_{3n} = A_n(t), \\ \sum_{i=0}^n \Lambda_{4n} = D_n(t), \\ \sum_{i=0}^n \Lambda_{5n} = R_n(t), \\ \sum_{i=0}^n \Lambda_{6n} = Q_n(t). \end{array} \right. \quad (3.37)$$

Taking the first Eq of system (3.36), Let we have

$$\begin{aligned} \Lambda_{1n} &= \left\| S_n(t) - S_{n-1}(t) \right\| \\ &= \left\| \Theta(\aleph) (\Pi_1(t, S_{n-1}) - \Pi_1(t, S_{n-2})) \right. \\ &\quad \left. + \Omega(\aleph) \int_0^t (\Pi_1(u, S_{n-1}) - \Pi_1(u, S_{n-2})) du \right\|. \end{aligned} \quad (3.38)$$

By applying the triangle inequality, we get

$$\begin{aligned} \|S_n(t) - S_{n-1}(t)\| &\leq \left\| \Theta(\aleph) (\Pi_1(t, S_{n-1}) - \Pi_1(t, S_{n-2})) \right. \\ &\quad \left. + \Omega(\aleph) \int_0^t (\Pi_1(u, S_{n-1}) - \Pi_1(u, S_{n-2})) du \right\|. \end{aligned} \quad (3.39)$$

$$\begin{aligned} \|S_n(t) - S_{n-1}(t)\| &\leq \Theta(\aleph) \left\| \Pi_1(t, S_{n-1}) - \Pi_1(t, S_{n-2}) \right\| \\ &\quad + \Omega(\aleph) \left\| \int_0^t (\Pi_1(u, S_{n-1}) - \Pi_1(u, S_{n-2})) du \right\|. \end{aligned} \quad (3.40)$$

From Eq (3.34), we indicate that kernel $\Pi_1(t, S)$ satisfy the Lipschitz condition. This implies that

$$\begin{aligned} \|S_n(t) - S_{n-1}(t)\| &\leq \Theta(\aleph) \mathcal{K}_1 \|S_{n-1} - S_{n-2}\| \\ &\quad + \Omega(\aleph) \mathcal{K}_1 \int_0^t \|S_{n-1}(u) - S_{n-2}(u)\| du. \end{aligned} \quad (3.41)$$

By using the Eq (3.37), we can write this as

$$\begin{aligned} \|\Lambda_{1n}\| &\leq \Theta(\aleph) \mathcal{K}_1 \|\Lambda_{1(n-1)}(t)\| \\ &\quad + \Omega(\aleph) \mathcal{K}_1 \int_0^t \|\Lambda_{1(n-1)}(u)\| du. \end{aligned} \quad (3.42)$$

Similarly, one can easily get the following results for other kernels:

$$\left\{ \begin{aligned} \|\Lambda_{2n}\| &\leq \Theta(\aleph) \mathcal{K}_2 \|\Lambda_{2(n-1)}(t)\| + \Omega(\aleph) \mathcal{K}_2 \int_0^t \|\Lambda_{2(n-1)}(u)\| du, \\ \|\Lambda_{3n}\| &\leq \Theta(\aleph) \mathcal{K}_3 \|\Lambda_{3(n-1)}(t)\| + \Omega(\aleph) \mathcal{K}_3 \int_0^t \|\Lambda_{3(n-1)}(u)\| du, \\ \|\Lambda_{4n}\| &\leq \Theta(\aleph) \mathcal{K}_4 \|\Lambda_{4(n-1)}(t)\| + \Omega(\aleph) \mathcal{K}_4 \int_0^t \|\Lambda_{4(n-1)}(u)\| du, \\ \|\Lambda_{5n}\| &\leq \Theta(\aleph) \mathcal{K}_5 \|\Lambda_{5(n-1)}(t)\| + \Omega(\aleph) \mathcal{K}_5 \int_0^t \|\Lambda_{5(n-1)}(u)\| du, \\ \|\Lambda_{6n}\| &\leq \Theta(\aleph) \mathcal{K}_6 \|\Lambda_{6(n-1)}(t)\| + \Omega(\aleph) \mathcal{K}_6 \int_0^t \|\Lambda_{6(n-1)}(u)\| du. \end{aligned} \right. \quad (3.43)$$

Theorem 3.14. *The analytical solution of model (3.22) exist if*

$$\Theta(\aleph) \mathcal{K}_i + \Omega(\aleph) \mathcal{K}_i \Lambda_{i,0} \leq 1 \quad \text{for} \quad i = 1, 2, \dots, 6 \quad (3.44)$$

Proof. Since the function $S(t)$, $E(t)$, $A(t)$, $D(t)$, $R(t)$ and $Q(t)$ are bounded and also satisfy the Lipschitz condition. So from recursive technique for Eq (3.42) and Eq (3.43), we have

$$\begin{aligned}
\| \Lambda_{1n} \| &\leq \| S(0) \| [\Theta(\aleph) \mathcal{K}_1 + \Omega(\aleph) \mathcal{K}_1 t]^n, \\
\| \Lambda_{2n} \| &\leq \| E(0) \| [\Theta(\aleph) \mathcal{K}_2 + \Omega(\aleph) \mathcal{K}_2 t]^n, \\
\| \Lambda_{3n} \| &\leq \| A(0) \| [\Theta(\aleph) \mathcal{K}_3 + \Omega(\aleph) \mathcal{K}_3 t]^n, \\
\| \Lambda_{4n} \| &\leq \| D(0) \| [\Theta(\aleph) \mathcal{K}_4 + \Omega(\aleph) \mathcal{K}_4 t]^n, \\
\| \Lambda_{5n} \| &\leq \| R(0) \| [\Theta(\aleph) \mathcal{K}_5 + \Omega(\aleph) \mathcal{K}_5 t]^n, \\
\| \Lambda_{6n} \| &\leq \| Q(0) \| [\Theta(\aleph) \mathcal{K}_6 + \Omega(\aleph) \mathcal{K}_6 t]^n.
\end{aligned} \tag{3.45}$$

Consequently, the system is continuous and has a solution. We now demonstrate how the functions mentioned above provide a solution for the model (3.22), considering.

$$\begin{cases}
S(t) - S(0) = S_n(t) - A_{1n}(t), \\
E(t) - E(0) = E_n(t) - A_{2n}(t), \\
A(t) - A(0) = A_n(t) - A_{3n}(t), \\
D(t) - D(0) = D_n(t) - A_{4n}(t), \\
R(t) - R(0) = R_n(t) - A_{5n}(t), \\
Q(t) - Q(0) = Q_n(t) - A_{6n}(t).
\end{cases} \tag{3.46}$$

$$\begin{aligned}
\| \Lambda_{1n} \| &= \left\| \Theta(\aleph) (\Pi_1(t, S) - \Pi_1(t, S_{n-1})) \right. \\
&\quad \left. + \Omega(\aleph) \int_0^t (\Pi_1(u, S) - \Pi_1(u, S_{n-1})) du \right\|.
\end{aligned} \tag{3.47}$$

Using the Lipschitz condition, we have

$$\begin{aligned}
\| \Lambda_{1n} \| &\leq \Theta(\aleph) \mathcal{K}_1 \| (S - S_{n-1}) \| \\
&\quad + \Omega(\aleph) \mathcal{K}_1 \| (S - S_{n-1}) \| t.
\end{aligned} \tag{3.48}$$

Thus,

$$\| \Lambda_{1n} \| \leq [\Theta(\aleph) + \Omega(\aleph) t]^{n+1} \mathcal{K}_1^{n+1} h. \tag{3.49}$$

At t_0 , we get

$$\| \Lambda_{1n} \| \leq [\Theta(\aleph) + \Omega(\aleph) t_0]^{n+1} \mathcal{K}_1^{n+1} h. \tag{3.50}$$

This implies that as $t_0 \rightarrow \infty$, we get the following result

$$\| \Lambda_{1n} \| \rightarrow 0. \tag{3.51}$$

Similarly, one can show that.

$$\left\{ \begin{array}{l} \|\Lambda_{2n}\| \rightarrow 0, \\ \|\Lambda_{3n}\| \rightarrow 0, \\ \|\Lambda_{4n}\| \rightarrow 0, \\ \|\Lambda_{5n}\| \rightarrow 0, \\ \|\Lambda_{6n}\| \rightarrow 0. \end{array} \right. \quad (3.52)$$

This proves the existence of the solution to model (1). $\square$

3.8.2 Uniqueness of the solution

Now we have to show whether the solution is unique or not. Let there be an alternative solution to the proposed model, such that.

$$\left\{ \begin{array}{l} S(t) - S_1(t) = \Theta(\aleph) (\Pi_1(t, S) - \Pi_1(t, S_1)) + \Omega(\aleph) \int_0^t (\Pi_1(u, S) - \Pi_1(u, S_1)) du, \\ E(t) - E_1(t) = \Theta(\aleph) (\Pi_2(t, E) - \Pi_2(t, E_1)) + \Omega(\aleph) \int_0^t (\Pi_2(u, E) - \Pi_2(u, E_1)) du, \\ A(t) - A_1(t) = \Theta(\aleph) (\Pi_3(t, A) - \Pi_3(t, A_1)) + \Omega(\aleph) \int_0^t (\Pi_3(u, A) - \Pi_3(u, A_1)) du, \\ D(t) - D_1(t) = \Theta(\aleph) (\Pi_4(t, D) - \Pi_4(t, D_1)) + \Omega(\aleph) \int_0^t (\Pi_4(u, D) - \Pi_4(u, D_1)) du, \\ R(t) - R_1(t) = \Theta(\aleph) (\Pi_5(t, R) - \Pi_5(t, R_1)) + \Omega(\aleph) \int_0^t (\Pi_5(u, R) - \Pi_5(u, R_1)) du, \\ Q(t) - Q_1(t) = \Theta(\aleph) (\Pi_6(t, Q) - \Pi_6(t, Q_1)) + \Omega(\aleph) \int_0^t (\Pi_6(u, Q) - \Pi_6(u, Q_1)) du. \end{array} \right. \quad (3.53)$$

Taking the norm of the above system, we have

$$\left\{ \begin{array}{l} \|S(t) - S_1(t)\| (1 - \Theta(\aleph) \mathcal{K}_1 + \Omega(\aleph) \mathcal{K}_1 t) \leq 0, \\ \|E(t) - E_1(t)\| (1 - \Theta(\aleph) \mathcal{K}_2 + \Omega(\aleph) \mathcal{K}_2 t) \leq 0, \\ \|A(t) - A_1(t)\| (1 - \Theta(\aleph) \mathcal{K}_3 + \Omega(\aleph) \mathcal{K}_3 t) \leq 0, \\ \|D(t) - D_1(t)\| (1 - \Theta(\aleph) \mathcal{K}_4 + \Omega(\aleph) \mathcal{K}_4 t) \leq 0, \\ \|R(t) - R_1(t)\| (1 - \Theta(\aleph) \mathcal{K}_5 + \Omega(\aleph) \mathcal{K}_5 t) \leq 0, \\ \|Q(t) - Q_1(t)\| (1 - \Theta(\aleph) \mathcal{K}_6 + \Omega(\aleph) \mathcal{K}_6 t) \leq 0. \end{array} \right. \quad (3.54)$$

Theorem 3.15. *The analytical solution is unique for the Caputo fractional model under the following conditions:*

$$(1 - \Theta(\aleph) \mathcal{K}_i + \Omega(\aleph) \mathcal{K}_i t) \geq 0 \quad \text{for } i = 1, 2, \dots, 6. \quad (3.55)$$

Proof. Suppose that Eq (3.55) holds. From system (3.54), we get

$$\left\{ \begin{array}{l} \|S(t) - S_1(t)\| (1 - \Theta(\aleph) \mathcal{K}_1 + \Omega(\aleph) \mathcal{K}_1 t) \leq 0, \\ \|E(t) - E_1(t)\| (1 - \Theta(\aleph) \mathcal{K}_2 + \Omega(\aleph) \mathcal{K}_2 t) \leq 0, \\ \|A(t) - A_1(t)\| (1 - \Theta(\aleph) \mathcal{K}_3 + \Omega(\aleph) \mathcal{K}_3 t) \leq 0, \\ \|D(t) - D_1(t)\| (1 - \Theta(\aleph) \mathcal{K}_4 + \Omega(\aleph) \mathcal{K}_4 t) \leq 0, \\ \|R(t) - R_1(t)\| (1 - \Theta(\aleph) \mathcal{K}_5 + \Omega(\aleph) \mathcal{K}_5 t) \leq 0, \\ \|Q(t) - Q_1(t)\| (1 - \Theta(\aleph) \mathcal{K}_6 + \Omega(\aleph) \mathcal{K}_6 t) \leq 0. \end{array} \right. \quad (3.56)$$

Hence, we can say that

$$\left\{ \begin{array}{l} \|S(t) - S_1(t)\| = 0, \\ \|E(t) - E_1(t)\| = 0, \\ \|A(t) - A_1(t)\| = 0, \\ \|D(t) - D_1(t)\| = 0, \\ \|R(t) - R_1(t)\| = 0, \\ \|Q(t) - Q_1(t)\| = 0. \end{array} \right. \quad (3.57)$$

This implies that

$$\left\{ \begin{array}{l} S(t) = S_1(t), \\ E(t) = E_1(t), \\ A(t) = A_1(t), \\ D(t) = D_1(t), \\ R(t) = R_1(t), \\ Q(t) = Q_1(t). \end{array} \right. \quad (3.58)$$

From Eq (3.58), we concluded that the solution of the proposed model (3.22) is unique. $\square$

3.9 Ulam's stability analysis of model

The Ulam-Hyers (UH) stability is a concept in the theory of functional equations. It tells us about the stability of functional equations when small perturbations occur. In other words, UH stability gauges how close a solution of functional equations is when equations are slightly perturbed. Multiple fields, including mathematics, physics, engineering, economics, etc, can benefit from its significant applications. Here we use the sense of UH stability to analyze the global stability of the Caputo fractional model (3.22) [164, 22]. We define the following inequality by assume $\varepsilon > 0$ be a positive real number then

$$\left| {}^C D_t^{\aleph} w(t) - h(t, w(t)) \right| \leq \varepsilon, \forall t \in [0, T]. \quad (3.59)$$

where $\varepsilon = \max(\varepsilon_j)$ for $j = 1, 2, 3, 4, 5, 6$.

$$w(t) = \begin{pmatrix} S(t) \\ E(t) \\ A(t) \\ D(t) \\ R(t) \\ Q(t) \end{pmatrix}, \quad w_0 = \begin{pmatrix} S(0) \\ E(0) \\ A(0) \\ D(0) \\ R(0) \\ Q(0) \end{pmatrix} \quad h(t, w(t)) = \begin{pmatrix} \mathbb{F}_1(S, E, A, D, R, Q) \\ \mathbb{F}_2(S, E, A, D, R, Q) \\ \mathbb{F}_3(S, E, A, D, R, Q) \\ \mathbb{F}_4(S, E, A, D, R, Q) \\ \mathbb{F}_5(S, E, A, D, R, Q) \\ \mathbb{F}_6(S, E, A, D, R, Q) \end{pmatrix}$$

$$h(t, w(t)) = \begin{pmatrix} a_1 + a_2 a_3 R(t) - a_{12} a_7 A(t) S(t) - (a_6 + a_4) S(t) \\ a_{12} a_7 S(t) A(t) - E(t) (a_9 + a_4) \\ a_{14} a_9 E(t) - (a_4 + a_5 + a_{11}) A(t) \\ a_5 A(t) + a_{11} (1 - a_{10}) A(t) - (a_8 + a_{13} + a_4) D(t) \\ (1 - a_{14}) a_9 E(t) + a_8 D(t) + a_{11} a_{10} A(t) - (a_4 + a_3) R(t) \\ a_6 S(t) + (1 - a_2) a_3 R(t) - a_4 Q(t) \end{pmatrix}.$$

with the initial conditions w_0 .

For $\bar{w} \in \mathcal{E}$ is solution of Caputo fractional model (3.22) $\iff$ there is $p \in \mathcal{E}$ such that:

$$(i) : |p(t)| \leq \varepsilon.$$

$$(ii) : {}^C D_t^{\aleph} \bar{w}(t) = h(t, \bar{w}(t)) + p(t), \quad \forall t \in [0, T].$$

We implement the R-LF integral to (ii), then we get

$$\bar{w} = \bar{w}_0 + \frac{1}{\Gamma(\aleph)} \int_0^t (t-u)^{\aleph-1} h(u, \bar{w}(u)) du + \frac{1}{\Gamma(\aleph)} \int_0^t (t-u)^{\aleph-1} p(u) du \quad (3.60)$$

$$\left| \bar{w} - \bar{w}_0 - \frac{1}{\Gamma(\aleph)} \int_0^t (t-u)^{\aleph-1} h(u, \bar{w}(u)) du \right| \leq \frac{\varepsilon}{\Gamma(\aleph)} \int_0^t (t-u)^{\aleph-1} p(u) du \quad (3.61)$$

$$\left| \bar{w} - \bar{w}_0 - \frac{1}{\Gamma(\aleph)} \int_0^t (t-u)^{\aleph-1} h(u, \bar{w}(u)) du \right| \leq \frac{\varepsilon T^{\aleph}}{\Gamma(\aleph+1)}. \quad (3.62)$$

Definition 3.16. (UH Stability) The Caputo-fractional model (3.22) is called UH stable if $\exists$ a real number $C_\Phi > 0$ such that, for every $\varepsilon > 0$ and any $\bar{w}(t)$ satisfying Eq (3.59) then the Caputo fractional model (3.22) possess a solution $w(t)$ satisfying

$$|\bar{w}(t) - w(t)| \leq C_\Phi \varepsilon, t \in [0, T]. \quad (3.63)$$

where

$$\varepsilon = \max (\varepsilon_j)^T, \quad C_\Phi = \max (C_{\Phi_j})^T \quad \text{for } j = 1, 2, 3, 4, 5, 6.$$

$$\begin{aligned} \bar{w} &= \begin{pmatrix} \bar{S}(t) \\ \bar{E}(t) \\ \bar{A}(t) \\ \bar{D}(t) \\ \bar{R}(t) \\ \bar{Q}(t) \end{pmatrix} & \bar{w}_0 &= \begin{pmatrix} \bar{S}(0) \\ \bar{E}(0) \\ \bar{A}(0) \\ \bar{D}(0) \\ \bar{R}(0) \\ \bar{Q}(0) \end{pmatrix} & h(t, \bar{w}(t)) &= \begin{pmatrix} {}_1(\bar{S}, \bar{E}, \bar{A}, \bar{D}, \bar{R}, \bar{Q}) \\ {}_2(\bar{S}, \bar{E}, \bar{A}, \bar{D}, \bar{R}, \bar{Q}) \\ {}_3(\bar{S}, \bar{E}, \bar{A}, \bar{D}, \bar{R}, \bar{Q}) \\ {}_4(\bar{S}, \bar{E}, \bar{A}, \bar{D}, \bar{R}, \bar{Q}) \\ {}_5(\bar{S}, \bar{E}, \bar{A}, \bar{D}, \bar{R}, \bar{Q}) \\ {}_6(\bar{S}, \bar{E}, \bar{A}, \bar{D}, \bar{R}, \bar{Q}) \end{pmatrix} \\ \varepsilon &= \max \begin{pmatrix} \varepsilon_1 \\ \varepsilon_2 \\ \varepsilon_3 \\ \varepsilon_4 \\ \varepsilon_5 \\ \varepsilon_6 \end{pmatrix} & C_\Phi &= \max \begin{pmatrix} C_{\Phi_1} \\ C_{\Phi_2} \\ C_{\Phi_3} \\ C_{\Phi_4} \\ C_{\Phi_5} \\ C_{\Phi_6} \end{pmatrix} \end{aligned}$$

Since $\exists$ a unique solution of model (3.22), we proposed the following result:

Theorem 3.17. Suppose $\bar{w}(t) = (\bar{S}(t), \bar{E}(t), \bar{A}(t), \bar{D}(t), \bar{R}(t), \bar{Q}(t))^T$, then

$$\left\| h(u, \bar{w}(u)) - h(u, w(u)) \right\| \leq r \left\| \bar{w}(u) - w(u) \right\| \quad (3.64)$$

where $r > 0$ is constant.

Theorem 3.18. Suppose that assumptions (i) and (ii) hold. Then Caputo fractional model (3.22) is UH stable $[0, T]$ if $\Gamma(\aleph + 1) \geq \varepsilon T^\aleph$.

Proof. Consider the unique solution of the Caputo fractional model (3.22) $w(t)$ and let it $\bar{w}(t)$ satisfy Eq (3.63). The R-LF integral implemented on both sides of model (3.22), we get

$$w(t) = w_0(t) + \frac{1}{\Gamma(\aleph)} \int_0^t (t-u)^{\aleph-1} h(u, w(u)) du \quad (3.65)$$

Now, using Eqs (3.65) and (3.62) we compute Norm $\left\| \bar{w}(t) - w(t) \right\|$ as

$$\left\| \bar{w}(t) - w(t) \right\| = \left\| \bar{w}(t) - w_0(t) - \frac{1}{\Gamma(\aleph)} \int_0^t (t-u)^{\aleph-1} h(u, w(u)) du \right\| \quad (3.66)$$

By adding and subtracting term $\frac{1}{\Gamma(\aleph)} \int_0^t (t-u)^{\aleph-1} h(u, \bar{w}(u)) du$ and applying the triangular inequality, we have,

$$\begin{aligned} \left\| \bar{w}(t) - w(t) \right\| &\leq \left\| \bar{w}(t) - w_0(t) - \frac{1}{\Gamma(\aleph)} \int_0^t (t-u)^{\aleph-1} h(u, \bar{w}(u)) du \right\| + \\ &\quad \left\| \frac{1}{\Gamma(\aleph)} \int_0^t (t-u)^{\aleph-1} (h(u, \bar{w}(u)) - h(u, w(u))) du \right\| \end{aligned} \quad (3.67)$$

Using Eq (3.62), Theorem (3.17) and norm property,

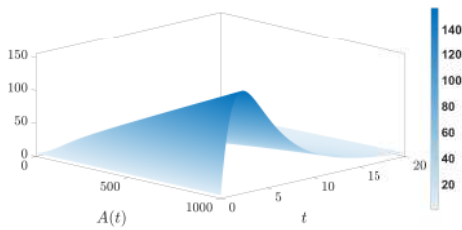
$$\left\| \bar{w}(t) - w(t) \right\| \leq \frac{\varepsilon T^\aleph}{\Gamma(\aleph+1)} + \frac{r T^\aleph}{\Gamma(\aleph+1)} \left\| \bar{w}(t) - w(t) \right\| \quad (3.68)$$

This implies that

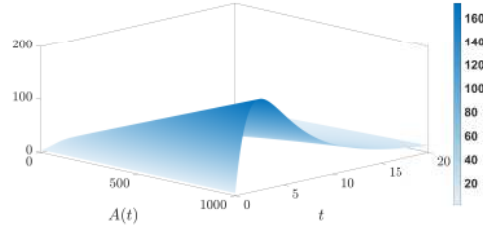
$$|\bar{w}(t) - w(t)| \leq C_\Phi \varepsilon \quad (3.69)$$

where $C_\Phi = \frac{T^\aleph}{\Gamma(\aleph+1) - r T^\aleph}$. We conclude that the Caputo fractional model (1) is UH stable. $\square$

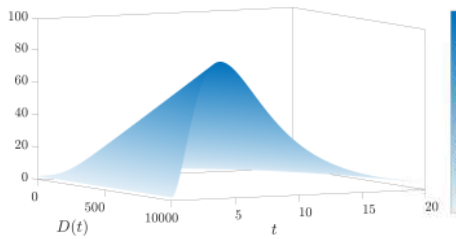
Ulam's stability defines the global impact by making a small perturbation around the system. UH stability prevents small errors from causing drastic errors in long-term disease prediction, crucial in real-life epidemiological modeling where disease transmission, rehabilitation periods, recovery process, etc, play a critical role. We define this global stability analysis in Fig (3.7) by perturbing the initial condition from its baseline in addiction and depression phenomena. We see that in both cases, like $R_0 > 1$ and $R_0 < 1$, the solution approaches their equilibrium states.



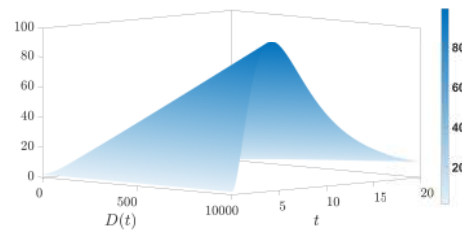
(a) Addicted Class $A(t)$ with $R_0 < 1$.



(b) Addicted Class $A(t)$ with $R_0 > 1$.



(c) Depressed Class $D(t)$ with $R_0 < 1$.



(d) Depressed Class $D(t)$ with $R_0 > 1$.

FIGURE 3.7: Simulation of addiction and depression phenomena for model (3.22) with Caputo fractional derivative presents here. first column shows present the case $R_0 < 1$ when we perturbate the initial condition of both classes with $a_4 = 0.09, a_{12} = 0.2$ while second column describe the case $R_0 > 1$ with $a_4 = 0.05, a_{12} = 0.8$.

3.10 Numerical simulation of model

Here we explain the numerical implementation of all schemes used for the model with the initial conditions (ICs) $S_0 = 100, E_0 = 1, A_0 = 5, D_0 = 2, R_0 = 0, Q_0 = 10$ and parameters given in Table 3.2. For case $R_0 = 0.2171 < 1$ we take parameter $a_4 = 0.09$ and $a_{12} = 0.2$ while parameter values $a_4 = 0.05$ and $a_{12} = 0.8$ used for case $R_0 = 1.7590 > 1$. In both cases, we take a step size of $h = T/N$ and $N = 1000$. Mathematical formulation of all schemes with respective models is shown in Appendix A.

3.10.1 Adams–Bashforth–Moulton PECE scheme

We examined model (3.22) under the Caputo derivative, using "fde12" [124], a MATLAB routine to solve the system of fractional differential equations using the Caputo operator, a popular approach in fractional calculus which is implemented by Garrapa and based on the Adams Bashforth Moulton PECE scheme. The convergence, accuracy, and stability of this method can be found in [95, 123]. The objective is to describe the qualitative properties of model (3.22) using the Caputo derivative of order $0 < \aleph < 1$ compared to the classical model at $\aleph = 1$ as shown in Fig 3.8. This study demonstrates that addiction and depression classes rapidly increase for $R_0 > 1$ compared to $R_0 < 1$ with $\aleph = 0.995$. Behavior of all classes on various fractional order $\aleph = \{0.7, 0.8, 0.85, 0.9, 0.95, 0.995, 1\}$ shown in Fig 3.9 for $R_0 < 1$. Our analysis reveals that susceptible individuals decrease rapidly with the variation of $\aleph$ approaching 1, becoming stable due to a high decay rate. This leads to increased transmission to addicted individuals, causing a sudden jump in exposed individuals. Addiction increases gradually due to increased contact rate, while media influence on addicted individuals increases depression. We investigate the effects of Caputo derivative for $R_0 > 1$ by examining the outcomes of all classes by adjusting the fractional order $\aleph = \{0.7, 0.8, 0.85, 0.9, 0.95, 0.995, 1\}$ in Fig 3.10 which shows that disease spreads rapidly due to susceptible individuals transmitting to infected individuals, leading to an exponential increase in the exposed population and rapid growth of depression in social media addicted individuals. Although the recovered class does not experience sufficient impact, permanently quieter populations experience rapid growth. Surface plots in Fig 3.11 represent the behavior of the model on fractional order $0 < \aleph < 1$ with the changing of time.

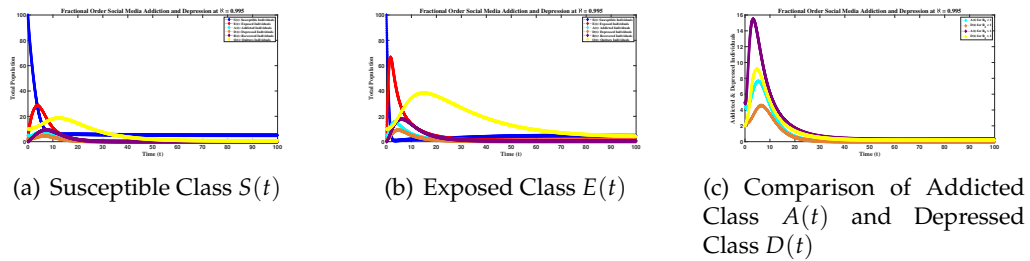


FIGURE 3.8: All classes simulation for model (3.22) with Caputo fractional derivative shown here. (a) present the case $R_0 < 1$ with $a_4 = 0.09, a_{12} = 0.2$ while (b) describe the case $R_0 > 1$ with $a_4 = 0.05, a_{12} = 0.8$ and (c) Compare Addicted and Depressed classes with Caputo derivative $\aleph = 0.995$ for $R_0 > 1$ and $R_0 < 1$.

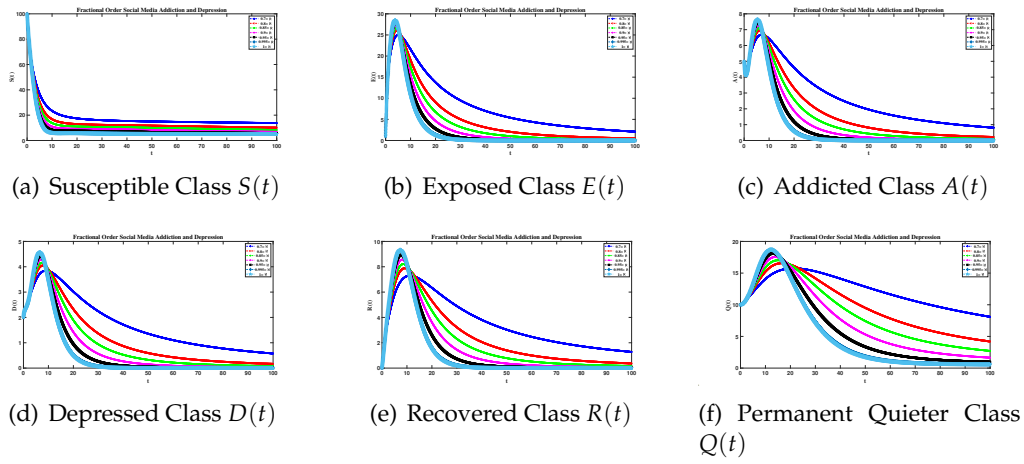


FIGURE 3.9: Results of model (3.22) with Caputo fractional derivative at order $\aleph = \{0.7, 0.8, 0.85, 0.9, 0.95, 0.995, 1\}$ for $R_0 < 1$.

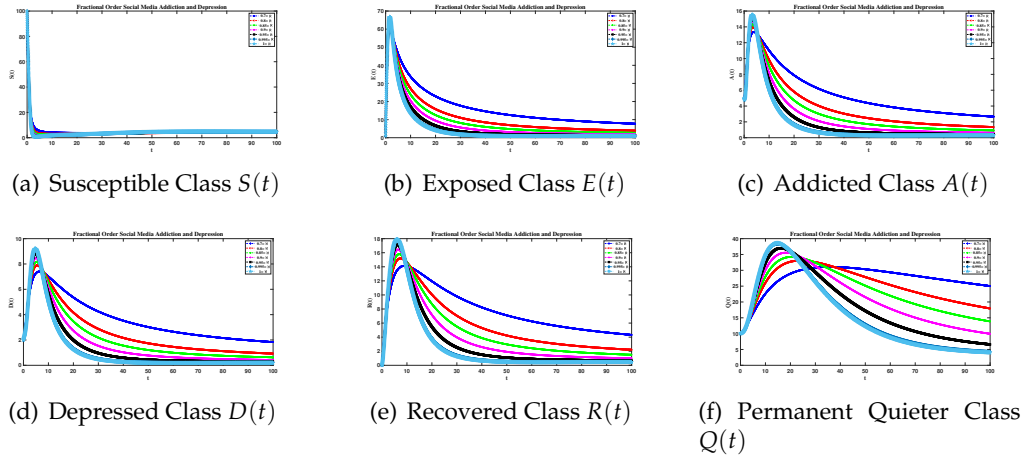


FIGURE 3.10: Results of model (3.22) with Caputo fractional derivative at order $\aleph = \{0.7, 0.8, 0.85, 0.9, 0.95, 0.995, 1\}$ for $R_0 > 1$.

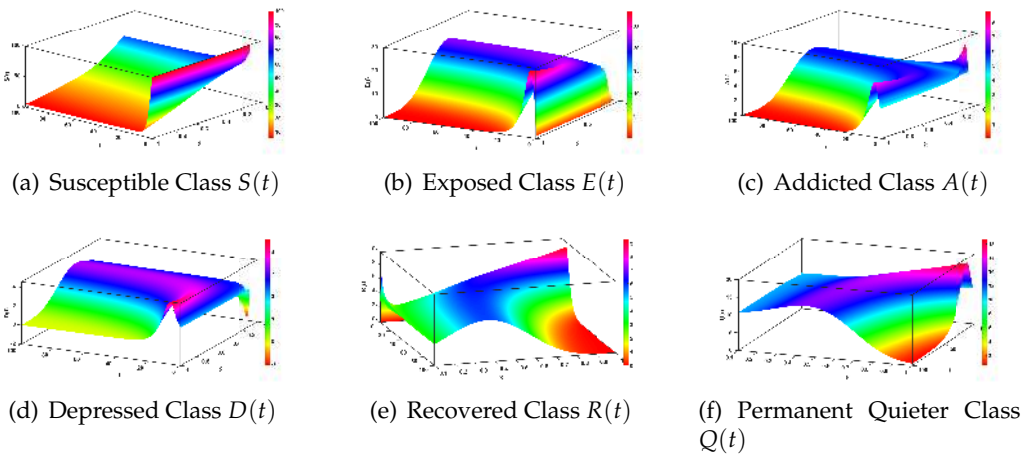


FIGURE 3.11: Analysis of Caputo fractional derivative $\aleph$ vs compartment's populations over time.

3.10.2 Fractional Forward Euler Scheme (FFE)

Here we discuss the simulation of the FFE scheme of model (3.22) with Caputo fractional derivative. We display the simulation for $R_0 < 1$, $R_0 > 1$, and comparison for addiction and depression classes of both cases in Fig 3.12 at fractional order $\aleph = 0.995$. Comparison shows the dynamic change for both cases. This clarifies the enormous spike in the addiction and depression class for $R_0 > 1$ relative to $R_0 < 1$ and shows the impact of parameters a_4 and a_{12} . We investigate the findings of all classes by adjusting the fractional order $\aleph = \{0.7, 0.8, 0.85, 0.9, 0.95, 0.995, 1\}$, as seen in Fig 3.13 for $R_0 < 1$, to verify the influence of non-integer order derivative. As we can observe, with a small value of $\aleph$, the decay rate rapidly increases. When the $\aleph$ approaches integer order 1, then the disease dynamics go to its equilibrium state. One can easily observe that there is some decay at the initial point for addicted individuals, which may be due to some external factors. Due to the restricted movement of individuals, we see how the susceptible individuals decline at varying rates and stabilize after ten days. This occurs because there is a sharp increase in the exposed population as a result of the rapid rate of decay of the exposed population, which increases the transmission to addicts. Similarly, for $R_0 > 1$ after examining the outcomes of all individuals by adjusting the fractional order $\aleph = \{0.7, 0.8, 0.85, 0.9, 0.95, 0.995, 1\}$ in Fig 3.14, we witness the rapid increase in depression dynamics concerning addiction to social media. Due to the increase in recovered class, the permanent quitter increased rapidly and did not stabilize even after 100 days, and may become susceptible after some time. Fig 3.15 shows the effects of fractional order $0 < \aleph < 1$ on all populations' cures versus time.

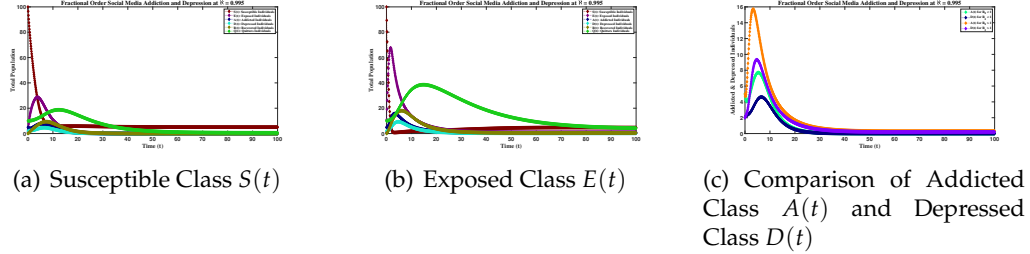


FIGURE 3.12: All classes simulation for model (3.22) with Caputo fractional derivative shown here. (a) present the case $R_0 < 1$ with $a_4 = 0.09, a_{12} = 0.2$ while (b) describe the case $R_0 > 1$ with $a_4 = 0.05, a_{12} = 0.8$ and (c) Compare Addicted and Depressed classes with Caputo derivative $\aleph = 0.995$ for $R_0 > 1$ and $R_0 < 1$.

3.10.3 Adams-Bashforth Molten Scheme (ABM)

We explore numerical solutions of model (3.22) with the ABM scheme under the Caputo fractional derivative. The numerical results of the whole population with fractional order $\aleph = 0.995$ are shown in Fig 3.16 for $R_0 < 1$ using parameters $a_4 = 0.09$ and $a_{12} = 0.2$, $R_0 > 1$ taking parameter $a_4 = 0.05$ and $a_{12} = 0.8$ and Comparison of both case for addiction and depression dynamics. It is evident that when $R_0 < 1$, the solution curves approach steady states uniformly. To confirm the impact of the Caputo derivative, we analyze the results of each compartment separately for a fractional order $\aleph = \{0.7, 0.8, 0.85, 0.9, 0.95, 0.995, 1\}$, as shown in Fig 3.17 for $R_0 < 1$. When $\aleph$ is small, the decay rate increases significantly. When the $\aleph$ approaches integer order 1, then the disease dynamics go to its equilibrium state. One can easily

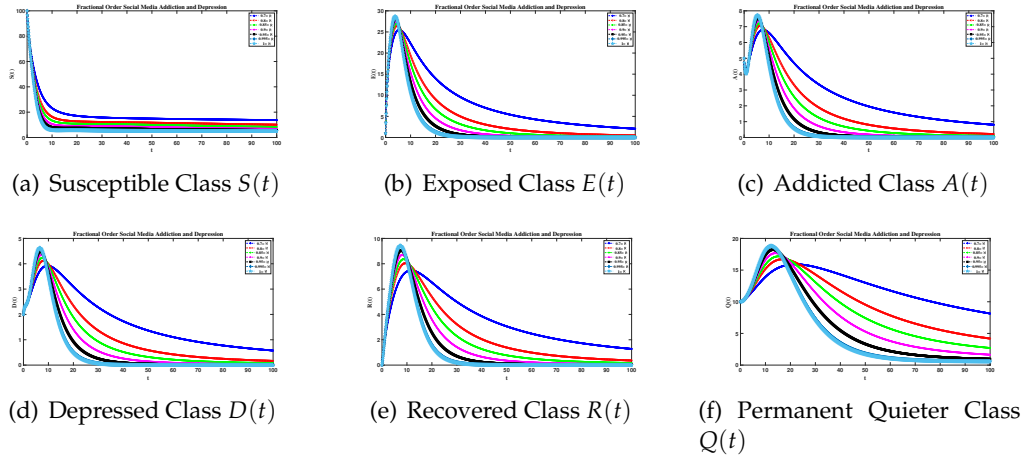


FIGURE 3.13: Results of model (3.22) with Caputo fractional derivative at order $\aleph = \{0.7, 0.8, 0.85, 0.9, 0.95, 0.995, 1\}$ for $R_0 < 1$

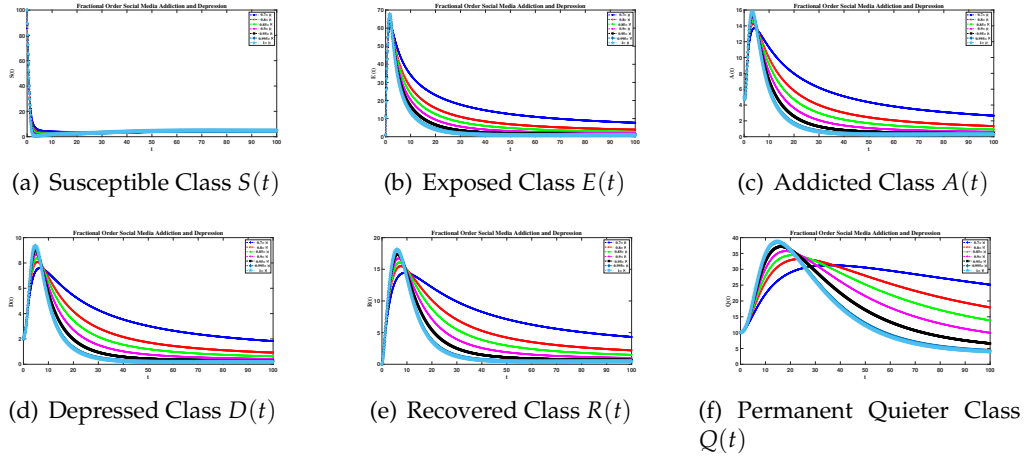


FIGURE 3.14: Results of model (3.22) with Caputo fractional derivative at order $\aleph = \{0.7, 0.8, 0.85, 0.9, 0.95, 0.995, 1\}$ for $R_0 > 1$

observe that there is a sharp decrease in susceptible individuals because the transmission parameters increase, due to which the addicted class increases gradually, with a sudden increase in the exposed class. Addicted individuals who may recover without some treatment may go to a permanent quieter or/and may become susceptible. Due to the strong influence of social media, addicted people become depressed and stable after a gradual increase. In Fig 3.18, we describe the effects of Caputo fractional derivative for $R_0 > 1$ by examining the outcomes of all individuals by taking the fractional order $\aleph = \{0.7, 0.8, 0.85, 0.9, 0.95, 0.995, 1\}$. We see that all the solution curves for each $\aleph$ collect slightly at a point because the decay rate is very high. Due to this, the exposed class suddenly increased. With the increase in contact rate, the depressed individuals increases along with the increase in the depression rate. This shows signs of the increase in recovered class because people start to complete the treatment process to take this as a prevention measure, due to which the solution curve for the recovered class stable after 50 days. Parameter quieter increases show the impact of the recovery rate. Effects of fractional order $0 < \aleph < 1$ on all

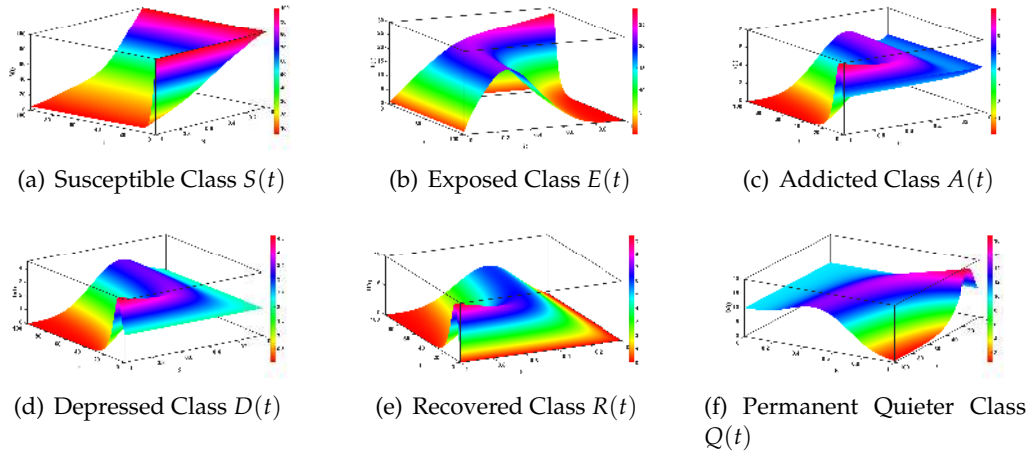


FIGURE 3.15: Analysis of Caputo fractional derivative $\aleph$ vs compartment's populations over time.

populations' cures with time seen in Fig 3.19.

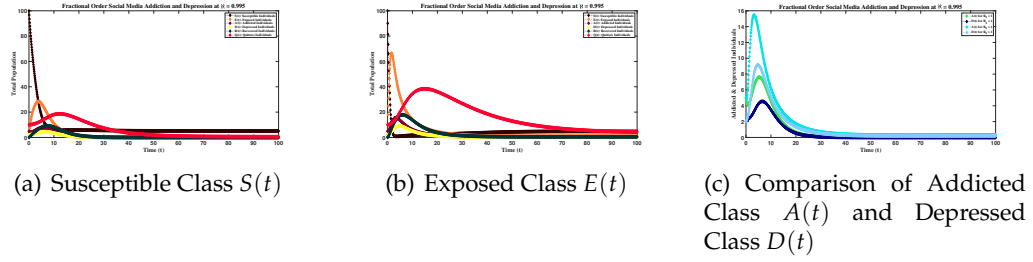


FIGURE 3.16: All classes simulation for model (3.22) with Caputo fractional derivative shown here. (a) present the case $R_0 < 1$ with $a_4 = 0.09, a_{12} = 0.2$ while (b) describe the case $R_0 > 1$ with $a_4 = 0.05, a_{12} = 0.8$ and (c) Compare Addicted and Depressed classes with Caputo derivative $\aleph = 0.995$ for $R_0 > 1$ and $R_0 < 1$.

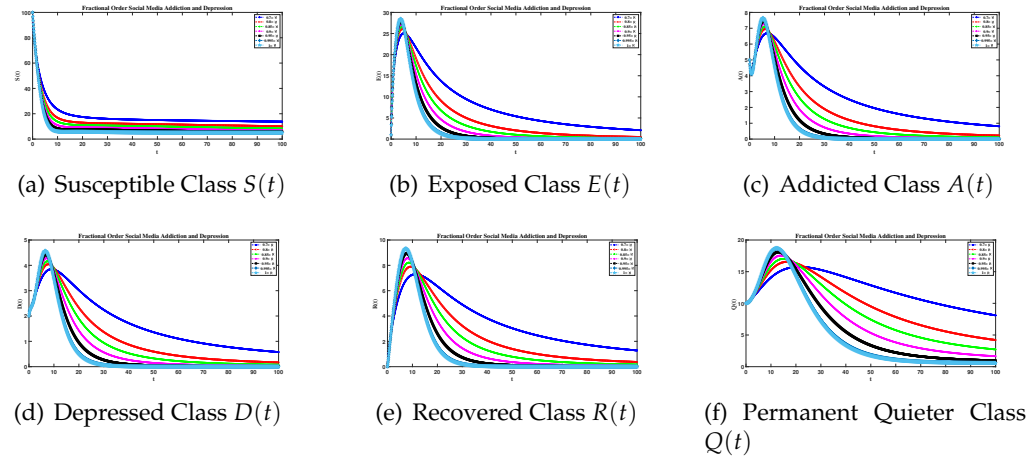


FIGURE 3.17: Results of model (3.22) with Caputo fractional derivative at order $\aleph = \{0.7, 0.8, 0.85, 0.9, 0.95, 0.995, 1\}$ for $R_0 < 1$

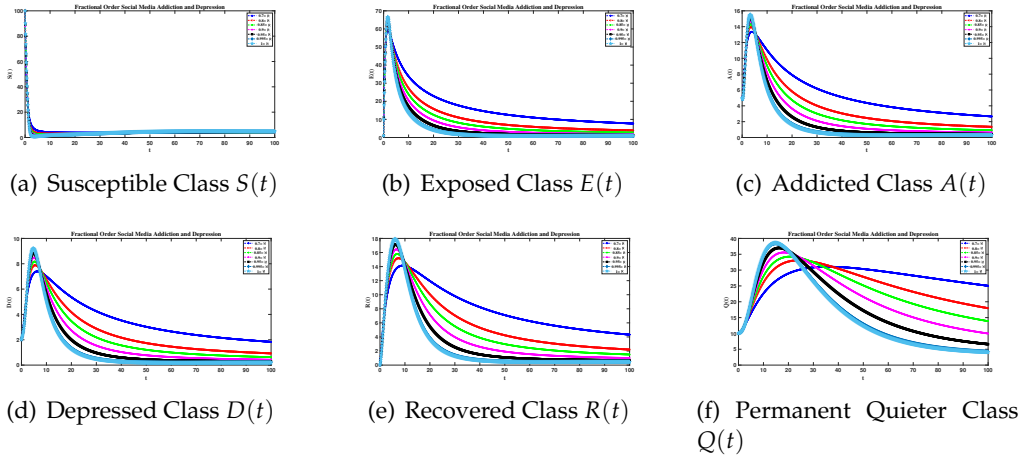


FIGURE 3.18: Results of model (3.22) with Caputo fractional derivative at order $N = \{0.7, 0.8, 0.85, 0.9, 0.95, 0.995, 1\}$ for $R_0 > 1$

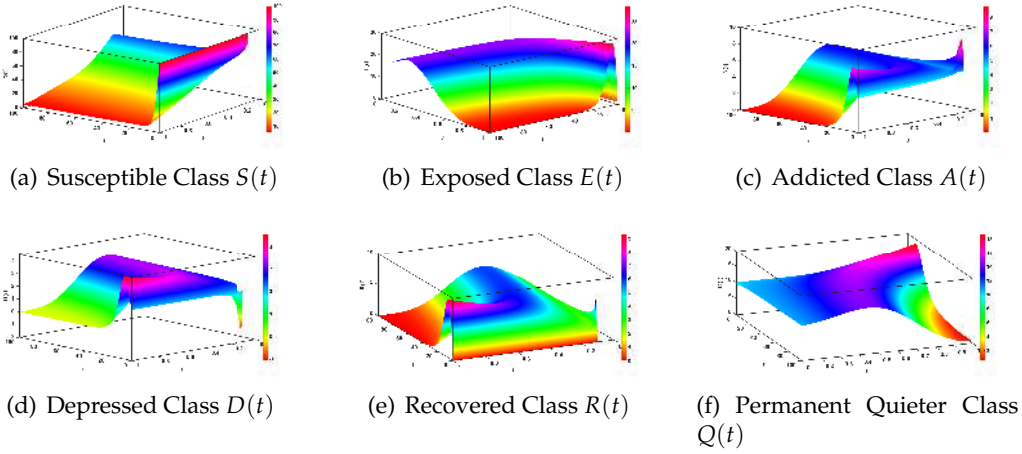


FIGURE 3.19: Analysis of Caputo fractional derivative N vs compartment's populations over time.

3.11 Effect of Parameters

In this section, we define the impact of some important parameters on model (3.22) to check the variation of addiction and depression dynamics. We define the four cases to check the prediction of these dynamic behaviors.

3.11.1 Case 1: The rate at which addiction spreads to susceptible people (a_{12}).

Here, we fixed the other parameters and varied the values of the rate at which susceptible people become addicted, which is (a_{12}). We estimate the model (3.22) for values of $a_{12} = \{0.2, 0.4, 0.6, 0.8\}$. This parameter is very important for the dynamic behavior of the model (3.22). Because when its values approach 0.8, then $R_0 > 1$. From Fig 3.20 we can easily conclude that there is a direct relation between the addicted and depressed class with parameter a_{14} . With the increase of a_{12} , the number

of addicted individuals increases, due to which the transmission of depression increases in addicted individuals. So, to control the addiction to social media and depression, the transmission rate of addiction to susceptible individuals (a_{12}) is an important factor.

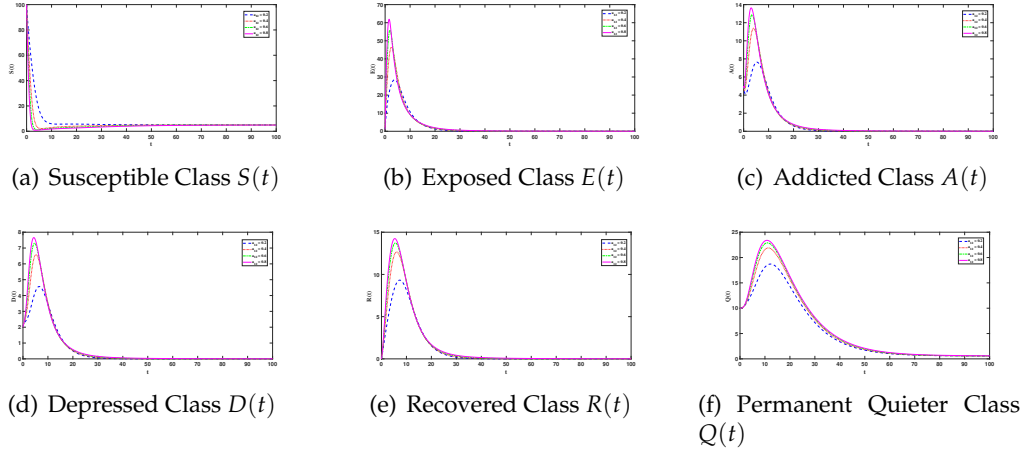


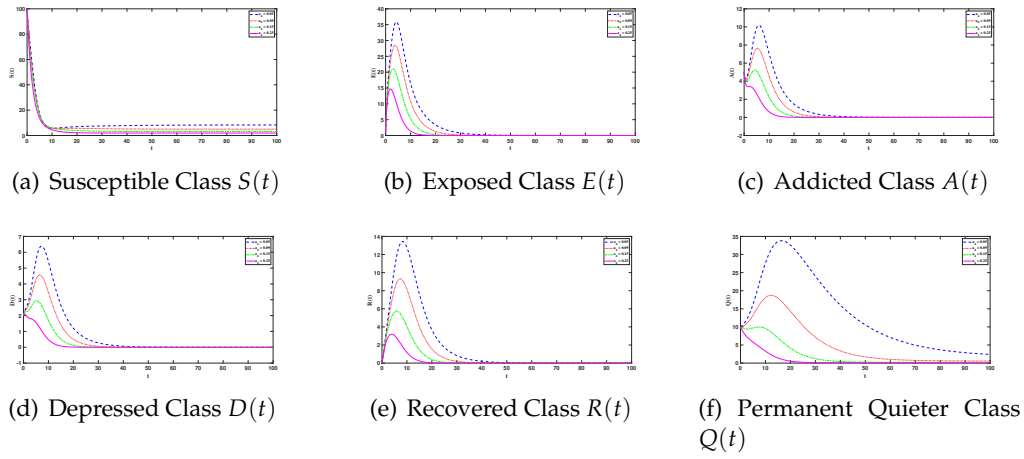
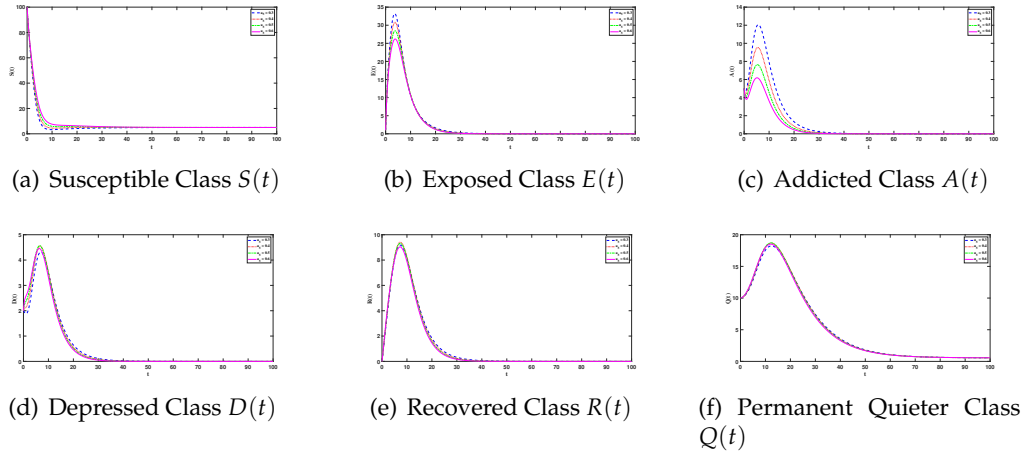
FIGURE 3.20: Variation of parameters a_{12} on model (3.22)

3.11.2 Case 2: Natural death rate (a_4).

In this subsection, we fixed the other parameters and varied the values of the natural death rate (a_4). We approximate the model (3.22) for values of $a_4 = \{0.05, 0.09, 0.15, 0.25\}$. a_4 plays an essential role in predicting the dynamic behavior of the model (3.22). Because $R_0 > 1$ when its values become closer to 0.25. From Fig 3.21, we can easily conclude that there is a direct relation between the addicted and depressed class with parameter a_4 . Fewer people are passing away from natural causes when the natural mortality rate is lower. This indicates that there are more people accessible for the infectious disease to spread among and that the population number is largely steady. More people are passing away from natural causes as the population's natural mortality rate rises to 0.25. Eventually, this causes the population's total size to decline. There are fewer susceptible individuals in smaller numbers for the infectious disease to propagate. As a result, on average, each infected individual infects fewer new individuals. So, to control the addiction to social media and depression dynamics, the natural death rate (a_4) is an essential thing.

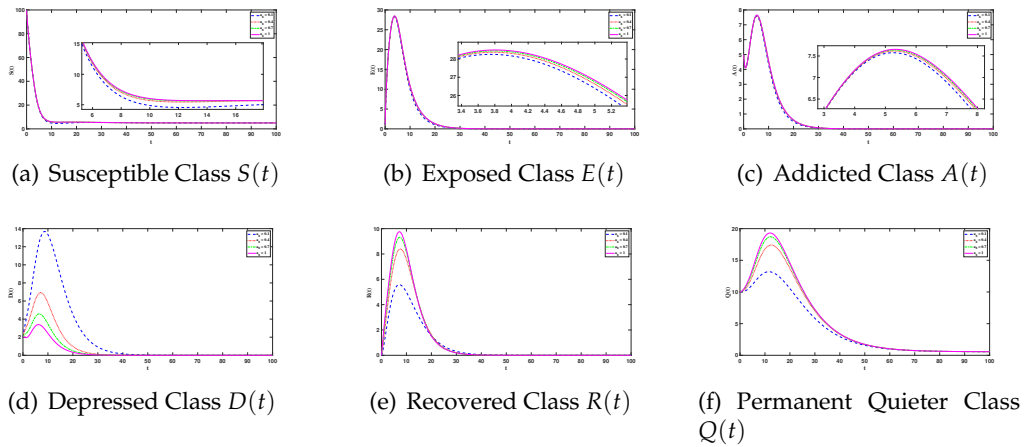
3.11.3 Case 3: The rate at which media effects become a cause of depression (a_5).

Here we explain the analysis of the variation of parameter a_5 . We fixed the other parameters and varied the values of the rate of media influence that causes depression in people (a_5). We approximate the model (3.22) for values of $a_5 = \{0.3, 0.4, 0.5, 0.6\}$. Fig 3.22 indicates that increasing the value of a_5 , the addicted individuals decrease gradually with the increase of depressed individuals because the addicted people get more influence from social media, due to which most of the people do not auto-recover. Therefore, a_4 is vital to predict the dynamic behavior of the model (3.22) and also to control this infection.

FIGURE 3.21: Variation of parameters a_4 on model (3.22)FIGURE 3.22: Variation of parameters a_5 on model (3.22)

3.11.4 Case 4: Depressed individuals who join the recovered class after the treatment (a_8).

The investigation of parameter a_8 is explained in this subsection. We keep the other parameters fixed and vary the values of the rate at which media influence causes people to become depressed (a_8) to approximate the model (3.22) for $a_5 = \{0.1, 0.4, 0.7, 1\}$. Fig 3.23 shows that increasing the value of a_8 and approaching 0.6, the depressed individuals decrease gradually because the affected people get treatment properly, and most of the people recovered. Hence, the recovery rate increases, so the permanent quieter increases instantly. But there is little impact on the other classes. Therefore, a_8 is vital to predict the dynamic behavior of the model (3.22) and also to control the recovery and permanent quieter individuals.

FIGURE 3.23: Variation of parameters a_8 on model (3.22)

3.12 Discussion

Traditional classical calculus models overlook memory effects in epidemic dynamics, while fractional order operators offer a more realistic framework for incorporating system memory and hereditary features in disease modeling. This section examines the dynamics of state variables in the proposed fractional model under the Atangana-Baleanu-Caputo (with non-singular kernel) and Caputo fractional (with singular kernel) operators. The model's significant biological parameters and the change in fractional-order $\aleph$ are used to create profiles for each state variable, selected from Table 3.2. This paper argues that only numerical simulations are affected by small changes in the fractional derivative order, not the general behavior of the resulting functions. It uses Adams Bashforth Moulton PECE (fde12), Fractional Forward Euler (FFE), and Adams Bashforth Molten (ABM) with Caputo derivative, which exhibits non-smooth behavior at the initial time, following the convergence properties of the numerical schemes. All the results are qualitatively similar for all schemes. As we can see from both fractional order operators' results, with time, the susceptible individuals gradually decrease and stabilize after some time when $\aleph$ approaches 1. This decline is due to a high decay rate and increases in the contact rate of susceptible individuals with infected individuals. Due to this reason, exposed individuals grow suddenly. The curve for exposed individuals goes up and comes to stability after a decline. People in this class are contaminated during the incubation phase, but they are not yet exhibiting symptoms or able to spread the disease.

Similarly, exposed individuals who start to continue using social media badly become social media addicted, which affects their study, sleeping, and work routine of students and other people, and they start to feel anxiety, tiredness, and laziness throughout the whole day. All these individuals moved to the addicted class. At that point, a few take some measures or self-control strategies like starting to manage their daily routine and using social media in a good sense; they recover and move to the recovered individuals, and again become susceptible. The renaming of people who are continuously becoming addicted to social media addicted. When someone starts using social media badly, they are easily affected in a good or bad way by the opinions of other people who are linked with them on social media apps like Facebook (Meta), Twitter (X), WhatsApp, YouTube, Instagram, etc. They are easily impressed by others' social posts, start feeling him/herself lonely, and do not

take an interest in true relationships. Sometimes they are badly affected by someone's bad comments or offensive opinions about beauty, wealth, racism, religious extremism, poverty, etc, on his/her social posts. If these things continue for a long time, they feel anxious, lonely, and depressed, and become depressed individuals. So, when the influence of social media increases on addicted individuals, they move to the depressed class. Those people who take proper treatment and complete the process recover and move to the recovered class, while those people who do not take medicine become mentally unstable, like madness, and in some cases, this leads to suicide. While others who take medicine but cut off the process after some time, again move to the addicted or susceptible class. So with the decrease in addicted individuals, the recovered class increases. With the increase of recovered individuals, the permanent quieter increases instantly, and some of the susceptible become recovered.

To check the comparison results for specifically addicted and depressed individuals, we utilize Toufik Atangana (TAS) and Adams type predictor-corrector (ATPC) with Atangana-Baleanu-Caputo fractional derivative, which can be seen in Fig 3.24 by assuming time in days on the x-axis and population on the y-axis. The results are predicted for $N=2000$ when $h=N/100$. All these schemes have a singular kernel that exhibits smooth behavior at the initial time, following the convergence properties of the numerical schemes. We see that the ATPC scheme predicts more addicted and depressed cases for the Atangana-Baleanu-Caputo operator as compared to the fde12, FFE, and ABM schemes for the Caputo operator. The main point to note is that addicted and depressed individuals become stable after 20 days of the ATPC scheme, while Caputo operator schemes become stable approximately after 40 days. This means the Caputo operator predicts a higher disease persistence ratio as compared to the Atangana-Baleanu-Caputo operator for the model. The error analysis

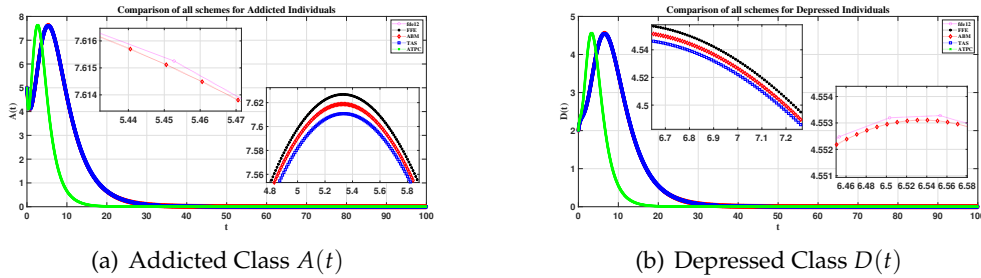


FIGURE 3.24: Comparison of addicted and depressed Class with Caputo and Atangana-Baleanu-Caputo operators

of all schemes with the Caputo operator is given in Tables 3.3, 3.4 by taking fde12 as reference solution due to its higher order convergence and min computational cost. Table 3.3 shows that FFE has less computation cost but exhibits more error. The difference between the solutions of fde12 and FFE for each class is large. Also, Table 3.4 predicts that ABM has relatively more computation cost but exhibits very small error as compared to FFE, and the difference between the solutions of fde12 and ABM is given for each class, which indicates that the difference is much less. So, one can use one of these schemes according to their Caputo fraction problem specification. Similarly, Tables 3.5-3.6 present the error analysis of all schemes with the Atangana-Baleanu-Caputo operator. Table 3.5 predicts that TAS has less computation cost but exhibits more error because it takes more time steps to converge the solution. The

difference between the solutions for each class is large. Table 3.6 predicts that ATPC has relatively more computation cost but exhibits much less error as compared to TAS because it takes fewer time steps to converge the solution. Also, the difference between the solutions for each class is very small. Hence, one can use one of these schemes according to the Atangana-Baleanu-Caputo fraction problem specification. We provide the following justification to support the suitability of the model for real-world applications.

- We used the Caputo fractional derivative, which has a memory effect property, making it a reasonable choice to analyze the interplay of addiction and depression. These physiological states happen due to pre-pressure of social media exposure, anxiety, mental health issues, and demographics because they emerge from the entire history, not just recent events, which is a non-local behavior.
- We utilize different numerical schemes to justify this behavior by taking a fractional order derivative $\aleph \in (0, 1)$ because this phenomenon can not be properly modeled using simple integer order differential equations models. By providing a comprehensive modeling approach that enables interpolation between dynamic behaviors as $\aleph$ varies, the fractional derivative improves its capacity to capture psychological impacts throughout time.
- The model reveals significant relapse dynamics and connections between social media addiction and depressed symptoms, as extensively studied in behavioral health and psychology literature. The simulations show qualitative behavior consistent with these patterns, including recovery processes under lower exposure, proper treatment, and increased depression risk with excessive social media use.
- Our focus is to provide a mathematical framework for understanding social media addiction and depression dynamics using fractional derivatives. It emphasizes the role of media influence, awareness against excessive use, and mental issues due to addiction in understanding these complex behaviors. The model uses parameter values from existing literature, ensuring consistency with real-world situations. Stability and sensitivity analyses are also conducted to provide more accuracy and prove the model's well-posedness.

TABLE 3.3: Error analysis of the numerical results achieved via Fractional Forward Euler Scheme (FFE) at $\aleph = 1$

Method	Δt	CPU Time (s)	Error	Remarks		
FFE	0.01	0.006485	5.186e-01	Stable		
	0.007	0.004849	2.579e-01	Stable		
	0.003	0.002760	1.286e-01	Stable		
	0.001	0.009253	6.422e-02	Stable		
	0.0009	0.031252	3.209e-02	Stable		
	0.0004	0.076402	1.604e-02	Stable		
	0.0002	0.135925	8.019e-03	Stable		
	0.0001	0.467784	4.009e-03	Stable		
	0.00006	1.911050	2.004e-03	Stable		
	0.00003	10.375891	1.002e-03	Stable		
	0.00001	55.772499	5.011e-04	Stable		
Values	$S(t)$	$E(t)$	$A(t)$	$D(t)$	$R(t)$	$Q(t)$
$\ fde12 - FFE\ _1$	2.449e-03	2.838e-03	5.854e-04	9.356e-05	5.853e-05	5.784e-05
$\ fde12 - FFE\ _2$	3.236e-04	3.744e-04	7.751e-05	1.326e-05	8.410e-06	8.852e-06
$\ fde12 - FFE\ _\infty$	4.803e-05	5.544e-05	1.160e-05	2.396e-06	1.629e-06	2.014e-06

TABLE 3.4: Error analysis of the numerical results achieved via Adams-Bashforth Molten Scheme (ABM) at $\aleph = 1$

Method	Δt	CPU Time (s)	Error	Remarks		
ABM	0.01	0.016615	2.875e-03	Stable		
	0.007	0.011957	7.121e-04	Stable		
	0.003	0.006981	1.772e-04	Stable		
	0.001	0.022558	4.420e-05	Stable		
	0.0009	0.064408	1.104e-05	Stable		
	0.0004	0.211950	2.757e-06	Stable		
	0.0002	0.323301	6.891e-07	Stable		
	0.0001	0.992854	1.723e-07	Stable		
	0.00006	4.885589	4.305e-08	Stable		
	0.00003	22.277127	1.076e-08	Stable		
	0.00001	102.221213	2.681e-09	Stable		
Values	$S(t)$	$E(t)$	$A(t)$	$D(t)$	$R(t)$	$Q(t)$
$\ fde12 - ABM\ _1$	1.191e-08	1.591e-08	4.489e-09	1.836e-09	1.611e-09	1.652e-10
$\ fde12 - ABM\ _2$	1.555e-09	2.079e-09	5.954e-10	2.404e-10	2.102e-10	2.260e-11
$\ fde12 - ABM\ _\infty$	2.376e-10	3.114e-10	8.960e-11	3.645e-11	3.214e-11	3.956e-12

TABLE 3.5: Error analysis of the numerical results achieved via Toufik-Atangana Scheme (TAS) at $\aleph = 1$ and $N = 2^{19}$ as reference solution

Method	Δt	CPU Time (s)	Error	Remarks		
TAS	0.003	0.537499	4.450e-04	Stable		
	0.001	0.203856	1.111e-04	Stable		
	0.0009	0.453970	2.774e-05	Stable		
	0.0004	1.069371	6.932e-06	Stable		
	0.0002	2.162431	1.732e-06	Stable		
	0.0001	8.188175	4.330e-07	Stable		
	0.00006	36.168801	1.082e-07	Stable		
	0.00003	72.705276	2.696e-08	Stable		
	0.00001	448.812087	6.660e-09	Stable		
	0.000007	1553.436691	1.588e-09	Stable		
	0.000003	4914.702484	3.173e-10	Stable		
Values	$S(t)$	$E(t)$	$A(t)$	$D(t)$	$R(t)$	$Q(t)$
$\ TAS - TAS_{3e-6}\ _1$	3.024e-09	3.645e-09	8.195e-10	3.518e-10	2.871e-10	3.040e-11
$\ TAS - TAS_{3e-6}\ _2$	1.991e-10	2.394e-10	5.399e-11	2.294e-11	1.873e-11	2.095e-12
$\ TAS - TAS_{3e-6}\ _\infty$	1.506e-11	1.770e-11	4.011e-12	1.727e-12	1.475e-12	1.865e-13

TABLE 3.6: Error analysis of the numerical results achieved via Adams-type Predictor-Corrector Scheme (ATPC) at $\aleph = 1$ and $N = 2^{19}$ as reference solution

Method	Δt	CPU Time (s)	Error	Remarks		
ATPC	0.003	0.060952	1.249e+00	Stable		
	0.001	0.045743	6.236e-01	Stable		
	0.0009	0.095342	3.113e-01	Stable		
	0.0004	0.188558	1.553e-01	Stable		
	0.0002	0.383357	7.732e-02	Stable		
	0.0001	1.155541	3.835e-02	Stable		
	0.00006	5.178137	1.887e-02	Stable		
	0.00003	25.539938	9.132e-03	Stable		
	0.00001	113.470768	4.261e-03	Stable		
	0.000007	495.325639	1.826e-03	Stable		
	0.000003	2891.464980	6.088e-04	Stable		
Values	$S(t)$	$E(t)$	$A(t)$	$D(t)$	$R(t)$	$Q(t)$
$\ ATPC - ATPC_{3e-6}\ _1$	8.711e-03	2.704e-03	7.495e-04	1.893e-04	8.671e-04	2.718e-04
$\ ATPC - ATPC_{3e-6}\ _2$	5.669e-04	2.415e-04	5.008e-05	1.490e-05	5.472e-05	1.915e-05
$\ ATPC - ATPC_{3e-6}\ _\infty$	6.503e-05	4.612e-05	5.142e-06	1.732e-06	4.007e-06	1.952e-06

3.13 Conclusion

In this manuscript, we provide the modeling and numerical simulation of the proposed fractional model by substituting the Caputo fractional derivative. The basic

reproduction number $\mathbf{R}_0$ is calculated by applying the next generation matrix approach, which works as a threshold parameter in the spread of addiction and depression and gives information about the persistence or vanishing of these physiological states over time. The stability analysis of both steady states E_0 and E_1 of the model is defined. According to the mathematical study, the disease-free equilibrium point (E_0) is locally asymptotically stable if $\mathbf{R}_0 < 1$, which indicates that the disease will eventually die out in the population. However, it will continue to exist in the population if $\mathbf{R}_0 > 1$. The endemic equilibrium point (E_1) is locally asymptotically stable if $\mathbf{R}_0 > 1$, which shows the persistence of the disease in the population. The sensitivity analysis of the $\mathbf{R}_0$ is simulated by using Normalized Sensitivity Index (NSI) and Partial Rank Correlation Coefficient (PRCC), which tells the significance of parameters. NSI defines the parameters' local sensitivity by small variation for its baseline, while PRCC offers the global sensitivity by capturing the possible non-monotonic relation of parameters with $\mathbf{R}_0$. We define the positivity and boundedness of the model to maintain biological significance and mathematical well-posedness. It preserves non-negativity of state variables, ensuring population densities, behaviors, and psychological states do not take unrealistic negative values. The use of the Banach contraction principle has demonstrated the model's unique solution. Global stability is described by Hyers–Ulam stability analysis, which measures the system's resilience, robustness, and reliability. We note that due to small deviations in parameters, initial values, or numerical errors, the solutions of the model do not diverge significantly and tend to return to the true trajectory over time. Numerical simulation by varying the fractional order $\aleph$ of the model is carried out to check the non-local behavior of this mutual dynamics because as $\aleph$ varies, the Caputo derivative improves its capacity to capture psychological impacts throughout time. This shows the advantage of capturing the qualitative behavior consistent with physiological patterns, including addiction and depression transmission, pre-influence, demographic impact, and recovery processes over the integer order dynamics. Numerical findings demonstrate that reducing susceptible interaction with addicted individuals, providing initial treatments to depressed individuals, and limiting the influence of social media on addicted individuals are crucial steps in effectively curbing the spread of social media addiction and depression. This can be done by applying self-control and government control distancing measures to get proper sleep, TV ads, physical therapies, and utilizing proper treatment for depressed individuals. Hereditary features and memory traces are utilized to demonstrate the model's disappearing behavior when $\aleph$ approaches 1. Comparing the Caputo fractional derivative results of mutual dynamics with Atangana–Baleanu–Caputo (ABC) fractional derivatives shows consistent non-local behavior and highlights distinct memory properties — Caputo derivative highlights power-law memory, while the ABC derivative captures non-singular, exponential decay memory. Both might be used to describe complicated real-world dynamics, depending on the type of memory effect. This study presents a new perspective on the addiction and depression interplay, focusing on the ABC and Caputo fractional derivatives. As this model provides insight into the transmission process of social media addiction and depression, it requires new tools to reveal hidden behavior in nonlinear epidemiological systems. Future research should use these fractional operators, such as Caputo-Fabrizio, Atangana-Gomez, Atangana beta-derivative, truncated M-derivative, and fractal-fractional. This study can also be explored in the context of the sensitivity and optimal control solution of the proposed model, allowing for the prediction of fractional derivative operator variations.

Part II

Forecasting, Data Augmentation and Control Problem

Chapter 4

Data augmentation in epidemic model with Feed Forward Neural Network and Nonlinear Autoregressive Network

A proof of concept is presented that integrates compartmental disease dynamics models with machine learning to enhance epidemic prediction. By applying data-driven methods and deep neural networks to a Susceptible-Infected-Recovered model featuring social aspects, the study demonstrates improved forecasting capabilities. The results indicate that a strong data augmentation strategy can enhance Feed-Forward Neural Networks and Nonlinear Autoregressive Networks, making them competitive with Physics-Informed Neural Networks. This novel approach addresses nonlinear dynamics and offers scalable solutions for epidemic forecasting. This chapter is based on the study [32].

4.1 Introduction

The COVID-19 pandemic has underscored the need for accurate and computationally efficient predictive models that not only describe the spread of infectious diseases but also provide fast and reliable forecasts to guide public health interventions. A key challenge in epidemic modeling is balancing mechanistic realism with computational tractability. Among the most widely used approaches are compartmental models, such as the classical Susceptible-Infected-Recovered (SIR) models, which describe disease dynamics by partitioning the population into epidemiologically relevant compartments [69, 142, 163]. Specifically, these models categorize individuals as susceptible (capable of contracting the disease), infected (currently infected and capable of transmitting the disease), and recovered (immune or isolated from the population, i.e. people that no longer contribute to disease transmission). Despite their simplicity and effectiveness in capturing some fundamental epidemic trends, classical SIR models assume constant transmission and recovery rates, limiting their ability to represent the complex, nonlinear, and time-varying nature of real-world epidemics.

To address these limitations, extended versions of the SIR model have been proposed in the recent past, incorporating time- and state-dependent transmission rates, saturated incidence functions, and heterogeneous population behaviors [9, 63, 91]. For example, models incorporating saturated or nonlinear incidence rates [99, 42,

92], as well as kinetic approaches to contact dynamics [49], have proven effective in capturing the impact of social distancing measures during the pandemic. These refinements enhance realism and allow for better representation of the diverse factors influencing epidemic trajectories.

In parallel, machine learning techniques, particularly neural networks, have emerged as powerful tools for studying epidemic spread [64, 38, 170]. By leveraging their ability to approximate complex, nonlinear relationships, neural networks complement traditional modeling approaches and extend their applicability. Recent advancements, such as Physics-Informed Neural Networks (PINNs), integrate mechanistic knowledge of underlying physical or biological processes directly into the training phase of neural networks [86]. This integration ensures model robustness and consistency with known dynamics, even when data are scarce or uncertain. For instance, PINNs have been successfully applied to solve forward and inverse problems in epidemic modeling, ensuring that predictions adhere to underlying physical laws [52, 158, 226, 47, 140].

Feed-Forward Neural Networks (FNNs) and Nonlinear Autoregressive Networks (NARs) also offer promising approaches for epidemiological predictions [129, 25, 181]. FNNs map inputs directly to outputs through layers of nonlinear transformations, making them effective for tasks such as interpolation and short-term forecasting. This approach has been applied across various domains, including early recolonization detection in Parkinson's disease, biological parameter estimation in disease models, COVID-19 data analysis, musculoskeletal disorder risk assessment, and statistical modeling for selecting optimal FNN architectures [110, 243, 203, 233, 76, 184]. However, FNNs do not account for temporal dependencies, limiting their ability to model the evolution of epidemics over time.

Conversely, NARs are specifically designed to process sequential data, making them well-suited for time-series forecasting in epidemic modeling. By incorporating previous values of the time series, NARs retain information about past states, enabling them to capture temporal dependencies in epidemic dynamics. These networks have been successfully applied in the recent past to forecast infection peaks, estimate recovery rates, and simulate intervention scenarios [181, 182, 211, 155, 238, 116, 59].

In this work, we introduce a hybrid epidemic forecasting approach that integrates mechanistic epidemiological models with data-driven FNNs and NARs, creating a surrogate model capable of generalizing across different epidemic scenarios. This approach enables fast, data-driven epidemic forecasting while preserving the interpretability of traditional mechanistic models. The resulting methodology is applied to analyze the lockdown and post-lockdown phase of the COVID-19 epidemic in Italy and Spain, leveraging real-world data and enhanced computational strategies to improve predictive accuracy. This framework enables us to augment real-world epidemic data with synthetic simulations generated from a generalized SIR model, which includes a saturated incidence rate to better reflect intervention effects and behavioral changes. The main idea consists of addressing the limitations posed by sparse real-world data by generating additional synthetic information using a generalized SIR data driven model. Specifically, we focus on an extension known as the social-SIR model with a saturated incidence rate, referred to here as the generalized SIR model in the following. This model modifies the transmission dynamics [99] by allowing the infection rate to vary with time and the number of infected individuals. Such modifications enable the incorporation of external interventions, such as lockdowns or vaccination campaigns, as well as psychological and behavioral adaptations [65].

The enriched dataset, combining real and synthetic data, is then used to train the neural networks, allowing them to effectively learn epidemic dynamics and improve predictive accuracy. By leveraging the flexibility of FNNs and the sequential modeling capabilities of NARs, our approach captures both short-term and long-term epidemic trends. Unlike PINNs, which explicitly encode governing equations into the network architecture, our method enables networks to infer dynamics implicitly through data-driven learning. This eliminates the need for embedding differential equations directly into the training process, enhancing adaptability to different scenarios while reducing computational complexity.

The remainder of this paper is structured as follows. In Section 4.2, we recall some standard features about the classical SIR model, and we introduce the social-SIR model with a saturated incidence rate used to produce synthetic data. Section 4.3 defines the optimization problems used to estimate parameters and the incidence function during the pre-lockdown, lockdown, and post-lockdown phases in Italy and Spain. In Section 4.4, we present the neural network structures, distinguishing between FNNs and NARs, and we perform some detailed numerical experiments to validate our strategy. Finally, Section 4.5 summarizes our findings and outlines potential future research directions.

4.2 Problem setting

The governing equations of the standard SIR model, which describes the spread of an infectious disease in a population of N individuals divided into three compartments—Susceptible (S), Infected (I), and Recovered (R)—are given by

$$\begin{aligned}\frac{dS(t)}{dt} &= -\beta \frac{S(t)I(t)}{N}, \\ \frac{dI(t)}{dt} &= \beta \frac{S(t)I(t)}{N} - \gamma I(t), \\ \frac{dR(t)}{dt} &= \gamma I(t),\end{aligned}\tag{4.1}$$

where the total population corresponds to $N = S(t) + I(t) + R(t)$, and the system is complemented with suitable initial conditions. The parameters $\beta > 0$ and $\gamma > 0$ represent the disease transmission rate and the recovery rate, respectively, and are assumed to be constant in (4.1). For simplicity, we also absorb N into the coefficient β in the following.

Despite its simplicity, the above model has been shown to be able to capture some features related to the spread of an epidemic. However, as expected, it also suffers from some drawbacks due to the inability to describe more complex structured solutions, which has been observed, for instance during the last COVID-19 epidemic [125]. In particular, recent studies have highlighted the crucial role of transmission coefficients in relation to social behavior, which significantly influences pathogen spread [67]. In this research direction, the works in [67, 99] explored how controlling the number of daily social contacts can guide partial lockdown strategies. This is a type of phenomena we address in this work and which cannot be captured with the system (4.1). Building on this perspective, some of the authors of the present work have developed in the recent past a new mathematical framework linking social contact distributions with disease transmission in multi-agent systems in [99]. This is achieved by integrating a classical compartmental epidemiological model with kinetic equations governing social contact formation. The resulting model is given

by

$$\begin{aligned}\frac{dS(t)}{dt} &= -\beta S(t)I(t)H(t, I(t)), \\ \frac{dI(t)}{dt} &= \beta S(t)I(t)H(t, I(t)) - \gamma I(t), \\ \frac{dR(t)}{dt} &= \gamma I(t),\end{aligned}\tag{4.2}$$

where $H(t, I(t))$ is a macroscopic incidence rate [99], representing a time-dependent adjustment to infection rates based on behavioral and policy changes (e.g., lockdowns). In the sequel, we consider the following bounded function that moderates infection spread

$$H(t, I(t)) = \frac{1}{1 + \frac{\nu}{\beta} \int_0^t I(s) ds},\tag{4.3}$$

where $\nu > 0$ is a constant that has been shown to reflect the variance in contact rates within the population. The system (4.2), even if built on a different basis, shares conceptual similarities with the models introduced in [69], where a nonlinear, bounded function $g(I)$ modulates disease transmission. This approach accounts for the fact that, as the number of infected individuals increases, the effective infection force may decrease due to factors such as behavioral adaptations or resource limitations. In the same direction as [186] it has been introduced a nonlinear, saturated incidence rate of the form

$$g(I)S = \frac{\beta I^m S}{1 + \alpha I^k}, \quad \text{with} \quad \frac{dS(t)}{dt} = -g(I(t))S(t)\tag{4.4}$$

where $\beta I^m S$ represents the baseline infection force, and $\frac{1}{1 + \alpha I^k}$ accounts for behavioral responses, such as increased caution among susceptible individuals. Here, β, m, k are positive constants, and α is a non-negative constant [83, 272, 246]. A specific case, with $m = 1, k = 1$, yields the form proposed in [69] to model the crowding effect observed during the 1973 cholera outbreak in Bari:

$$g(I) = \frac{\beta I}{1 + \alpha I}.\tag{4.5}$$

Alternatively, setting $m = 1, k = 2$ results in the Monod–Haldane (M–H) function,

$$g(I) = \frac{\beta I}{1 + \alpha I^2},\tag{4.6}$$

which is commonly used to model physiological constraints in infection dynamics.

The so-called social-SIR model (4.2) exhibits a similar incidence rate behavior, extending the form in (4.5) to account for the impact of protective measures such as lockdown strategies and infection control. Unlike classical models, this generalized framework incorporates a time-dependent, saturated incidence rate to capture the cumulative effects of infection dynamics and behavioral adaptations in response to an epidemic. This model has previously been successfully employed to analyze the effects of partial lockdown strategies in various European countries. For the purpose of demonstrating the proposed data augmentation strategy, we select a dataset corresponding to a period marked by COVID-19 lockdowns. It is worth mentioning

that the complexity introduced by lockdown measures makes the forecasting process more challenging compared to standard scenarios. Therefore, in the following, we employ system (4.2) to develop our data-driven strategy and the associated deep learning framework, enabling epidemic forecasting.

4.3 On the the construction of a data driven social-SIR model

In the context of epidemic prediction, mathematical models such as those presented in the previous section provide valuable insights into the spread of infectious diseases. However, these models often rely on predefined assumptions and parameters that may not fully capture the complexities of real-world epidemics. Neural networks offer a powerful alternative by learning directly from data, enabling them to model complex, nonlinear relationships that traditional epidemic models may struggle to represent. However, one major limitation of training neural networks exclusively on available data is that, while they can effectively interpolate within a given time interval, they struggle to make accurate predictions beyond the observed data points [170, 25, 52]. To address this issue, we propose an approach that combines a mathematical epidemiological model—the social-SIR model (4.2)—with real outbreak data. The key idea is to generate additional synthetic information by solving this generalized SIR model, thereby filling the gaps between available real or measured data points. With this enriched dataset, one can train a neural network more effectively to predict future infection trends in a given population. As we demonstrate later, this approach leverages both real and synthetic data, significantly improving predictive accuracy and enhancing the model’s ability to forecast epidemic dynamics. However, to implement this strategy, the model must be capable of interpolating through the experimental data points. Therefore, in the following section, we discuss how the parameters in (4.2) are defined.

4.3.1 Parameters estimation

To develop our approach, we focus on the COVID-19 outbreak in two European countries, Italy and Spain, during the 2020 pandemic. Recognizing that data collection processes inherently introduce errors, particularly due to underreporting of the number of infected individuals in the pandemic’s early stages, we assume an intrinsic uncertainty is embedded in the collected data, capturing the variability and limitations of real-world measurements, [78]. While the development of advanced uncertainty quantification techniques falls outside the scope of this work, it represents a promising avenue for future research. In the present study, we instead adopt the well-established framework described in [99, 9]. Given that both countries implemented restrictive measures during this period, we proceed as follows. We define two optimization phases: in the first phase, we estimate the parameters β and γ for the pre-lockdown period using the model (4.2) with $H(t, I(t)) = 1$; in the second phase, we estimate the function $H(t, I(t))$ from lockdown and post-lockdown data.

The COVID-19 data for Italy are available from the [GitHub repository](#) of the Italian Civil Protection Department, while data for Spain can be accessed [here](#). In practice, to estimate the parameters β and γ , we formulate the following minimization problem:

$$\min_{\beta, \gamma} \mathcal{J}(\hat{I}, I), \quad (4.7)$$

subject to the constraint that the equations of system (4.2) have to be satisfied when $H(t, I(t)) = 1$. The objective function to be minimized is defined as

$$\mathcal{J}(\hat{I}, I) = \|\hat{I}(t) - I(t)\|^2. \quad (4.8)$$

where $\hat{I}(t)$ represents the reported infected cases from the data set at disposal, while $I(t)$ is the model output, and $\|\cdot\|$ denotes the L_2 norm. Throughout the manuscript, the variables S , I , and R and the data are assumed to be normalized and to denote fractions of the total population.

We consider data over the time interval $[t_0, t_l]$, where t_0 (set to February 24, 2020) marks the initial time, and t_l represents the lockdown start date (March 9 for Italy and March 14 for Spain). We search for a minimum under the constraints

$$0 \leq \beta \leq 0.6, \quad 0 \leq \gamma \leq 0.06, \quad (4.9)$$

where the bounds are chosen according to the available estimates [125]. To solve the optimization problems in (4.7), we rely on the use of the Matlab functions `fmincon` in combination with an RK4 integration method of the system of ODEs. The result of the optimization procedure on the pre-lockdown phase gives, for Italy, the following values

$$\beta_{est}^{It} = 0.3107, \quad \gamma_{est}^{It} = 0.0328, \quad (4.10)$$

while for Spain

$$\beta_{est}^{Sp} = 0.2340, \quad \gamma_{est}^{Sp} = 0.0224. \quad (4.11)$$

Both the estimated transmission rates β_{est} and the recovery rates γ_{est} have units of inverse time (e.g., day^{-1}), ensuring the correct dimensional consistency of the model equations. In Figure 4.1 on the left the evolution of the dynamics in the pre-lockdown phase in Italy obtained by solving the social-SIR model (4.2) with $H(t, I(t)) = 1$, using the estimated parameters $\beta = \beta_{est}^{It}$ and $\gamma = \gamma_{est}^{It}$ in (4.10). This is compared, in the same image, to the experimental data from the pre-lockdown phase, spanning up to time $t_l = 14$ with a time step $\Delta t = 1$ corresponding to 14 days with 1 day interval between the measurements. The estimated dynamics align quite well with the available data, as confirmed by the embedded plot, which shows the pointwise error over time between the solution of the generalized SIR model and the data at disposal, i.e.

$$\mathcal{E}(t) = |\hat{I}(t) - I(t)|. \quad (4.12)$$

In the same Figure on the right the pre-lockdown dynamics in Spain computed by solving the social-SIR model (4.2) with $H(t, I(t)) = 1$, using the estimated parameters $\beta = \beta_{est}^{Sp}$ and $\gamma = \gamma_{est}^{Sp}$ in (4.11) compared with the experimental data spanning up to time $t_l = 18$. The two solutions are in good agreement, as confirmed by the plot showing the pointwise error computed as in (4.12).

Once β and γ have been estimated, we proceed to determine the pointwise values $H(t, I(t))$ in (4.2) in such a way that the model fits as well as possible the lockdown and post-lockdown data, i.e., data in the interval $[t_l, T]$, where T (set to July 1st) is the final observation time for both countries. To this end, we solve the following new optimization problem

$$\min_{H(t)} \mathcal{J}(\hat{I}, I), \quad (4.13)$$

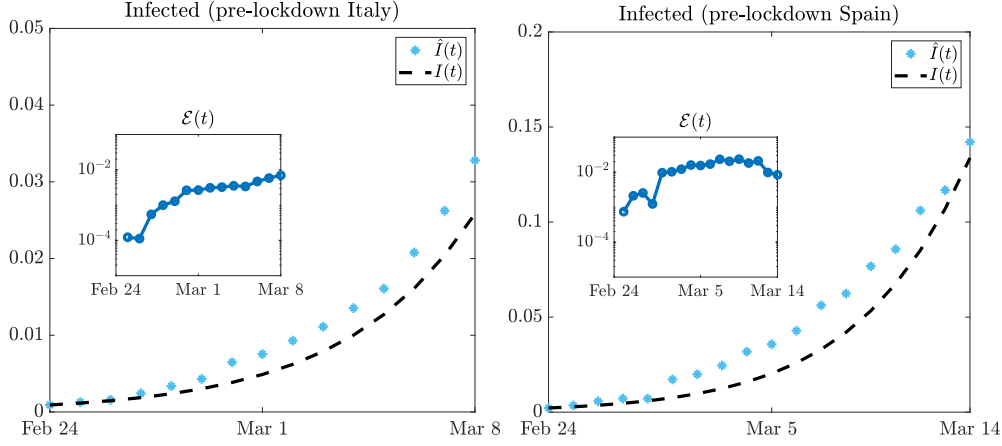


FIGURE 4.1: Pre-lockdown phase. Dynamics of the infected population computed by solving the social-SIR model (4.2) with β and γ as in (4.10) (Italy), and as in (4.11) (Spain) and $H(t, I(t)) = 1$, compared with the experimental data. In the embedded plot, the pointwise error between the two quantities is computed as in (4.12). On the left, Italy. On the right, Spain.

subject to the constraint that the solution has to satisfy the equations of the system (4.2), with $\mathcal{J}(\cdot)$ defined as in (4.8), and that

$$0 \leq H(t) \leq 1. \quad (4.14)$$

In order to determine the optimal value of H , we split the time interval $[t_l, T]$ into N_t time intervals of length Δt , and we solve a sequence of optimal control problems

$$\min_H \mathcal{J}^{\Delta t}(\hat{I}, I) := \int_t^{t+\Delta t} \|\hat{I}(s) - I(s)\|^2 ds, \quad (4.15)$$

s.t. the explicit time discretization of the generalized SIR dynamics

$$\begin{aligned} S^{n+1} &= S^n + \Delta t \beta I^n S^n H^n, \\ I^{n+1} &= I^n + \Delta t (\beta I^n S^n H^n - \gamma I^n), \\ R^{n+1} &= R^n + \Delta t \gamma I^n, \end{aligned} \quad (4.16)$$

where we denoted by $t^n = n\Delta t$ for any $n = 1, \dots, N_t$. We then consider a discretization of the integral in (4.15) using the rectangle rule

$$\mathcal{J}^{\Delta t}(\hat{I}, I) = \Delta t (\hat{I}^{n+1} - I^{n+1}). \quad (4.17)$$

By direct computation, it can be shown that the expression for H at each discrete time t^n is given by

$$H^n = \mathbb{P}_{[0,1]} \left(\frac{\hat{I}^n - I^n + I^n \gamma \Delta t}{I^n S^n \beta \Delta t} \right), \quad (4.18)$$

where $\mathbb{P}_{[0,1]}(\cdot)$ denotes the projection over the interval $[0, 1]$. As demonstrated later, the function H computed as in (4.18) tends to exhibit oscillatory behavior over time. To reduce these oscillations, we propose to replace the projection operator $\mathbb{P}_{[0,1]}(\cdot)$

in equation (4.18) with a smoother approximation of the type

$$S(x) = \frac{1}{1 + e^{-kx}}, \quad (4.19)$$

for a certain $k > 0$, and $x \in \mathbb{R}$. This modification helps to regularize the value of H . The results of this study are reported in Figure 4.2 where on the left we show the lockdown and post-lockdown experimental data and the solution of the generalized SIR model with the contact function H as in (4.18) and, alternatively, with the smoothed version (4.19) setting $k = 1.6$. The estimated and true solutions are in good agreement as shown in the embedded plot, which represents the corresponding pointwise error computed as in (4.12). On the right, we plot the estimated values of H^n over time, comparing the values obtained from the original projection-based approach and the smoothed operator. The function H computed as in (4.18) exhibits oscillations over time, suggesting potential measurement errors in the data, such as a non-exponential decay of the number of infected, especially in the late post-lockdown phase, mimicking the intrinsic uncertainty that may affect data. These oscillations are significantly reduced when using the smoothed operator. However, a reduction in the accuracy of the numerical solution can be observed. First row, Italy. Second row, Spain.

4.4 Estimation and prediction using real COVID-19 data and Neural Networks design for epidemic prediction

In this section, we consider two distinct types of neural network architectures: Feed-Forward Neural Networks (FFNs) and Nonlinear Autoregressive Networks (NARs). Both approaches are implemented using a general neural network framework with $L + 1$ layers, as follows

$$\begin{aligned} x^1 &= W^1 x + b^1, & x^l &= \sigma \circ (W^l x^{l-1} + b^l), \quad l = 2, \dots, L-1, \\ U^{NN}(x; \mathbf{b}, \mathbf{W}) &= W^L z^{L-1} + b^L, \end{aligned} \quad (4.20)$$

where $x = (x^1, \dots, x^d) \in \mathbb{R}^d$ is the input, σ is an activation function to convert the input signals to output signals, and $\mathbf{W} = (W^1, \dots, W^L)$ and $\mathbf{b} = (b^1, \dots, b^L)$ represent the weights and biases. The weights and biases associated with these connections serve as the parameters of the network, adjusted iteratively through techniques like gradient descent or Adam method during the training phase to minimize prediction errors and enhance model performance. In our context, the idea is to solve the following minimization problem

$$\theta^* = \arg \min_{\theta} \mathcal{L}(\theta), \quad (4.21)$$

to find the optimal parameters $\theta = (W^1, b^1, \dots, W^L, b^L)$ which minimize a certain loss function $\mathcal{L}(\theta)$ that includes errors from both real and synthetic data generated by solving the generalized SIR model. Real data provides an accurate representation of the epidemic dynamics, while synthetic data fills gaps between observed data points and provides high-resolution trajectories.

The loss function is expressed as

$$\mathcal{L}(\theta) = \omega_r \mathcal{L}_r(\theta) + \omega_s \mathcal{L}_s(\theta), \quad (4.22)$$

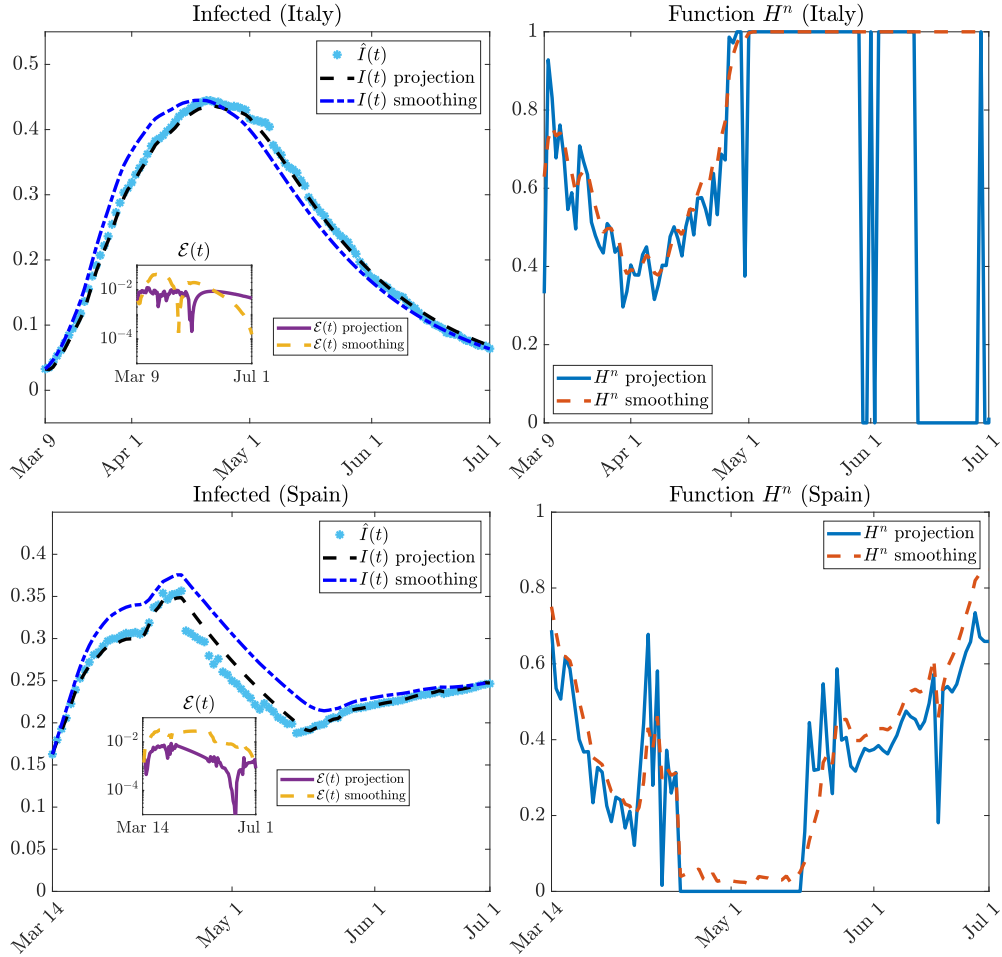


FIGURE 4.2: Lockdown and post-lockdown phase. On the left the dynamics of the infected population computed by solving the generalized SIR model (4.2) with β and γ as in (4.10) (Italy), and as in (4.11) (Spain), assuming the contact function H to be defined as in (4.18) and, alternatively, with the smoothed version (4.19) setting $k = 1.6$ compared with the experimental data. In the embedded plot the pointwise error between the two infected populations is computed as in (4.12). On the right the contact function H is computed as in (4.18) and its smoothed version is computed introducing an operator as in (4.19). First row, Italy. Second row, Spain.

where $\omega_r, \omega_s > 0$ are given weights, and

$$\mathcal{L}_j(\theta) = \frac{1}{N_j} \sum_{i=1}^{N_j} \|I_j(t) - I_j^{NN}(t, \theta)\|^2. \quad (4.23)$$

for $j = \{r, s\}$, where $I_j(t)$ is the actual number of infected. These are either the real data or the data generated by solving the social SIR model (synthetic data), while $I_j^{NN}(t, \theta)$ is the predicted state. Finally, N_r, N_s refer to the number of available real and synthetic data points respectively. With this choice, it is possible to appropriately weight the contribution of real data alongside the data generated by the model.

Feed-Forward Neural Networks. FFNs are a foundational type of neural network architecture designed to map inputs directly to outputs without considering temporal dependencies. For epidemic prediction, we design a FFN that takes the current time t as input and produces the value of infected $I(t)$ as output. This approach relies on learning a direct mapping between the time and the epidemic dynamics, effectively interpolating the trajectory based on the training data. The architecture of the FFN consists of the following components

- **Input Layer:** The single input feature is the current time t .
- **Hidden Layers:** One or more fully connected layers with nonlinear activation functions (e.g., ReLU or tanh) capture the relationship between time and the epidemic states.
- **Output Layer:** The output consists of a node corresponding to the predicted value of the infected at time t , i.e. $I(t)$.

It is well known that, while FFNs are effective for interpolation tasks, their inability to model sequential dependencies limits their accuracy in predicting future epidemic dynamics. This limitation arises because FFNs lack memory mechanisms, meaning they cannot leverage information from previous states to influence current predictions. For this reason, in the following we propose an alternative strategy.

Nonlinear Autoregressive Networks. To capture the temporal dynamics of the epidemic, we then rely on Nonlinear Autoregressive Networks (NARs) capable of modeling sequential data. Unlike FFNs, NARs incorporate information about past states, making them particularly effective for time-series predictions. The NAR is designed to predict the future state of the epidemic based on the previous state of the solution. The architecture consists of the following components:

- **Input Layer:** The input features are the values of the infected at time $t - d, \dots, t - 1$, i.e. $I(t - d), \dots, I(t - 1)$ for a certain $d \geq 1$.
- **Hidden Layers:** One or more fully connected layers with nonlinear activation functions (e.g., ReLU or tanh) capture nonlinear relationships in the time series data.
- **Output Layer:** Produces the predicted next state, $I(t)$, representing the system's state at time t .

Once trained, NAR networks can predict the future states of the epidemic compartments by recursively feeding its outputs back as inputs for subsequent time steps (closed loop strategy). This enables the generation of full epidemic trajectories from a given initial condition.

4.4.1 Neural network for epidemic prediction

To evaluate the performances of FFNs and NARs in epidemic prediction, we conduct some numerical experiments using both real and synthetic data. The real data covers the period from the lockdown start date (in Italy March 9th, in Spain March 14th) to July 1st. These data are available on a daily basis, corresponding to a time step of $\Delta t_r = 1$. The synthetic data are generated by solving the generalized SIR model given in (4.2), with β and γ as in (4.10) (Italy), and as in (4.11) (Spain) over the same time interval, using a finer time step $\Delta t_s = 0.2$, and with H estimated by solving the minimization problem in (4.13). In the following, we will focus on the solution generated by solving the social-SIR model with H as in (4.18) to generate synthetic data. The version with the smoothing operator is not considered further due to the loss of accuracy it introduces. Furthermore, since H is originally defined at daily intervals as in (4.18), we use linear interpolation to extend its definition to the finer grid. Both networks are trained using the Levenberg-Marquardt optimizer for 500 epochs. In the case of real data we set $\omega_r = 1$ and $\omega_s = 0$, while in the case of synthetic data we set $\omega_r = 0$ and $\omega_s = 1$ in (4.22).

Feed forward neural network

We design a FFN with three fully connected layers, each consisting of 10 nodes, and use the tanh activation function, relying on the use of the Matlab function `feedforwardnet`. The input to the network is the time interval $[t_l, T]$, being t_l the lockdown starting date and T the final time supposed to be July 1st. The output is the normalized number of infected $I(t)$, which corresponds to either real data or synthetic data generated as described above. We train the neural network using the Matlab function `train`. To evaluate the neural network's performance, we first compute its solution over the training set for both Italy and Spain. Specifically, we consider the time interval $[t_l, T]$ with a time step $\Delta t_r = 1$. In the left panel of Figure 4.3 and of Figure 4.4, we compare the solution obtained using a neural network trained on both real and synthetic data, with the available data for respectively the Italian and the Spanish case. The neural network provides a good approximation of the solution, making it well-suited for interpolation tasks. Next, to assess its predictive capabilities, we focus on the time interval $(T, T_{test}]$, where T (July 1st) is the starting time and T_{test} (July 15th) is the final time, using the same daily discretization. The right panels of Figures 4.3 and 4.4 present a comparison between the network's predictions for the test set and the daily real data available in that period. Additionally, the figures display the pointwise error between the neural network's outputs and the real data, calculated as in (4.12). The standard FNN as expected faces challenges in forecasting future dynamics and the data augmentation does not provide a clear improvement in the results.

Nonlinear autoregressive neural network

We design now a NAR network with three fully connected layers, each consisting of 10 nodes, and use the tanh activation function. To implement the neural network we use the Matlab function `narnet`. We consider a time interval $[t_l, T]$, being t_l the time in which the lockdown started and T the final time supposed to be July 1st. We discretize the interval with time step $\Delta t_r = 1$ if we deal with real data, or with time step $\Delta t_s = 0.2$ if we deal with synthetic data as before. The input to the network is the number of infected at time $t - 2\Delta t, t - \Delta t$, and the output is $I(t)$ that is the number of infected at time t . Both the input and output data are generated as

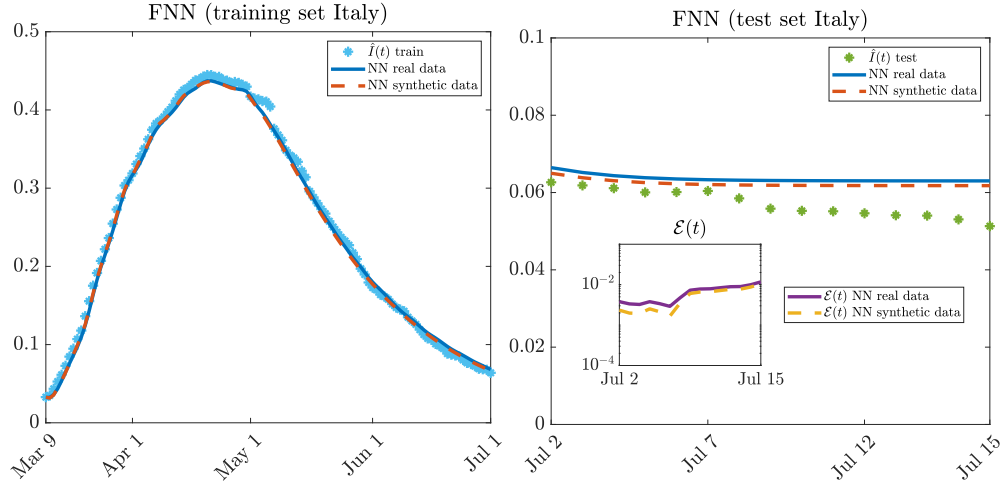


FIGURE 4.3: Feedforward neural network for Italy. On the left the dynamics of the infected population computed by training a FNN on real and synthetic data and tested over the train interval $[t_l, T]$ compared to the available data. On the right the solution computed with the same FNN tested over the time interval $(T, T_{test}]$ compared to the available data. Embedded the error between the available data test and the neural network solution computed as in (4.12).

described above by considering a social-SIR model. As for the FNN we first evaluate the performance of the NAR network on the training set. Since the number of infected individuals during the training interval is known, we can use an open-loop prediction approach using the true training set values to forecast the next time step's solution. Figure 4.5 shows the solution obtained by training a NAR network on both real and synthetic data, then testing it on the same dataset and comparing it to the available data. On the left, we have the results for Italy, and on the right, for Spain. Both the NAR networks, trained on real and synthetic data, are able to accurately approximate the underlying dynamics when one considers the training interval. We then test the NAR network for both short-term and long-term predictions. For short-term predictions, we compare the neural network solution with a test set containing daily data from July 2nd to July 8th for both Italy and Spain, while for long-term predictions, we extend the test period to July 15th. Since these data are assumed to be unknown a priori, we convert the open-loop NAR network into a closed-loop form. This allows the network to generate predictions for the desired number of steps by using its previous predictions as input, without relying on the true values for evolution. Figure 4.6 shows the solution produced by the NAR network trained on both real and synthetic data, compared with the available true data. Additionally, we plot the pointwise error in time computed as in (4.12). In the first row, the short-term prediction while in the second row, the long term prediction. On the left, Italy. On the right, Spain. The NAR network trained on synthetic data is able to provide a very accurate approximation of the solution and demonstrates strong predictive capabilities. Notably, it significantly outperforms the results obtained when training exclusively on real data, as it is particularly evident in the case of Italy.

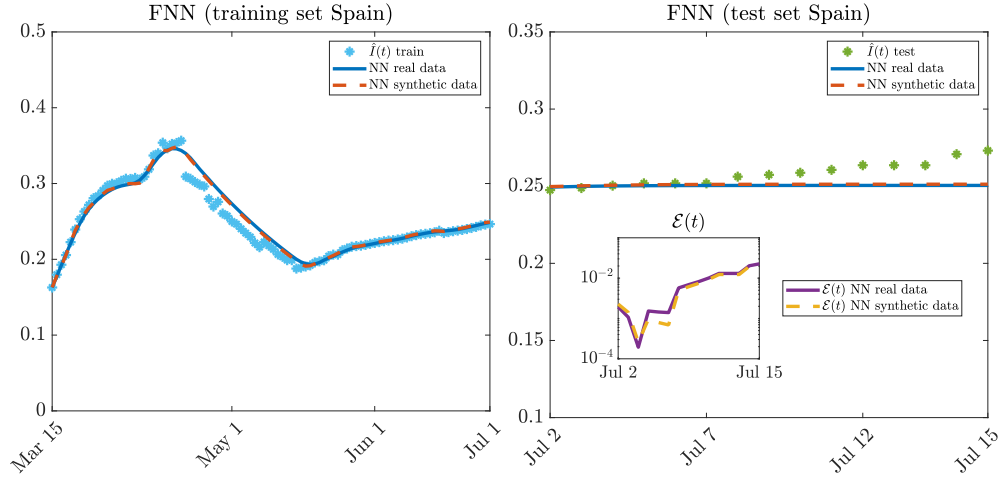


FIGURE 4.4: Feedforward neural network for the Spain case. On the left the dynamics of the infected population computed by training a FNN on real and synthetic data and tested over the train interval $[t_l, T]$ compared to the available data. On the right the solution computed with the same FNN tested over the time interval $(T, T_{test}]$ compared to the available data. Embedded the error between the available data test and the neural network solution computed as in (4.12).

First row, Italy. Second row, Spain.

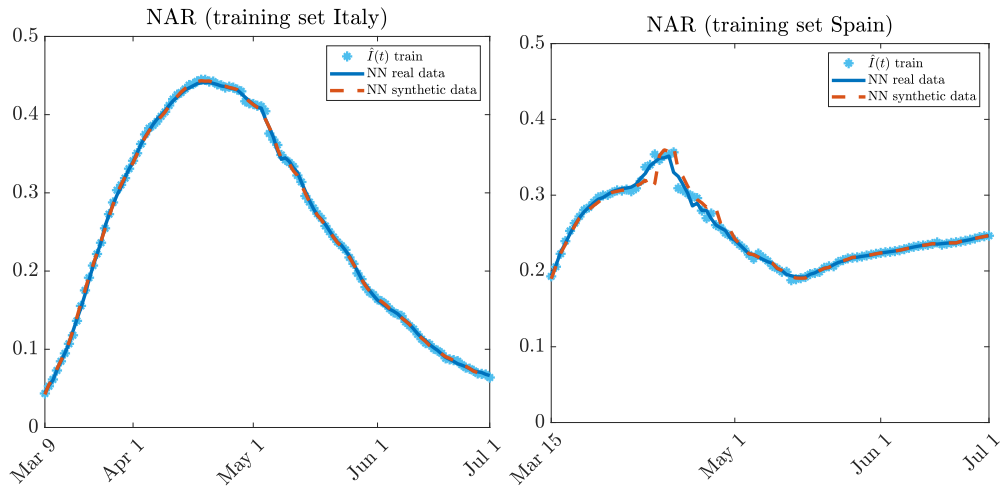


FIGURE 4.5: Nonlinear autoregressive network: training data. Solution obtained by training a NAR network on both real and synthetic data, then testing it on the same dataset and comparing it to the available data. On the left, the case for Italy, and on the right, the one for Spain.

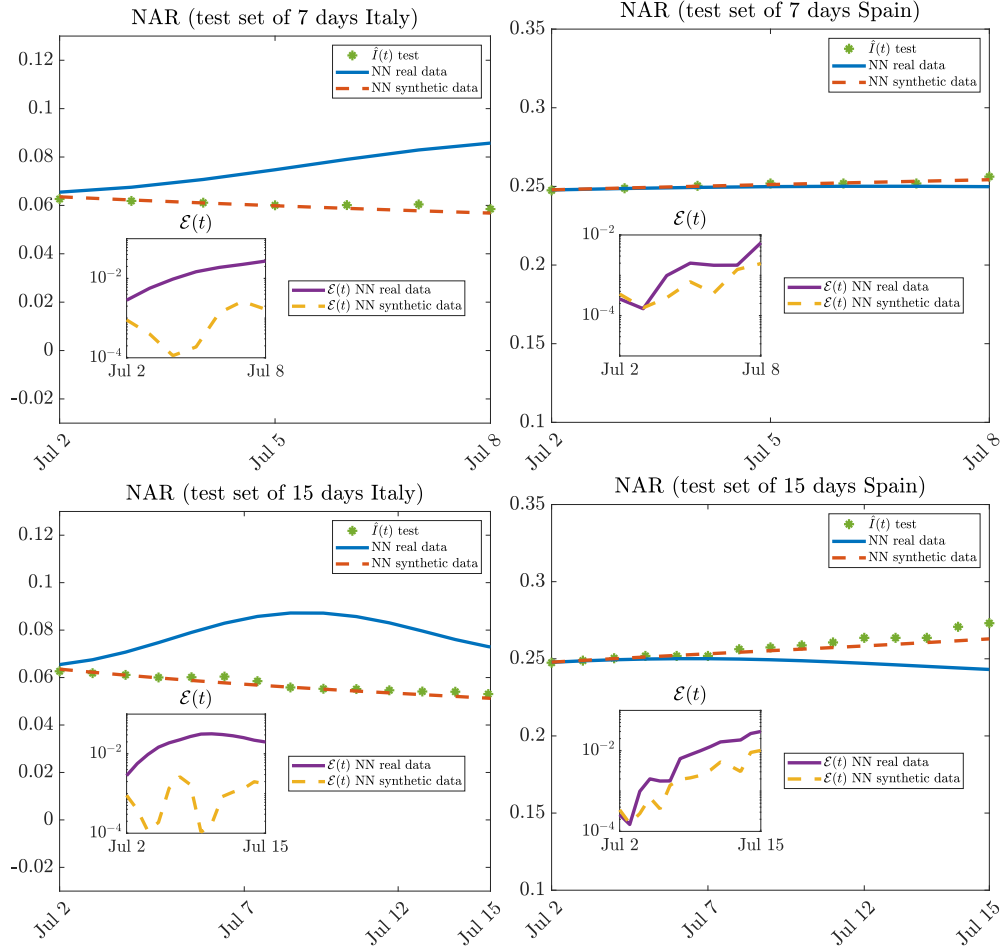


FIGURE 4.6: Nonlinear autoregressive network: short and long time predictions. Solution obtained by training a NAR network on both real and synthetic data, then testing it to produce daily data from July 2nd to July 8th (first row), and extend to July 15th (second row), compared with the available true data. On the left, the case for Italy, and on the right, the one for Spain.

4.5 Conclusion

In this study, we explored the impact of a data augmentation strategy on enhancing the forecasting capabilities of Feed-Forward Neural Networks and Nonlinear Autoregressive Networks in analyzing and predicting the dynamics of the COVID-19 epidemic during the lockdown and post-lockdown phase in Italy and Spain. By integrating real data with synthetic data generated from a generalized SIR model, we demonstrated the effectiveness of neural networks in capturing complex epidemic dynamics. FNNs performed well in interpolating within observed data, while NARs were particularly effective in capturing temporal dependencies, making them suitable for long-term forecasting. The generalized SIR model provided a robust foundation for generating high-resolution synthetic data, enriching the neural network training process. The key outcome of this work is the development of a hybrid methodology that bridges the gap between traditional mathematical modeling and machine learning techniques, such as Physics-Informed Neural Networks, offering a scalable and flexible framework for epidemic modeling and prediction in real-world scenarios.

While this study has focused on FNNs and NARs, other neural network architectures could be explored to further improve epidemic forecasting. For example, Long Short-Term Memory (LSTM) networks and Transformer-based models have been increasingly used for time-series prediction and could provide additional advantages in learning long-term dependencies [25, 212, 58]. Similarly, hybrid AI-mechanistic models, which integrate deep learning with traditional compartmental models, have shown promise in epidemic modeling [291]. Future research could investigate whether these approaches provide added value in capturing epidemic trends compared to the models used in this study, particularly in scenarios with limited or noisy data.

An important direction for future research is the incorporation of uncertainty quantification (UQ) to better account for variability and noise in real-world data [51, 9, 68, 78]. While neural networks provide powerful forecasting tools, their deterministic nature limits their ability to express confidence in predictions, which is crucial in public health decision-making. Approaches such as Bayesian deep learning, Monte Carlo Dropout, and ensemble learning can be explored to quantify the range of possible epidemic trajectories. Additionally, integrating probabilistic surrogate models with mechanistic epidemiological frameworks can improve risk assessment and early-warning systems for future outbreaks. By embedding UQ techniques within the proposed hybrid framework, epidemic forecasting can become more robust, interpretable, and reliable to better account for variability and noise in real-world data.

Chapter 5

Parameters identification and optimal control of Behavioral dynamics using Physics Informed Neural Network

Studying the dynamics of addiction and depression is vital, especially among adolescents and young adults in today's digital landscape. This work presents a deterministic model that explores the interplay between these two conditions, incorporating effective treatment strategies. It highlights the increased suicide risk due to cyber toxicity and utilizes a data-driven approach with Feed Forward Neural Network (FNN) and Physics Informed Neural Network (PINN) to estimate model parameters from real data, validating it against synthetic data to ensure robustness. An optimal control model is developed to mitigate the adverse effects of addiction-depression dynamics in communities, analyzing various intervention strategies based on Pontryagin's minimal principle, laying the groundwork for effective prevention and control of social media addiction and depression dynamics.

5.1 Introduction

In the modern age, social media is very significant since it has an impact on numerous areas of communication, culture, and society through connectivity, information dissemination, cultural exchange, influencing public opinion, personal branding, business marketing, entertainment, social activism, privacy concerns, educational resources, etc. Social media use is closely associated with a variety of emotional, relational, physical, and performance-related issues. Caution should be expressed regarding the wider adoption of social networking sites, especially in view of the fact that people are spending a lot of time on them [101, 113, 107, 296]. People utilize social sites like Google, Instagram, Facebook, YouTube, Twitter, etc, in a variety of contexts based on their personal interests. Effective use of social media could contribute to the development of critical skills, including problem-solving, self-directed learning, information access, and business, but those who misuse it have negative impacts on it. Addiction to social media is one of the primary elements causing more serious adverse consequences. People who spend excessive amounts of daily working hours on these social sites become social media addicts and feel anxiety whenever they do not get access to these sites [145, 259, 34].

Although it has a lot of advantages, it's important to use it sensibly and be careful of any possible negative aspects, such as risks associated with cyberbullying,

privacy concerns, overload of data, and media addiction. Multiple research studies conducted across the world led to diverse findings about the spread of Internet addiction. For example, a study in the UK found that 18% young people suffer from addiction to social media. It is not just an issue on college campuses; students in middle and high schools are also affected. A Hong Kong-based longitudinal study found that about 26.7% of high school pupils were addicted to the Internet [303]. Scientists caution that social media may significantly harm teens by creating misunderstandings about how easy it is to establish and end relationships. Research by [227] highlights an obvious connection between social media use and educational outcomes, indicating an adverse effect on academic performance among social media users. The investigation also emphasizes the growing trend of using social media for communication with others rather than for academic goals.

All the studies indicate continue engagements with social media cause low self-esteem, disappointment, loneliness, isolation, social anxiety disorder, fear of missing out, decreased physical activities, disturbed sleep patterns, poor work performance, negating relationships in real life, decreased capacity for compassion, anger, sorrow, poor effectiveness assessment, and depression [152, 119, 208]. There has been significant research and concern about the link between depression and addiction states. Overuse of social media has been linked to a variety of psychological disorders, including depression. It can exacerbate depression symptoms by creating feelings of loneliness, inadequacy, and poor self-worth. The World Health Organization (WHO) published an article in 2019 that addressed the possible drawbacks of extended use of social media. The report included study results showing that excessive social media use may contribute to mental health problems such as stress and depression [133].

Various studies and reports suggest that depression is a result of media addiction. Currently, there are more than seven million American women who meet the criteria for clinical depression. Even after accounting for fluctuations in self-reporting behavior across genders, a reliable study indicates that women are over twice as likely as men to have clinical or sub-clinical depression [146, 183]. However, it's important to note that the connection between social media addiction and depression has to be investigated further because it is very complex and each individual's perspective is distinct. In order to prevent more negative effects, coupled addiction-depression dynamics must be treated and approached like any other disease or epidemic. Workshops and education activities on the detrimental impacts of social media are viewed as a kind of control. Clinical depression therapy, guidance, and advertising, and therapeutic strategies, including turning off notifications, setting time limits, limiting internet usage, and avoiding smart-phone use just before bed, are some of the current treatments. As with addictions to alcohol, cigarettes, video games, etc, coupled addiction-depression epidemic is actually an addictive disease with depression. Mathematical models are essential for understanding the real-world techniques for anticipating and controlling the dynamics of epidemics [209, 79, 13].

In order to address alcohol issues, [192] proposed a mathematical model that includes time delay and awareness training. After analyzing the results of these actions, it is determined that awareness campaigns are successful in reducing alcohol-related issues. Follows [210] talk about a mathematical model that looks at the patterns of methamphetamine addiction treatment, both inpatient and outpatient, in the Western Cape region of South Africa. The study [176] developed a model of online gaming addiction. Using Pontryagin's maximum principle, they provided the numerical findings of the optimum control model and talked about the stability of the

steady state in their study. Also [185] examined a synthetic drug transmission model involving treatment and relapse, as well as its existence and backward bifurcation. To determine ways to reduce the spread of the aforementioned addictions while minimizing implementation costs, authors utilized optimal control approaches [136, 293, 12]. The latest work [15] studies the Social media addiction and depression model by examining the Threshold parameter R_0 , local & global stability of equilibrium states, and bifurcation of the model. The simulation results indicate that with the increase in addicted individuals, depressed individuals also increase. Refs [195, 196] proposed a model to examine the social media addiction with peer-influence relapse and dual perspectives of individual needs or caring. In 2025, [193] investigated the connection between depression and social media addiction by integrating gamma delay and Dirac delta distributions to evaluate the stability of equilibria and the presence of Hopf bifurcation. Recent study [218] uses mathematical modeling to analyze depression trends, identifying primary and secondary depressed populations and saboteurs who may influence depression prevalence while [161] investigates the SEAR social media addiction model with optimal control to provide the information for prevention measures for the individuals.

All existing models analyzing the link between social media addiction and depression primarily focus on individual behavior disorders but exhibit significant limitations. They fail to provide insights into the incidence rate of depression linked to social media use, which is essential for monitoring the emergence of cases over time. The overlap between social media interaction and depression symptoms complicates the assessment of health campaigns aimed at addressing these issues, making it clear that tailored prevention measures are needed for each condition. Moreover, these models do not adequately capture the phenomenon of relapse in depression treatment, as many individuals abandon their treatment after initial improvements, which affects overall treatment strategies. The influence of media peer pressure on those suffering from depression is significant, as it exacerbates suicidal tendencies, a factor commonly linked to cyberbullying; however, this aspect remains unexamined in literature's models. In this work, we developed a mathematical model of couple addiction-depression dynamics that studied the dual transmission rate for both physiological states of each individual, and internet harassment may increase the suicide risk, significantly complicating the behavioral dynamics. The model consists of susceptible, exposed, addicted, depressed, recovered, quitter, and suicide classes. We utilize separate treatments for depression and addiction, and effectively evaluate the relapse phenomena in depressed populations. Suicide compartment give the critical aspects of how internet scams, fake lifestyles, and cyberbullying increase the severity of behavioral dynamics. A key contribution of this study is to define the global sensitivity analysis of the model through the Partial Rank Correlation Coefficient (PRCC) and identify the significant parameters that can be effective in changing the behavioral dynamics. The global stability of the model system was accomplished using an extended Lyapunov and Castillo-Chavez theorem to predict the long term evaluation stability. Another aim of this work is to address the coupled addiction-depression behavior disorder. An optimal control model derived to manage their worst impact in the community, considering Pontryagin's minimal principle, we simulate the impact of various control strategies using the prevention and treatment controls. This provides an empirical foundation for the prevention and control of behavioral dynamics. A data-driven model constructed with actual data used to observe the depression dynamics emerges with social media addiction. Due to uncertainty in observed data and the selection of some parameters from literature, parameter validation is a critical factor. We utilize

the role of a deep learning neural network within *Feed Forward Neural Network* (FNN) and comparative analysis with *Physics Informed Neural Network* (PINN) to precisely forecast and parameter identification of the model.

The paper is structured in the following manner. In Sec 5.2, the formulation of coupled addiction-depression dynamics is presented, whose complex nature provides a realistic perception of behavioral dynamics in the community. Sec 5.3 focuses on the basic properties of compartment models, through investigation of qualitative analysis, including the stability and the fundamental reproduction number of the model. To check the model's long-term behavior, local and global stability were explored. The well-posedness of a model is defined by demonstrating the existence and uniqueness of model solutions. All the theoretical aspects indicated that model formulation is biologically meaningful. Sec 5.4 explicitly explores the estimation of biological parameters using real data of depression dynamics. To validate the proposed model's accuracy and credibility, modern machine learning tools, including FNN and PINN, were implemented as a data-driven process. This increases the parameter's biological significance through literature learning mechanics. In Sec 5.5 we perform the global sensitivity analysis of the model, giving the significance information about the influential parameters, which have the capacity to control the propagation of infection in the population. Sec 5.6 comprises the formulation of the optimum control strategies and the optimality system of the model to control the behavioral dynamics. Alternatively, Sec 5.7 presents numerical simulations of the proposed model and respective control evaluation, truly capturing the coupled addiction-depression dynamics behavior and providing effective strategies for policymakers. In Sec 5.8, we finally provide our conclusions.

5.2 Model formulation

Here, we presented a mathematical model to explore the interaction between depression and addiction dynamics through the use of social media. The model is divided into seven classes: $S(t)$, $E(t)$, $I_1(t)$, $I_2(t)$, $R(t)$, $Q(t)$ and $D(t)$ while total population $N(t)$. Figure 1 shows the schematic diagram of Model (1), and Table (1) contains all the parameter values used to simulate the model results.

Susceptible class $S(t)$:

$S(t)$ refers to the susceptible population, containing all social media users. We consider the recruitment rate Γ over time t . The susceptible population decreases when they interact with the addicted population at a rate λ_1 and with the depressed population at a rate λ_2 . After this interaction, they become exposed, due to which the exposed population increases, and diseases are transmitted to the individuals. Also, with the initial condition, the pre-recovered individuals are ready to make new susceptible individuals at a rate of π and leave the recovered class with a rate θ . Some individuals instantly follow some prevention and stop or avoid the use of social media and move to a permanent quieter class at a rate β . Also, there is some loss in susceptibility in terms of the mortality rate ξ .

$$\frac{dS}{dt} = \Gamma + \pi \theta R - \lambda_1 \frac{I_1 S}{N} - \lambda_2 \frac{I_2 S}{N} - (\beta + \xi) S.$$

Exposed class $E(t)$:

$E(t)$ shows the individuals who have been exposed to SMA but are still not infected. Because we are modeling the scenario in which infection is caused by public daily

routine activities like the use of internet things, which lead to addiction, we have to add an exposed class. The exposed population increased at a rate of λ_1 and λ_2 when susceptible to contact with the addicted and depressed population, respectively. This population decreases at a rate of γ , which can be referred to as the average length of the Latin period, and individuals begin to move to the addiction population. Also, they decrease w.r.t the mortality rate ξ .

$$\frac{dE}{dt} = \lambda_1 \frac{I_1 S}{N} + \lambda_2 \frac{I_2 S}{N} - (\gamma + \xi) E.$$

Addicted class $I_1(t)$:

$I_1(t)$ represents the people who become addicted to the excessive use of social media. The addicted population increases with the number of individuals exposed who later become addicted at a rate ν . The important factor we will analyze in this class is the effect of treatment rate on the addicted population. When initial prevention measures are taken from the addicted individuals, like turning off mobile notifications, using social media once a day for a fixed duration, not using smart phones in bed, etc, and later taking some physiotherapy like cognitive behavioral therapy (CBT) for the seriously addicted individual, then the individuals recovered at a treatment rate of ϕ_1 and moved to recover class. They decrease with respect to the mortality rate ξ . Also, there is pressure from social media on some individuals of the addicted population who move to the depressed class at a rate α .

$$\frac{dI_1}{dt} = \nu \gamma E - \phi_1 I_1 - (\xi + \alpha) I_1.$$

Depressed class $I_2(t)$:

$I_2(t)$ indicates all the individuals who are depressed by the excessive use of social media or peer pressure from the media. The depressed population increased with the rate $\phi_1 (1 - r_1)$ when addicted individuals do not recover with the initial treatment effect. Also, many people who are social media addicts become victims of cyberbullying, which refers to the use of social/communication tools to harass, threaten, or abuse someone, which can happen at any time. So all depressed individuals go through a complete treatment process at a rate of ϕ_2 and after successful treatment move to the recovered class. Also, they decrease w.r.t the mortality rate ξ .

$$\frac{dI_2}{dt} = \alpha I_1 + \phi_1 (1 - r_1) I_1 + q R - \phi_2 I_2 - (\mu + \xi) I_2.$$

Recovered class $R(t)$:

$R(t)$ describes the individuals who have recovered from the disease. The recovered population increases with the effective treatment rate $\phi_1 r_1$ and ϕ_2 for the addicted and depressed populations. The notable factor is the relapse parameters q . When depressed individuals are treated at a rate of ϕ_2 , then some individuals leave the treatment after some improvement and try to overthink. In such people, due to negative thinking patterns, biological factors, anxiety, mental health disorders, etc, the addiction symptoms return after some time of improvement. So they again move to the depressed class at a rate q . Also, they decrease w.r.t the mortality rate ξ .

$$\frac{dR}{dt} = (1 - \nu) \gamma E + \phi_1 r_1 I_1 + \phi_2 I_2 - q R - (\xi + \theta) R,$$

Permanent Quieter Individuals class $Q(t)$:

$Q(t)$ refers to all individuals who stop or avoid using social media. Some individuals

in the susceptible class took prevention measures and started to quit or avoid using social media and move to the permanently quieter individual population at a rate β . Also, the recovered individuals who start avoiding social apps consistently move to $Q(t)$ at a rate θ . But still, some individuals are now in the domain of becoming susceptible, and transmitting the infection moves to the susceptible class at a rate $\pi\theta$. They decrease with respect to the mortality rate ξ .

$$\frac{dQ}{dt} = \beta S + \theta(1 - \pi) R - \xi Q.$$

Suicide class $D(t)$:

$D(t)$ shows the individual who commits suicide during the infection. The depressed individual whose mental health is not controllable. They are always at risk of attempting or committing suicide and move to a suicide class at a rate μ . They decrease with respect to the mortality rate ξ .

$$\frac{dD}{dt} = \mu I_2 - \xi D.$$

Here, we write the non-dimensional form of the proposed model with bilinear incidence rate and treatment rate

$$\begin{cases} \frac{dS}{dt} = \Gamma + \pi\theta R - \lambda_1 \frac{I_1 S}{N} - \lambda_2 \frac{I_2 S}{N} - (\beta + \xi) S, \\ \frac{dE}{dt} = \lambda_1 \frac{I_1 S}{N} + \lambda_2 \frac{I_2 S}{N} - (\gamma + \xi) E, \\ \frac{dI_1}{dt} = \nu\gamma E - \phi_1 I_1 - (\xi + \alpha) I_1, \\ \frac{dI_2}{dt} = \alpha I_1 + \phi_1(1 - r_1) I_1 + qR - \phi_2 I_2 - (\mu + \xi) I_2, \\ \frac{dR}{dt} = (1 - \nu)\gamma E + \phi_1 r_1 I_1 + \phi_2 I_2 - qR - (\xi + \theta) R, \\ \frac{dQ}{dt} = \beta S + (1 - \pi)\theta R - \xi Q, \\ \frac{dD}{dt} = \mu I_2 - \xi D. \end{cases} \quad (5.1)$$

The initial conditions at $t = 0$ are given as:

$$S_0, E_0, I_{1,0}, I_{2,0}, R_0, Q_0, D_0 \geq 0.$$

Based on the following assumptions, the coupled addiction-depression dynamics are proposed: This epidemic occurs in a closed environment, with the total population denoted by N , written as

$$N(t) = S(t) + E(t) + I_1(t) + I_2(t) + R(t) + Q(t) + D(t).$$

Sexuality, race, or financial status have no impact on the chances of acquiring addiction-depression states. Each individual engages in interactions with others in a similar way. People who are not already social media addicts might develop an addiction if they are affected by the influence of those who are social media addicts. Media exposure significantly influences the transmission of depression in individuals with

media addiction, with this effect being moderated or mediated by individual differences such as socialization and body dissatisfaction.

Before moving towards mathematical analysis, the system (5.1) deserves some remarks that distinguish it from existing models present in the literature. The proposed model follows the pathway of a standard susceptible-exposed-infected-recovered (SEIR) compartment model with extensive sub-populations that define the disease dynamics in the population. The recent COVID-19 pandemic defines the role of media awareness in shaping individuals' beliefs towards social distancing and vaccination as a prevention measure to decrease the spread of the infection. Due to the lockdown whole world complete shift to remote working strategies, leading people towards social seclusion and mental anguish. Social media platforms emerge as a significant source of fast information, social connectivity, and emotional expressions. Excessive reliance on these platforms increases the behavioral changing tendencies towards addiction, depression, anxiety, etc. This also increases the false information, cyberbullying, and online scams. Many studies formally investigate social media addiction [15, 161, 14, 12, 96, 176] with a modeling perspective. The proposed model (5.1) defines the behavioral dynamics, follows the idea of [15, 14] precisely, and includes the bidirectional transmission of addiction and depression with pre-pressure of media that effectively evaluates the suicide rate. To make the model more realistic, we adopt the physical therapies for addicts and initial treatment to seriously depressed individuals with relapse mechanics because most people become vulnerable after recovery, and many cannot afford the expensive treatment. The model reliability is defined through the use of actual data, which is briefly discussed in Sec 5.4 by constructing data data-driven model. The model formulation clearly shows that the pervasive and predictable impact of social media activities leads to the persistent challenges of addiction and depression, independent of the validity of recovery or transition measures. This motivates us to define an optimal control problem for the system (5.1) to optimize the behavioral burden by controlling the treatment cost and increasing awareness through media management, etc.

5.3 Mathematical analysis

In this section, the focus is on demonstrating the positivity of solutions within a bounded region. The analysis includes an examination of the dynamics of the reproduction number, denoted as $\mathbb{R}_0$, and the strength number, $\mathbb{A}_0$, which are assessed in conjunction with the equilibrium states of the model. Furthermore, the discussion extends to cover both local and global stability concerning $\mathbb{R}_0$, emphasizing its importance in understanding the underlying dynamics of the system.

To demonstrate the positivity and boundedness of solutions for the model referenced in equation (5.1), it is crucial to note that every related rate in the proposed model defined in equation (5.1) is non-negative. This characteristic implies that the direction of the vector field remains inward on all bounding planes. Therefore, if the initial condition is set anywhere within the non-negative bounds of $\mathbb{R}^7$, the trajectory of the system will consistently remain within this defined cone. Consequently, it can be confidently asserted that all solutions to the system represented by equation (5.1) are non-negative. Furthermore, the derived differential equation stems from analyzing the rate of change of the total population $N(t)$, defined as $\frac{dN(t)}{dt} = \Gamma - \xi N$, further simplification leads to $\limsup_{t \rightarrow \infty} N(t) \leq \frac{\Gamma}{\xi}$. Consequently, the solutions of system (5.1) are bounded by $\frac{\Gamma}{\xi}$. Thus, the following positively-invariant region is the

biologically viable region of model (5.1).

$$\Psi = \left\{ (S, E, I_1, I_2, R, Q, D) \in \mathbb{R}_+^7 \mid 0 \leq N \leq \frac{\Gamma}{\xi} \right\}.$$

Theorem 5.1. *For given initial conditions, all solutions of model (5.1) are positive for all $t \geq 0$.*

Proof. Assuming $(S(t), E(t), I_1(t), I_2(t), R(t), Q(t), D(t))$ be any positive solution of model system (5.1) with given initial conditions in Sec 5.2, we define

$$\mathfrak{B}(t) = \min\{S, E, I_1, I_2, R, Q, D\},$$

clearly shows that $\mathfrak{B}(t) > 0$ at $t = 0$. To prove positivity of S for any $t \geq 0$, suppose $\exists t_1 > 0$ s.t. $\mathfrak{B}(t_1) = 0$ and $\mathfrak{B}(t) > 0 \forall t \in (0, t_1]$. Taking $\mathfrak{B}(t_1) = S(t_1) = 0, \forall t \in (0, t_1]$, we have

$$E, I_1, I_2, R, Q, D \geq 0, \quad \forall t \geq 0.$$

Using first compartment of model (5.1), we write

$$\dot{S}(t) = \Gamma + \pi \theta R(t) - \lambda_1 \frac{I_1(t)S(t)}{N(t)} - \lambda_2 \frac{I_2(t)S(t)}{N(t)} - (\beta + \xi) S(t).$$

As, $I_1(t), I_2(t), R(t) \geq 0$ with $\beta, \xi > 0$, we have

$$\dot{S}(t) \geq \Gamma - \omega(t)S(t), \quad t \in (0, t_1]$$

where $\omega(t) = \left(\lambda_1 \frac{I_1(t)}{N(t)} + \lambda_2 \frac{I_2(t)}{N(t)} + (\beta + \xi) \right)$. Applying the integration factor and rearranging the expression, we get

$$S(t_1) \geq S(0)e^{-\int_0^{t_1} \omega(\theta) d\theta} + e^{-\int_0^{t_1} \omega(\theta) d\theta} \times \Gamma \int_0^{t_1} e^{\int_0^s \omega(\theta) d\theta} ds.$$

Since $S(0) > 0$ and both exponents are positive, we conclude

$$S(t_1) > 0$$

which contradicts our assumption. Hence susceptible population is not zero, i.e., $S(t) > 0$ for all $t \in 0$. Similarly, one can prove for renaming compartments. $\square$

Above, this approach ensures that the conclusions drawn about solutions maintain their positivity and bounded nature throughout the analysis. This mathematical property shows that the model is biologically and behaviorally well-posed and exhibits significant dynamics over time. The steady-state evaluation appears essential to appreciating the model's long-term behavior. This investigation clarifies the particular circumstances influencing the persistence or die out of behavioral diseases like depression and social media addiction in that population. By considering these conditions, we can make a possible assumption about the evaluation of these variables and potential strategies to reduce their negative consequences. In the context of model (5.1), consider the system in compact form.

$$\frac{d\mathcal{U}(t)}{dt} = \mathcal{F}(\mathcal{U}(t); \theta),$$

where $\mathcal{U}(t) \in \mathbb{R}^{+7}$ presents the state vectors and $\theta \in \mathbb{R}^n$ is the all parameters in model. To evaluate the steady states, we consider

$$\mathcal{F}(\mathcal{U}(t); \theta) = 0,$$

leads to the system of equations simulating them simultaneously with precise algebraic expression, we get two steady states of the model that are reached in the feasible region Ψ . The disease-free steady state (DFE) of the model is given as

$$E^{q_0} = (S^0, E^0, I_1^0, I_2^0, R^0, Q^0, D^0).$$

All the state vectors are zero except $S^0 = \frac{\Gamma}{(\beta + \xi)}$ and $Q^0 = \frac{\beta \Gamma}{(\beta + \xi) \xi}$, which means there is no infection defined in the healthy phase in behavioral dynamics. This demonstrates that all other groups gradually vanish, leaving just susceptible and long-term quitters. Furthermore, the endemic steady state (EEP) indicates the persistence as well as coexistence of addiction and depression dynamics over time. The formulation of EEP is written as

$$E^{q_1} = (S^1, E^1, I_1^1, I_2^1, R^1, Q^1).$$

where

$$\begin{aligned} S^1 &= \frac{(\xi^2 + (\theta + \phi_2 + q + \mu) \xi + (\phi_2 + \mu) \theta + \mu q) (\alpha + \xi + \phi_1) (\gamma + \xi)}{(\nu \xi^2 \lambda_1 + (((-q + (1 - r_1) \phi_1 + \alpha) \lambda_2 + \lambda_1 (\theta + \phi_2 + q + \mu)) \nu + \lambda_2 q) \xi + l_1) \gamma}, \\ E^1 &= \frac{I_1^1 (\alpha + \xi + \phi_1)}{\nu \gamma}, \\ I_1^1 &= \frac{l_{10} \nu (-\xi^5 - l_9 \xi^4 + l_5 \xi^3 + l_6 \xi^2 + l_7 \xi + l_8 \gamma)}{(\xi^4 + l_9 \xi^3 + l_2 \xi^2 + l_3 \xi + l_{11} \gamma) l_4}, \\ I_2^1 &= \frac{(-\nu \theta \phi_1 r_1 - \nu \xi \phi_1 r_1 + \alpha \nu \theta + \alpha \nu \xi - \nu q \xi + \nu \theta \phi_1 + \nu \xi \phi_1 + \alpha q + q \xi + q \phi_1) I_1^1}{\nu (\mu q + \mu \theta + \mu \xi + q \xi + \theta \xi + \theta \phi_2 + \xi^2 + \xi \phi_2)}, \\ R^1 &= \frac{(((r_1 - 1) \phi_1 - \xi - \alpha) \nu + \xi + \alpha + \phi_1) I_1^1 + \nu I_2^1 \phi_2}{\nu (q + \xi + \theta)}, \\ Q^1 &= \frac{(l_{12} \nu - I_1^1 (\pi - 1) (\alpha + \xi + \phi_1)) \theta + S^1 \nu \beta (q + \xi)}{\nu (q + \xi + \theta) \xi}, \\ D^1 &= \frac{\mu I_1^1 (-\nu \theta \phi_1 r_1 - \nu \xi \phi_1 r_1 + \alpha \nu \theta + \alpha \nu \xi - \nu q \xi + \nu \theta \phi_1 + \nu \xi \phi_1 + \alpha q + q \xi + q \phi_1)}{\nu (\mu q + \mu \theta + \mu \xi + q \xi + \theta \xi + \theta \phi_2 + \xi^2 + \xi \phi_2) \xi}. \end{aligned} \quad (5.2)$$

Thus, values of $l_i, i=1, 2, \dots, 12$ are given in Appendix B. The values of all state vectors in EEP are nonzero, which shows that positive solutions of the nonlinear equilibrium state equations of exposed, addicted, and depressed individuals exist with the constraint that the reproduction number is greater than unity.

The mathematical model's dynamics can be defined by the threshold parameter, the so-called reproduction number ($\mathbb{R}_0$). A disease spreads when an infected person comes into contact with individuals who are all susceptible to the disease. Consequently, $\mathbb{R}_0$ represents the total number of new infections developed by an infected individual at time $t \geq 0$. It quietly predicts the dynamic behavior of a model, proposing that it produces better comprehension of possible future spreading or control of the disease. According to the epidemiological $\mathbb{R}_0$ criteria, if $\mathbb{R}_0 < 1$, then the one infected person in the susceptible population infects less than one person. Therefore, it is feasible to eliminate the disease from the population, and the

disease-free steady state is asymptotically stable. That is, there would not be an epidemic spreading. If $\mathbb{R}_0 > 1$, then more than one person in the entire susceptible population is infected by a single infected individual. Therefore, the disease-free steady state is unstable, and the disease may continue to exist in the population. If $\mathbb{R}_0 = 1$, then one infected individual in the entire susceptible population infects one new person. Thus, in the absence of an epidemic, the disease is going to persist in the total population [21]. To calculate the $\mathbb{R}_0$, we apply the next-generation approach provided by [102].

Taking model (5.1) where the entire population is divided into seven classes based on the Behavioral state of individuals. We rearrange system (5.1) so that the infection compartments occupy the first n positions. Let $\mathcal{F}_i(y)$ denote the rate of new infection in infected group, $\mathcal{V}_i^+(y)$ represent the rate of transfer of individuals into the infected group by means other than new infections, and $\mathcal{V}_i^-(y)$ denote the rate of individuals removed from infected class.

$$y'_i = \mathcal{F}_i(y) - \mathcal{V}_i(y), \quad \text{where} \quad \mathcal{V}_i(y) = \mathcal{V}_i^-(y) - \mathcal{V}_i^+(y)$$

In the presence of the infected free equilibrium y_0 , the $n \times n$ matrices $\mathbb{F}$ and $\mathbb{V}$ are formed

$$\mathcal{F} = \left. \frac{\partial \mathcal{F}_i}{\partial y_j} \right|_{y_0}, \quad \mathcal{V} = \left. \frac{\partial \mathcal{V}_i}{\partial y_j} \right|_{y_0}, \quad \text{where} \quad 1 \leq i, j \leq n.$$

Following the next-generation matrix approach, the $\mathcal{F}\mathcal{V}^{-1}$ is defined [106]. Thus, $\mathcal{R}_0$ denote the largest eigenvalue. Applying this strategy, by rearranging (5.1) and writing the infected compartments as

$$\begin{aligned} \frac{dE}{dt} &= \lambda_1 \frac{I_1 S}{N} + \lambda_2 \frac{I_2 S}{N} - (\gamma + \xi) E \\ \frac{dI_1}{dt} &= \nu \gamma E - \phi_1 I_1 - (\xi + \alpha) I_1, \\ \frac{dI_2}{dt} &= \alpha I_1 + \phi_1 (1 - r_1) I_1 + q R - \phi_2 I_2 - (\mu + \xi) I_2, \\ \frac{dR}{dt} &= (1 - \nu) \gamma E + \phi_1 r_1 I_1 + \phi_2 I_2 - q R - (\xi + \theta) R, \end{aligned} \tag{5.3}$$

leads to the $\mathbf{F}$ and $\mathbf{V}$ vectors, given as

$$\mathbf{F} = \begin{pmatrix} \lambda_1 \frac{I_1 S}{N} + \lambda_2 \frac{I_2 S}{N} \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

$$\mathbf{V} = \begin{pmatrix} (\gamma + \xi) E \\ -\nu \gamma E + \phi_1 r_1 I_1 + (\xi + \alpha) I_1 \\ -\alpha I_1 - \phi_1 (1 - r_1) I_1 - q R + \phi_2 I_2 + (\mu + \xi) I_2 \\ -(1 - \nu) \gamma E - \phi_1 r_1 I_1 - \phi_2 I_2 + q R + (\xi + \theta) R \end{pmatrix}$$

Then

$$\begin{aligned} \frac{\partial \mathcal{F}_1}{\partial E} &= -S \frac{\lambda_1 I_1 + \lambda_2 I_2}{N^2}, & \frac{\partial \mathcal{F}_1}{\partial I_1} &= S \left(\frac{\lambda_1}{N} - \frac{\lambda_1 I_1 + \lambda_2 I_2}{N^2} \right), \\ \frac{\partial \mathcal{F}_1}{\partial I_2} &= S \left(\frac{\lambda_2}{N} - \frac{\lambda_1 I_1 + \lambda_2 I_2}{N^2} \right), & \frac{\partial \mathcal{F}_1}{\partial R} &= -S \frac{\lambda_1 I_1 + \lambda_2 I_2}{N^2}. \end{aligned}$$

Putting the above expressions of derivatives, we write the matrices using the NGM approaches

$$\mathcal{F} = \begin{pmatrix} 0 & \frac{\lambda_1 S^0}{S^0 + Q^0} & \frac{\lambda_2 S^0}{S^0 + Q^0} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

$$\mathcal{V} = \begin{pmatrix} \gamma + \xi & 0 & 0 & 0 \\ -\gamma \nu & \phi_1 r_1 + (\xi + \alpha) & 0 & 0 \\ 0 & -\alpha - \phi_1 (1 - r_1) & \phi_2 + (\mu + \xi) & -q \\ -(1 - \nu) \gamma & -\phi_1 r_1 & -\phi_2 & q + (\xi + \theta) \end{pmatrix}$$

Hence, the expression for $\mathbb{R}_0$ is given as:

$$\ell_1 = \phi_1 r_1 + (\xi + \alpha), \quad \ell_2 = -\alpha - \phi_1 (1 - r_1), \quad \ell_3 = \phi_2 + (\mu + \xi),$$

$$S^0 = \frac{\Gamma}{\beta + \xi}, \quad Q^0 = \frac{\beta \Gamma}{(\beta + \xi) \xi}.$$

$$\mathbb{R}_0 = \frac{S_0 \gamma \lambda_1 \nu}{\ell_1 (Q_0 + S_0) (\gamma + \xi)} - \frac{S_0 \lambda_2 \gamma (\ell_1 q (\nu - 1) + \ell_2 \nu (q + \theta + \xi) - \nu \phi_1 q r_1)}{(Q_0 + S_0) (\gamma + \xi) (\ell_1 \ell_3 (q + \theta + \xi) - \ell_1 \phi_2 q)} \quad (5.4)$$

The basic reproduction number is a vital measure in epidemiology to evaluate the initial transmission capacity of the pandemic, but it fails to deliver insight into the temporal structure of an epidemic's evolution. Particularly, it does not reflect whether disease incidence exhibits oscillatory tendencies or grows monotonically. The strength number $\mathbb{A}_0$ addresses these limitations by providing secondary information about the shape or rapidity of the pathogen's trajectory. The biological worth of $\mathbb{A}_0$ is found in its control over direction curvature near the disease-free steady state; it dictates whether solutions converge monotonically or produce damped oscillations as experienced in the COVID-19 outbreak. This affects the predictable nature of epidemic spikes and the optimum period for preventative measures.

The strength number has been widely used in epidemiological modeling during the last few decades, appearing in multiple research publications. It is considered a powerful mathematical tool for optimizing reproduction in certain diseases. The principle is utilized as well to assess how far the disease has expanded. Several scholars have pointed out several shortcomings in modeling, such as the fact that it is not distinctive and may be created through a variety of methods. The fact that this number is just a function of time and not a constant value popped up as a further concern. The so-called strength number is an alternate value that is currently proposed in research [29, 30].

$$\frac{\partial^2 \mathcal{F}_1}{\partial E^2} = \frac{2S(\lambda_1 I_1 + \lambda_2 I_2)}{N^3}, \quad \frac{\partial^2 \mathcal{F}_1}{\partial I_1^2} = -\frac{2S\lambda_1}{N^2} + \frac{2S(\lambda_1 I_1 + \lambda_2 I_2)}{N^3},$$

$$\frac{\partial^2 \mathcal{F}_1}{\partial I_2^2} = -\frac{2S\lambda_2}{N^2} + \frac{2S(\lambda_1 I_1 + \lambda_2 I_2)}{N^3}, \quad \frac{\partial^2 \mathcal{F}_1}{\partial R^2} = \frac{2S(\lambda_1 I_1 + \lambda_2 I_2)}{N^3}.$$

The Jacobian $\mathcal{F}$ and $\mathcal{V}$ at the DFE are given as:

$$\mathcal{F} = \begin{pmatrix} 0 & \frac{-2\lambda_1 S^0}{(S^0 + Q^0)^2} & \frac{-2\lambda_2 S^0}{(S^0 + Q^0)^2} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}.$$

$$\mathcal{V} = \begin{pmatrix} \gamma + \xi & 0 & 0 & 0 \\ -\gamma \nu & \phi_1(1 - r_1) + \phi_1 r_1 + (\xi + \alpha) & 0 & 0 \\ 0 & -\alpha - \phi_1(1 - r_1) & -\phi_2 q + \phi_2 r_2 + (\mu + \xi) & 0 \\ -(1 - \nu)\gamma & -\phi_1 r_1 & -\phi_2 r_2 + \phi_2 q & \xi + \theta \end{pmatrix}$$

$$\mathbb{A}_0 = -\frac{\Delta_1 \lambda_2 \gamma \nu (\ell_1 q / \nu + \alpha q + \alpha \theta + \alpha \xi - \ell_1 q + \phi_1 q + \phi_1 \theta + \phi_1 \xi - \phi_1 r_1 \theta - \phi_1 r_1 \xi)}{\ell_1 \ell_3 (q + \theta \xi) - \ell_1 \phi_2 q} + \Delta_2. \quad (5.5)$$

where $\Delta_1 = \frac{2S_0}{(Q_0 + S_0)^2(\gamma + \xi)}$ and $\Delta_2 = \frac{\gamma \lambda_1 \nu}{\ell_1}$. The investigated model has no sign of a second wave and slowdown due to the fact that $\mathbb{A}_0 < 0$. A single magnitude when $\mathbb{A}_0 = 0$, indicating that the spread will not have a renewal process. If $\mathbb{A}_0 > 0$, this indicates that strength exists and will start a renewal process, which will produce several waves in the disease spread. In the renewal process, the infection will rise after a minimum point and either move to stabilization or end after a while. Either the maximum and two infection points exhibit a single wave or quickly break down an infection from the DFE.

Stability analysis plays a critical role in describing the long-term evaluation of disease outbreak in the population and gives information about the persistence or die out of the infection and ensures the model outcomes are biologically reasonable, i.e, lie in the feasible region Ψ . A steady state $\mathcal{U}^*$ of the system (5.1) is

$$\frac{d\mathcal{U}(t)}{dt} = \mathcal{F}(\mathcal{U}),$$

stable if the solution started in neighborhood $\mathcal{B}_\eta(\mathcal{U}^*)$ of $\mathcal{U}^*$, it remain within $\mathcal{B}_\eta(\mathcal{U}^*)$ for at $t > 0$ as $\mathcal{U}^* \rightarrow \infty$. Most frequent analysis of model stability lies in the local and global sense. Mathematically, we define the local stability of the steady states as, for any $\epsilon > 0$ there exists a constant δ such that if $\|\mathcal{U}(0) - \mathcal{U}^*\| < \delta$ then

$$\|\mathcal{U}(t) - \mathcal{U}^*\| < \epsilon \quad \forall t \geq 0,$$

the steady state is stable. For asymptotic local stability, it follows the formulation.

$$\lim_{t \rightarrow \infty} \mathcal{U}(t) = \mathcal{U}^*.$$

System local stability is examined by using the linearization operation at a DFE, which entails studying the Jacobian matrix's eigenvalues. Local asymptotic stability is established when all eigenvalues are negative. The steady state is globally stable if for any admissible initial conditions, all trajectory of solutions in the feasible region converges to it. Evaluation of the global stability of the steady state is particularly complex. We use LaSalle's invariance principle for DFE and Lyapunov functions

formulation for EEP to define their global stability. Recalling that the reproduction number $\mathbb{R}_0$ is a threshold condition to define the stability analysis of steady states in compartment models. Using this concept, we write the following statement with the assumption that the total population is constant for simplicity.

Theorem 5.2. *The disease-free equilibrium of the model (5.1) is locally asymptotically stable if $\mathbb{R}_0 < 1$.*

Proof. The evaluation of the Jacobian of the system (5.1) at the DFE is given as

$$J = \begin{pmatrix} -l_1 & 0 & -\lambda_1 S^0 & -\lambda_2 S^0 & \pi\theta & 0 & 0 \\ 0 & l_4 & \lambda_1 S^0 & \lambda_2 S^0 & 0 & 0 & 0 \\ 0 & \nu\gamma & l_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & l_5 & l_3 & q & 0 & 0 \\ 0 & (1-\nu)\gamma & \phi_1 r_1 & \phi_2 & l_6 & 0 & 0 \\ \beta & 0 & 0 & 0 & (1-\pi)\theta & -(\xi + \tau) & 0 \\ 0 & 0 & 0 & \mu & 0 & 0 & -(\xi + \tau) \end{pmatrix}$$

where

$$\begin{aligned} l_1 &= (\beta + \xi + \tau), & l_2 &= -\phi_1 - (\xi + \alpha + \tau), & l_3 &= -\phi_2 - (\mu + \xi + \tau), \\ l_4 &= -(\gamma + \xi + \tau), & l_5 &= \alpha + \phi_1(1 - r_1), & l_6 &= -(q + \xi + \theta + \tau). \end{aligned}$$

After solving the determinant of this Jacobian matrix J , we found the polynomial given as

$$-(\xi + \tau)^2(-\beta - \xi - \tau)(\tau^4 + a_3\tau^3 + a_2\tau^2 + a_1\tau + a_0) = 0.$$

leads to the eigenvalues written as $\tau_1 = -\xi$ and $\tau_2 = -\beta - \xi$. where the remaining eigenvalues will be obtained by solving the fourth-order polynomial defined as

$$\tau^4 + a_3\tau^3 + a_2\tau^2 + a_1\tau + a_0 = 0 \quad (5.6)$$

The coefficients of (5.6) are given in Appendix B. we found according to the Routh-Hurwitz stability criterion, if $a_1, a_2, a_3, a_4 > 0, a_1a_2 > a_3, a_1a_2a_3 > a_1^2a_4 + a_3^2$. Since all the parameters are non-negative. Thus, the DFE is locally asymptotically stable. $\square$

Theorem 5.3. *The endemic equilibrium of model (5.1) is locally asymptotically stable if $\mathbb{R}_0 > 1$.*

Proof. The Jacobian for system (5.1) calculated at the EEP can be obtained as

$$J^* = \begin{pmatrix} -l_1 & 0 & -\lambda_1 S^1 & -\lambda_2 S^1 & \pi\theta & 0 & 0 \\ l_4 & l_5 & \lambda_1 S^1 & \lambda_2 S^1 & 0 & 0 & 0 \\ 0 & \nu\gamma & l_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & l_6 & l_3 & q & 0 & 0 \\ 0 & (1-\nu)\gamma & \phi_1 r_1 & \phi_2 & -(q + \xi + \theta + \tau) & 0 & 0 \\ \beta & 0 & 0 & 0 & (1-\pi)\theta & -(\xi + \tau) & 0 \\ 0 & 0 & 0 & \mu & 0 & 0 & -(\xi + \tau) \end{pmatrix}$$

where

$$l_1 = (\lambda_1 I_1^1 + \lambda_2 I_2^1 + \beta + \xi + \tau), \quad l_2 = -\phi_1 - (\xi + \alpha + \tau), \quad l_3 = -\phi_2 - (\mu + \xi + \tau)$$

$$l_4 = \lambda_1 I_1^1 + \lambda_2 I_2^1, \quad l_5 = -(\gamma + \xi + \tau), \quad l_6 = \alpha + \phi_1 (1 - r_1).$$

After solving this Jacobian matrix, we found the polynomial given as

$$(-\xi - \tau)(-\phi_2 - \mu - \xi - \tau)(-q - \xi - \theta - \tau)P(\tau) = 0$$

leads to eigenvalues $\tau_1 = -\xi$, $\tau_2 = -(\phi_2 + \mu + \xi)$ and $\tau_3 = -(q + \xi + \theta)$. The renaming eigenvalues calculated through the cubic polynomial $P(\tau)$ are defined as

$$\tau^3 + b_2\tau^2 + b_1\tau + b_0 = 0. \quad (5.7)$$

where the coefficients of (5.7) given in Appendix B. The discriminant of the polynomial $P(\tau)$ is described by

$$D(P) = 18u_1u_2u_3 + (u_1u_2)^2 - 4u_3u_1^2 - 4u_2^2 - 27u_3^2 \quad (5.8)$$

and using [33], we have following corollary:

Corollary 5.4. For $R_0 > 1$ the EEP of model (5.1) is LAS if one of the these conditions satisfy for $P(\tau)$

1. If $D(P) > 0$, then $u_1 > 0$, $u_3 > 0$, and $u_1u_2 > u_3$ provide a necessary and sufficient condition for local stability of the EEP.
2. If $D(P) < 0$, $u_1 \geq 0$, $u_2 \geq 0$, $u_3 > 0$, $\theta < \frac{2}{3}$ then EEP is LAS.
3. If $D(P) < 0$, $u_1 < 0$, $u_2 < 0$, and $\theta > \frac{2}{3}$, then all solutions of the (5.8) fulfill the criteria $|\arg(\lambda_j)| < \theta \frac{\pi}{2}$, $j = 1, 2, 3, 4$.

□

To define the global stability analysis of the steady states of system (5.1), we follow the following theorems.

Theorem 5.5. If model (5.1) can be re-written in the form:

$$\frac{d\mathfrak{X}}{dt} = \mathcal{F}(\mathfrak{X}, \mathcal{Y})$$

$$\frac{d\mathcal{Y}}{dt} = \mathcal{G}(\mathfrak{X}, \mathcal{Y}), \mathcal{G}(\mathfrak{X}, 0) = 0$$

where $\mathfrak{X}$ belongs to the positive real numbers and $\mathcal{Y}$ lies in $\mathbb{R}_+^4$. $\mathfrak{X} = (S)^T$ and $\mathcal{Y} = (E, I_1, I_2, R)^T$ represent uninfected and infected groups; respectively.

$$Eq_0 = (\mathfrak{X}_0, 0) = \left(\frac{\Gamma}{\beta + \xi}, 0\right)$$

For the DFE of the model to be globally asymptotically stable around the DFE, the following conditions H_1 and H_2 must be preserved

$$\mathbf{H}_1 : \quad \frac{d\mathfrak{X}}{dt} = \mathcal{F}(\mathfrak{X}_0, 0), \quad \mathfrak{X}_0 \text{ is globally asymptotically stable.}$$

$$\mathbf{H}_2 : \quad \mathcal{L}(\mathfrak{X}, \mathcal{Y}) = A\mathcal{Y} - \hat{\mathcal{L}}(\mathfrak{X}, \mathcal{Y}), \quad \hat{\mathcal{L}}(\mathfrak{X}, \mathcal{Y}) \geq 0.$$

Note that $A = D_y L(\mathfrak{X}_0, 0)$, calculated at $(\mathfrak{X}_0, 0)$, is an M -matrix with nonnegative off diagonal elements. If the system meets $\mathbf{H}_1$ and $\mathbf{H}_2$, the equilibrium E_{q_0} is globally asymptotically stable for $\mathcal{R}_0 < 1$.

Theorem 5.6. *The system (5.1) is globally stable if $\mathcal{R}_0 < 1$ and conditions $\mathbf{H}_1$ and $\mathbf{H}_2$ hold.*

Proof. Below, $\mathcal{F}$ and L are derived from (5.1):

$$\mathcal{F} = \Gamma + \pi \theta R - \lambda_1 I_1 S - \lambda_2 I_2 S - (\beta + \xi) S \quad (5.9)$$

and

$$L(\mathfrak{X}, \mathcal{Y}) = \begin{pmatrix} \lambda_1 I_1 S + \lambda_2 I_2 S - (\gamma + \xi) E \\ \nu \gamma E - \phi_1 I_1 - (\xi + \alpha) I_1 \\ \alpha I_1 + \phi_1 (1 - r_1) I_1 + qR - \phi_2 I_2 - (\mu + \xi) I_2 \\ (1 - \nu) \gamma E + \phi_1 r_1 I_1 + \phi_2 I_2 - qR - (\xi + \theta) R \end{pmatrix} \quad (5.10)$$

Solving Eq. (5.20) at $\mathcal{Y} = 0$ yields

$$\mathcal{F}(\mathfrak{X}_0, 0) = \Gamma - \xi S$$

and further simplification yields

$$S(t) = \frac{\Gamma}{\xi}$$

For any initial value $S(0)$, as $t \rightarrow \infty$ we have $S(t) \rightarrow \frac{\Gamma}{\xi} - S(0)e^{-\mu t}$. Hence, the condition $\mathbf{H}_1$ is satisfied. Further, the infected compartments of the m given by

$$L(\mathfrak{X}, \mathcal{Y}) = A\mathcal{Y} - \hat{L}(\mathfrak{X}, \mathcal{Y})$$

where

$$A = D_y L(\mathfrak{X}_0, 0) = \begin{pmatrix} -(\gamma + \xi) & \lambda_1 S & \lambda_2 S & 0 \\ \nu \gamma & -\phi_1 - (\xi + \alpha) & 0 & 0 \\ 0 & \phi_1 (1 - r_1) & \phi_2 - (\mu + \xi) & q \\ (1 - \nu) \gamma E & \phi_1 r_1 & \phi_2 & -(\xi + \theta) \end{pmatrix}$$

which simplifies to:

$$L(\mathfrak{X}, \mathcal{Y}) = A\mathcal{Y} - \hat{L}(\mathfrak{X}, \mathcal{Y}) = A\mathcal{Y} - \begin{pmatrix} \hat{L}_1(\mathfrak{X}, \mathcal{Y}) \\ \hat{L}_2(\mathfrak{X}, \mathcal{Y}) \\ \hat{L}_3(\mathfrak{X}, \mathcal{Y}) \\ \hat{L}_4(\mathfrak{X}, \mathcal{Y}) \end{pmatrix} = \begin{pmatrix} (\lambda_1 I_1 + \lambda_2 I_2)(S_0 - S) \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

Since $S \leq S_0$, it follows that $L \geq 0$. Hence, the disease-free point of system (5.1) is globally stable. $\square$

Theorem 5.7. *If $\mathbb{R}_0 > 1$, then the endemic equilibrium point of the proposed model (5.1) is globally asymptotically stable.*

Proof. Let $\mathcal{L}(t)$ be a Lyapunov function defined by

$$\begin{aligned}\mathcal{L}(t) = & \left(S - S^* - S^* \ln \frac{S}{S^*} \right) + \left(E - E^* - E^* \ln \frac{E}{E^*} \right) + \left(I_1 - I_1^* - I_1^* \ln \frac{I_1}{I_1^*} \right) \\ & + \left(I_2 - I_2^* - I_2^* \ln \frac{I_2}{I_2^*} \right) + \left(R - R^* - R^* \ln \frac{R}{R^*} \right) + \left(Q - Q^* - Q^* \ln \frac{Q}{Q^*} \right) \\ & + \left(D - D^* - D^* \ln \frac{D}{D^*} \right).\end{aligned}$$

The derivative of $\mathcal{L}(t)$ along the system's trajectory is given as

$$\begin{aligned}\frac{d\mathcal{L}(t)}{dt} = & \left(1 - \frac{S^*}{S} \right) [\Gamma + \pi \theta R - \lambda_1 I_1 S - \lambda_2 I_2 S - (\beta + \xi) S] \\ & + \left(1 - \frac{E^*}{E} \right) [\lambda_1 I_1 S + \lambda_2 I_2 S - (\gamma + \xi) E] + \left(1 - \frac{I_1^*}{I_1} \right) [\nu \gamma E - \phi_1 I_1 - (\xi + \alpha) I_1] \\ & + \left(1 - \frac{I_2^*}{I_2} \right) [\alpha I_1 + \phi_1 (1 - r_1) I_1 + q R - \phi_2 I_2 - (\mu + \xi) I_2] \\ & + \left(1 - \frac{R^*}{R} \right) [(1 - \nu) \gamma E + \phi_1 r_1 I_1 + \phi_2 I_2 - q R - (\xi + \theta) R] \\ & + \left(1 - \frac{Q^*}{Q} \right) [\beta S + (1 - \pi) \theta R - \xi Q] + \left(1 - \frac{D^*}{D} \right) [\mu I_2 - \xi D].\end{aligned}$$

Using the endemic conditions,

$$\begin{aligned}\Gamma = & -\pi \theta R^* + \lambda_1 I_1^* S^* + \lambda_2 I_2^* S^* + (\beta + \xi) S^*, \\ \lambda_1 I_1^* S^* + \lambda_2 I_2^* S^* = & (\gamma + \xi) E^*, \\ \nu \gamma E^* = & [\phi_1 + \xi + \alpha] I_1^*, \\ \alpha I_1^* + \phi_1 (1 - r_1) I_1^* + q R^* = & [\phi_2 + \mu + \xi] I_2^*, \\ (1 - \nu) \gamma E^* + \phi_1 r_1 I_1^* + \phi_2 I_2^* = & [q + \xi + \theta] R^*, \\ \beta S^* + (1 - \pi) \theta R^* = & \xi Q^*, \\ \mu I_2^* = & \xi D^*.\end{aligned}$$

we get

$$\frac{d\mathcal{L}(t)}{dt} = T_1 + T_2 + T_3 + T_4 + T_5 + T_6 + T_7,$$

where T_i values are defined below

$$\begin{aligned}
T_1 &= \pi \theta (R - R^*) \left(1 - \frac{S^*}{S}\right) + \lambda_1 (S^* I_1 - S I_1) + \lambda_2 (S^* I_2 - S I_2) - (\beta + \xi) (S - S^*), \\
T_2 &= (\lambda_1 I_1 S + \lambda_2 I_2 S) \left(1 - \frac{E^*}{E}\right) - (\gamma + \xi) (E - E^*), \\
T_3 &= \nu \gamma E \left(1 - \frac{I_1^*}{I_1}\right) - (\phi_1 + \xi + \alpha) (I_1 - I_1^*), \\
T_4 &= \left((\alpha + \phi_1 (1 - r_1)) (I_1 - I_1^*) + q (R - R^*) \right) \left(1 - \frac{I_2^*}{I_2}\right) - (\phi_2 + \mu + \xi) (I_2 - I_2^*), \\
T_5 &= \left((1 - \nu) \gamma E + \phi_1 r_1 I_1 + \phi_2 I_2 \right) \left(1 - \frac{R^*}{R}\right) - (q + \xi + \theta) (R - R^*), \\
T_6 &= \left(\beta S + (1 - \pi) \theta R \right) \left(1 - \frac{Q^*}{Q}\right) - \xi (Q - Q^*), \\
T_7 &= \mu I_2 \left(1 - \frac{D^*}{D}\right) - \xi (D - D^*).
\end{aligned}$$

Simplifying the algebraic expressions bounded by the arithmetic and geometric mean inequality, we obtain

$$\begin{aligned}
\frac{d\mathcal{L}}{dt} &= -(\beta + \xi) \frac{(S - S^*)^2}{S} - (\gamma + \xi) \frac{(E - E^*)^2}{E} - (\phi_1 + \xi + \alpha) \frac{(I_1 - I_1^*)^2}{I_1} \\
&\quad - (\phi_2 + \mu + \xi) \frac{(I_2 - I_2^*)^2}{I_2} - (q + \xi + \theta) \frac{(R - R^*)^2}{R} - \xi \frac{(Q - Q^*)^2}{Q} - \xi \frac{(D - D^*)^2}{D}.
\end{aligned} \tag{5.11}$$

Since all parameters are positive and $(\mathcal{X} - \mathcal{X}^*)^2 / \mathcal{X} > 0$ for all $\mathcal{X} \neq \mathcal{X}^*$ and $\mathcal{X} > 0$, we have:

$$\frac{d\mathcal{L}}{dt} \leq 0$$

with equality if and only if $S = S^*, E = E^*, I_1 = I_1^*, I_2 = I_2^*, R = R^*, Q = Q^*, D = D^*$. Hence, by LaSalle's invariance principle, the endemic equilibrium is globally asymptotically stable whenever $R_0 > 1$. $\square$

As the behavioral model (5.1) composes individual health concerns monotonically by coupled addiction-depression dynamics in the community. To justify the model integrity, we must have to show the existence and uniqueness of the model solution for given initial conditions. This mechanism ensures that the model is well posed and its forecasting is accurate. Now we describe the fixed point theorem of existence and uniqueness of the model solution. Using initial data by applying the

integral operator to each compartment of the model (5.1) yields

$$\begin{aligned}
S(t) - S(0) &= \int_0^t \left(\Gamma + \pi\theta R(\tau) - \lambda_1 \frac{I_1(\tau)S(\tau)}{N(\tau)} - \lambda_2 \frac{I_2(\tau)S(\tau)}{N(\tau)} - (\beta + \xi)S(\tau) \right) d\tau, \\
E(t) - E(0) &= \int_0^t \left(\lambda_1 \frac{I_1(\tau)S(\tau)}{N(\tau)} + \lambda_2 \frac{I_2(\tau)S(\tau)}{N(\tau)} - (\gamma + \xi)E(\tau) \right) d\tau, \\
I_1(t) - I_1(0) &= \int_0^t (\nu\gamma E(\tau) - (\phi_1 + \xi + \alpha)I_1(\tau)) d\tau, \\
I_2(t) - I_2(0) &= \int_0^t ((\alpha + \phi_1(1 - r_1))I_1(\tau) + qR(\tau) - (\phi_2 + \mu + \xi)I_2(\tau)) d\tau, \\
R(t) - R(0) &= \int_0^t ((1 - \nu)\gamma E(\tau) + \phi_1 r_1 I_1(\tau) + \phi_2 I_2(\tau) - (q + \xi + \theta)R(\tau)) d\tau, \\
Q(t) - Q(0) &= \int_0^t (\beta S(\tau) + (1 - \pi)\theta R(\tau) - \xi Q(\tau)) d\tau, \\
D(t) - D(0) &= \int_0^t (\mu I_2(\tau) - \xi D(\tau)) d\tau.
\end{aligned} \tag{5.12}$$

Therefore, we write the following kernels as

$$\begin{aligned}
\Pi_1(t, S) &= \Gamma + \pi\theta R(t) - \lambda_1 \frac{I_1(t)S(t)}{N(t)} - \lambda_2 \frac{I_2(t)S(t)}{N(t)} - (\beta + \xi)S(t), \\
\Pi_2(t, E) &= \lambda_1 \frac{I_1(t)S(t)}{N(t)} + \lambda_2 \frac{I_2(t)S(t)}{N(t)} - (\gamma + \xi)E(t), \\
\Pi_3(t, I_1) &= \nu\gamma E(t) - (\phi_1 + \xi + \alpha)I_1(t), \\
\Pi_4(t, I_2) &= (\alpha + \phi_1(1 - r_1))I_1(t) + qR(t) - (\phi_2 + \mu + \xi)I_2(t), \\
\Pi_5(t, R) &= (1 - \nu)\gamma E(t) + \phi_1 r_1 I_1(t) + \phi_2 I_2(t) - (q + \xi + \theta)R(t), \\
\Pi_6(t, Q) &= \beta S(t) + (1 - \pi)\theta R(t) - \xi Q(t), \\
\Pi_7(t, D) &= \mu I_2(t) - \xi D(t).
\end{aligned} \tag{5.13}$$

Since the state variables of model are non-negative and bounded, there exist positive constants $\mathcal{E}_i, i = 1, \dots, 7$, like

$$\begin{aligned}
\|S(t)\| &\leq \mathcal{E}_1, & \|E(t)\| &\leq \mathcal{E}_2, & \|I_1(t)\| &\leq \mathcal{E}_3, & \|I_2(t)\| &\leq \mathcal{E}_4, \\
\|R(t)\| &\leq \mathcal{E}_5, & \|Q(t)\| &\leq \mathcal{E}_6, & \|D(t)\| &\leq \mathcal{E}_7.
\end{aligned}$$

Additionally, we introduce the following short notation of the total population and the parameter combinations as

$$\begin{aligned}
N^+ &= \min_{t \in [0, T]} N(t), & \mathfrak{M}_1 &= \frac{\lambda_1 \mathcal{E}_3 + \lambda_2 \mathcal{E}_4}{N^+} + \beta + \xi, & \mathfrak{M}_2 &= \gamma + \xi, & \mathfrak{M}_3 &= \phi_1 + \xi + \alpha, \\
\mathfrak{M}_4 &= \phi_2 + \mu + \xi, & \mathfrak{M}_5 &= q + \xi + \theta, & \mathfrak{M}_6 &= \xi, & \mathfrak{M}_7 &= \xi,
\end{aligned}$$

then (5.12) can be written in the compact integral form

$$\begin{aligned}
 S - S_0 &= \int_0^t \Pi_1(t, S) d\tau, & E - E_0 &= \int_0^t \Pi_2(t, E) d\tau, \\
 I_1 - I_{1,0} &= \int_0^t \Pi_3(t, I_1) d\tau, & I_2 - I_{2,0} &= \int_0^t \Pi_4(t, I_2) d\tau, \\
 R - R_0 &= \int_0^t \Pi_5(t, R) d\tau, & Q - Q_0 &= \int_0^t \Pi_6(t, Q) d\tau, \\
 D - D_0 &= \int_0^t \Pi_7(t, D) d\tau.
 \end{aligned} \tag{5.14}$$

Theorem 5.8. *If $0 \leq \Gamma^* = \max_{1 \leq i \leq 7} \{\mathfrak{M}_i\} < 1$, then the kernels Π_i , $1 \leq i \leq 7$, satisfy the Lipschitz condition and exhibit contraction mappings.*

Proof. We initially establish the assertion for the kernel Π_1 and then provide approximations for the other kernels.

$$\Pi_1(t, S) = \Gamma + \pi\theta R(t) - \lambda_1 \frac{I_1(t)S(t)}{N(t)} - \lambda_2 \frac{I_2(t)S(t)}{N(t)} - (\beta + \xi)S(t).$$

Consider $S(t)$ and $S_1(t)$ be two arbitrary functions and applying the boundedness of I_1, I_2 with $N(t) \geq N^+ > 0$, we get

$$\begin{aligned}
 \|\Pi_1(t, S) - \Pi_1(t, S_1)\| &= \left\| -\lambda_1 \frac{I_1(t)S(t)}{N(t)} - \lambda_2 \frac{I_2(t)S(t)}{N(t)} - (\beta + \xi)S(t) \right. \\
 &\quad \left. + \lambda_1 \frac{I_1(t)S_1(t)}{N(t)} + \lambda_2 \frac{I_2(t)S_1(t)}{N(t)} + (\beta + \xi)S_1(t) \right\| \\
 &\leq \lambda_1 \frac{|I_1(t)|}{N^+} \|S(t) - S_1(t)\| + \lambda_2 \frac{|I_2(t)|}{N^+} \|S(t) - S_1(t)\| \\
 &\quad + (\beta + \xi) \|S(t) - S_1(t)\| \\
 &\leq \left(\frac{\lambda_1 \mathcal{E}_3 + \lambda_2 \mathcal{E}_4}{N^+} + \beta + \xi \right) \|S(t) - S_1(t)\| \\
 &= \mathfrak{M}_1 \|S(t) - S_1(t)\|.
 \end{aligned}$$

Equivalent assumptions produce for various kernels $i = 2, \dots, 7$ as

$$\begin{aligned}
 \|\Pi_2(t, E) - \Pi_2(t, E_1)\| &\leq \mathfrak{M}_2 \|E - E_1\|, \\
 \|\Pi_3(t, I_1) - \Pi_3(t, I_1^*)\| &\leq \mathfrak{M}_3 \|I_1 - I_1^*\|, \\
 \|\Pi_4(t, I_2) - \Pi_4(t, I_2^*)\| &\leq \mathfrak{M}_4 \|I_2 - I_2^*\|, \\
 \|\Pi_5(t, R) - \Pi_5(t, R_1)\| &\leq \mathfrak{M}_5 \|R - R_1\|, \\
 \|\Pi_6(t, Q) - \Pi_6(t, Q_1)\| &\leq \mathfrak{M}_6 \|Q - Q_1\|, \\
 \|\Pi_7(t, D) - \Pi_7(t, D_1)\| &\leq \mathfrak{M}_7 \|D - D_1\|.
 \end{aligned}$$

Therefore, a contraction mapping on the appropriate Banach space is defined by every kernel. $\square$

The integral system (5.14) is used to present the subsequent recursive formulations.

$$S_n = \int_0^t \Pi_1(t, S_{n-1}(\tau)) d\tau, \quad E_n = \int_0^t \Pi_2(t, E_{n-1}(\tau)) d\tau, \quad I_{1,n} = \int_0^t \Pi_3(t, I_{1,n-1}(\tau)) d\tau,$$

$$I_{2,n} = \int_0^t \Pi_4(t, I_{2,n-1}(\tau)) d\tau, \quad R_n = \int_0^t \Pi_5(t, R_{n-1}(\tau)) d\tau, \quad Q_n = \int_0^t \Pi_6(t, Q_{n-1}(\tau)) d\tau,$$

$$D_n = \int_0^t \Pi_7(t, D_{n-1}(\tau)) d\tau.$$

The differences of consecutive terms in the recursion are:

$$\begin{aligned} \mathcal{Q}_n^1(t) &= S_n - S_{n-1} = \int_0^t (\Pi_1(\tau, S_{n-1}) - \Pi_1(\tau, S_{n-2})) d\tau, \\ \mathcal{Q}_n^2(t) &= E_n - E_{n-1} = \int_0^t (\Pi_2(\tau, E_{n-1}) - \Pi_2(\tau, E_{n-2})) d\tau, \\ \mathcal{Q}_n^3(t) &= I_{1,n} - I_{1,n-1} = \int_0^t (\Pi_3(\tau, I_{1,n-1}) - \Pi_3(\tau, I_{1,n-2})) d\tau, \\ \mathcal{Q}_n^4(t) &= I_{2,n} - I_{2,n-1} = \int_0^t (\Pi_4(\tau, I_{2,n-1}) - \Pi_4(\tau, I_{2,n-2})) d\tau, \\ \mathcal{Q}_n^5(t) &= R_n - R_{n-1} = \int_0^t (\Pi_5(\tau, R_{n-1}) - \Pi_5(\tau, R_{n-2})) d\tau, \\ \mathcal{Q}_n^6(t) &= Q_n - Q_{n-1} = \int_0^t (\Pi_6(\tau, Q_{n-1}) - \Pi_6(\tau, Q_{n-2})) d\tau, \\ \mathcal{Q}_n^7(t) &= D_n - D_{n-1} = \int_0^t (\Pi_7(\tau, D_{n-1}) - \Pi_7(\tau, D_{n-2})) d\tau. \end{aligned} \quad (5.15)$$

Hence, it follows that

$$\begin{aligned} S_n(t) &= \sum_{i=1}^n \mathcal{Q}_i^1(t), & E_n(t) &= \sum_{i=1}^n \mathcal{Q}_i^2(t), & I_{1,n}(t) &= \sum_{i=1}^n \mathcal{Q}_i^3(t), & I_{2,n}(t) &= \sum_{i=1}^n \mathcal{Q}_i^4(t), \\ R_n(t) &= \sum_{i=1}^n \mathcal{Q}_i^5(t), & Q_n(t) &= \sum_{i=1}^n \mathcal{Q}_i^6(t), & D_n(t) &= \sum_{i=1}^n \mathcal{Q}_i^7(t). \end{aligned} \quad (5.16)$$

We now compute the recursive inequalities for the differences in (5.15) as follows:

$$\begin{aligned} \|\mathcal{Q}_n^1(t)\| &\leq \int_0^t \|\Pi_1(\tau, S_{n-1}) - \Pi_1(\tau, S_{n-2})\| d\tau \leq \int_0^t \mathfrak{M}_1 \|\mathcal{Q}_{n-1}^1(\tau)\| d\tau = \mathfrak{M}_1 \|\mathcal{Q}_{n-1}^1(t)\|, \\ \|\mathcal{Q}_n^2(t)\| &\leq \mathfrak{M}_2 \|\mathcal{Q}_{n-1}^2(t)\|, & \|\mathcal{Q}_n^3(t)\| &\leq \mathfrak{M}_3 \|\mathcal{Q}_{n-1}^3(t)\|, & \|\mathcal{Q}_n^4(t)\| &\leq \mathfrak{M}_4 \|\mathcal{Q}_{n-1}^4(t)\|, \\ \|\mathcal{Q}_n^5(t)\| &\leq \mathfrak{M}_5 \|\mathcal{Q}_{n-1}^5(t)\|, & \|\mathcal{Q}_n^6(t)\| &\leq \mathfrak{M}_6 \|\mathcal{Q}_{n-1}^6(t)\|, & \|\mathcal{Q}_n^7(t)\| &\leq \mathfrak{M}_7 \|\mathcal{Q}_{n-1}^7(t)\|. \end{aligned} \quad (5.17)$$

Theorem 5.9. *If for a time $T_0 > 0$ the following inequalities hold like $0 < \mathfrak{M}_i T_0 < 1$, $i = 1, 2, \dots, 7$. then a unique solution exists for model (5.1).*

Proof. We divide the proof into separate parts.

(i): Existence of solution

The functions in model (5.1) are bounded, and the kernels satisfy Lipschitz conditions. Therefore, using (5.17), the following inequality formulas are derived.

$$\begin{aligned} \|\mathcal{Q}_n^1\| &\leq \mathfrak{M}_1 t \|\mathcal{Q}_{n-1}^1\| \leq (\mathfrak{M}_1 t)^2 \|\mathcal{Q}_{n-2}^1\| \leq \dots \leq \|S(0)\| (\mathfrak{M}_1 t)^n, \\ \|\mathcal{Q}_n^2\| &\leq \|E(0)\| (\mathfrak{M}_2 t)^n, & \|\mathcal{Q}_n^3\| &\leq \|I_1(0)\| (\mathfrak{M}_3 t)^n, & \|\mathcal{Q}_n^4\| &\leq \|I_2(0)\| (\mathfrak{M}_4 t)^n, \\ \|\mathcal{Q}_n^5\| &\leq \|R(0)\| (\mathfrak{M}_5 t)^n, & \|\mathcal{Q}_n^6\| &\leq \|Q(0)\| (\mathfrak{M}_6 t)^n, & \|\mathcal{Q}_n^7\| &\leq \|D(0)\| (\mathfrak{M}_7 t)^n. \end{aligned} \quad (5.18)$$

The inequalities (5.18) indicate that the functions specified in (5.16) exist and are uniform. We demonstrate that the $S_n, E_n, I_{1,n}, I_{2,n}, R_n, Q_n, D_n$, converge to system

(5.1) solutions. $\mathcal{U}_n^i(t)$, $i = 1, 2, \dots, 7$, is defined as the remaining terms following n iterations, that is,

$$\begin{aligned} S - S_0 &= S_n + \mathcal{U}_n^1(t), \\ E - E_0 &= E_n + \mathcal{U}_n^2(t), & I_1 - I_{1,0} &= I_{1,n} + \mathcal{U}_n^3(t), & I_2 - I_{2,0} &= I_{2,n} + \mathcal{U}_n^4(t), \\ R - R_0 &= R_n + \mathcal{U}_n^5(t), & Q - Q_0 &= Q_n + \mathcal{U}_n^6(t), & D - D_0 &= D_n + \mathcal{U}_n^7(t). \end{aligned} \quad (5.19)$$

Using the Lipschitz condition for K_1 leads to

$$\|\mathcal{U}_n^1(t)\| = \left\| \int_0^t (\Pi_1(\tau, S) - \Pi_1(\tau, S_{n-1})) d\tau \right\| \leq \mathfrak{M}_1 t \|S - S_{n-1}\|.$$

Since

$$\|S - S_{n-1}\| \leq \mathfrak{M}_1 \|S - S_{n-2}\| \leq \dots \leq (\mathfrak{M}_1 t)^n \|\mathcal{U}_0^1(t)\|,$$

It follows that

$$\|\mathcal{U}_n^1(t)\| \leq (\mathfrak{M}_1 t)^n \mathcal{E}_1.$$

By setting $t = T_0$, one obtains

$$\|\mathcal{U}_n^1(t)\| \leq (\mathfrak{M}_1 T_0)^n \mathcal{E}_1. \quad (5.20)$$

Using the constraint $0 < \mathfrak{M}_1 T_0 < 1$ and the limit of (5.20) as $n \rightarrow \infty$ yields $\|\mathcal{U}_n^1(t)\| \rightarrow 0$. Consequently, $\lim_{n \rightarrow \infty} S_n(t) = S(t)$. Similarly, the following inequalities are obtained

$$\begin{aligned} \|\mathcal{U}_n^2(t)\| &\leq (\mathfrak{M}_2 T_0)^n \mathcal{E}_2, & \|\mathcal{U}_n^3(t)\| &\leq (\mathfrak{M}_3 T_0)^n \mathcal{E}_3, & \|\mathcal{U}_n^4(t)\| &\leq (\mathfrak{M}_4 T_0)^n \mathcal{E}_4, \\ \|\mathcal{U}_n^5(t)\| &\leq (\mathfrak{M}_5 T_0)^n \mathcal{E}_5, & \|\mathcal{U}_n^6(t)\| &\leq (\mathfrak{M}_6 T_0)^n \mathcal{E}_6, & \|\mathcal{U}_n^7(t)\| &\leq (\mathfrak{M}_7 T_0)^n \mathcal{E}_7. \end{aligned} \quad (5.21)$$

Limiting inequalities (5.21) as $n \rightarrow \infty$ results in $\|A_n^i(t)\| \rightarrow 0$, $i = 1, \dots, 7$. Consequently, it is demonstrated that the solutions to model (5.1) exist.

(ii): Uniqueness of solution

To prove the uniqueness of solution, contrary assume that $\mathcal{W}(t)$ and $\mathcal{W}^*(t)$ are two solution sets of the model (5.1) such that

$$\mathcal{W}(t) = (S(t), E(t), I_1(t), I_2(t), R(t), Q(t), D(t),$$

$$\mathcal{W}^*(t) = (S^*(t), E^*(t), I_1^*(t), I_2^*(t), R^*(t), Q^*(t), D^*(t)).$$

Then, using the condition $0 < \mathfrak{M}_1 t < 1$, one has

$$\|S(t) - S^*(t)\| = \left\| \int_0^t (\Pi_1(t, S(\tau)) - \Pi_1(t, S^*(\tau))) d\tau \right\| \leq \mathfrak{M}_1 t \|S(t) - S^*(t)\|.$$

This implies that $(1 - \mathfrak{M}_1 t) \|S(t) - S^*(t)\| \leq 0$. Finally, one gets $\|S(t) - S^*(t)\| = 0$ or $S(t) = S^*(t)$. This contradicts the supposition. Hence $S(t)$ has a unique solution. Similarly, one obtains

$$E(t) = E^*(t), \quad I_1(t) = I_1^*(t), \quad I_2(t) = I_2^*(t),$$

$$R(t) = R^*(t), \quad Q(t) = Q^*(t), \quad D(t) = D^*(t).$$

As a result, all related components of the two solutions coincide, demonstrating the model (5.1) solution is unique. $\square$

5.4 Construction of data driven model

Mathematical models are crucial in epidemic forecasting for understanding disease propagation dynamics. Although they frequently rely on parameters that are not entirely known or quantifiable from the real data. In case of limited data, they can be estimated from synthetic data through parameter identification. In order to better understand the addiction and depression dynamics using model (5.1), we use data fitting to find correlations in complicated data sets. We want to improve our comprehension of depression dynamics by incorporating real-world data such as demographics, medical evaluations, and social factors into the model. Consequently, in the following subsection, we cover how to define the parameters for the model.

5.4.1 Parameter estimation

A critical phase in mathematical modeling is parameter estimation, which entails deriving unknown parameters and initial conditions from real data. Optimization approaches such as constrained nonlinear programming are employed to reduce the differences between observations and forecasts while preserving the biological importance of parameter values. To develop our approach, we focus on the depression data of Spain from 2011 to 2022 available on [204]. For estimate the parameters λ_1 , λ_2 , α , q , and ϕ_2 with description are given in Table 5.1, we formulate the following minimizing problem

$$\begin{aligned} \min_{\theta, x_0} \quad & J(\theta, x_0) \\ \text{subject to} \quad & \frac{dx}{dt} = f(x, \theta), \\ & x_0^{\min} \leq x_0 \leq x_0^{\max}, \\ & \theta^{\min} \leq \theta \leq \theta^{\max}. \end{aligned} \quad (5.22)$$

with the compartments of model (5.1) in compact form written as

$$\frac{dx(t)}{dt} = f(x(t), \theta), \quad x(0) = x_0, \quad (5.23)$$

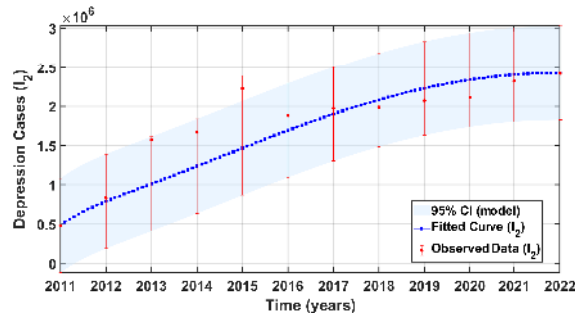


FIGURE 5.1: Graphical representation of the model fits actual depression instances in Spain.

$$x(t) = \begin{bmatrix} S(t) \\ E(t) \\ I_1(t) \\ I_2(t) \\ R(t) \\ Q(t) \\ D(t) \end{bmatrix}, \quad x_0 = \begin{bmatrix} S(0) \\ E(0) \\ I_1(0) \\ I_2(0) \\ R(0) \\ Q(0) \\ D(0) \end{bmatrix}, \quad \theta = \begin{bmatrix} \lambda_1 \\ \lambda_2 \\ \alpha \\ q \\ \phi_2 \end{bmatrix}.$$

where $x(t)$ is the state vector, and θ is the vector of unknown parameters, and x_0 represents the unknown initial conditions. The objective function is defined as

$$J(\theta, x_0) = \sum_{i=1}^n \left(\frac{I_2^{\text{model}}(t_i; x_0, \theta)}{I_2^{\text{max}}} - \frac{I_2^{\text{obs}}(t_i)}{I_2^{\text{max}}} \right)^2,$$

where $I_2^{\text{model}}(t_i; x_0, \theta)$ is the model-predicted value of the severe depression compartment at time t_i , $I_2^{\text{obs}}(t_i)$ is the reported depressed cases in Spain data, and $I_2^{\text{max}} = \max \{ I_2^{\text{obs}}(t_i) \}$ is used for normalization. We choose the time interval $t \in (2011, 2022)$ with step size $\Delta t = 0.01$ and define the following constrain to search minimum of θ

$$0 \leq \lambda_1 \leq 2, \quad 0 \leq \lambda_2 \leq 2, \quad 0 \leq \alpha \leq 0.15, \quad 0 \leq q \leq 1, \quad 0 \leq \phi_2 \leq 1$$

where the bounds are determined based on the available literature. Matlab routines *fmincon* and RK4 approach for the system of ODE's are used to tackle the optimization problem in (5.22). The simulation results of this optimization problem over the model are shown in Fig 5.1 with the estimated parameter values following as

$$\lambda_1^{\text{est}} = 0.9791, \quad \lambda_2^{\text{est}} = 0.0917, \quad \alpha^{\text{est}} = 0.1491, \quad q^{\text{est}} = 0.9704, \quad \phi_2^{\text{est}} = 0.0069$$

The predicted dynamics accurately depict the general trend of the available data, although certain discrepancies persist because the actual data is nonlinear and non-smooth. The majority of data points, however, fall within the model prediction's 95% confidence interval, suggesting that the fitted parameters offer a statistically sound depiction of the dynamics that are being observed. To calculate the mismatch, we define the error as

$$\text{Error}(t) = |I_2^{\text{model}}(t) - I_2^{\text{obs}}(t)|$$

who visualize in Fig 5.1. Here we explicitly describe all the parameters used for the simulation of model (5.1) as follows

1. The recruitment rate (Γ) is the number of new individuals who are supposed to be susceptible to social media users, leading to depression. Based on the data available in [268], we take its value $\frac{7.816}{10000}$.
2. Addiction transmission is defined as the rate at which susceptible individuals $S(t)$ interact with the class $I_1(t)$. Its value is approximated from observed data as $\lambda_1=0.9790$.
3. The depression transmission is the interaction of susceptible individuals with the depressed class is the rate at which individuals of $S(t)$ interact with $I_2(t)$. We approximate that $S(t)$ has $\lambda_2 = 0.0917$ such contacts.

4. In the recovered class, there is some percentage of individuals who are now susceptible and ready to develop SMA. It means that these individuals recovered from some initial conditions, but they are at risk of developing the effects of SMA. The value is $\pi=0.35$ [168].
5. In the recovered class, individuals who are ready to quit the use of social media will be moved to the permanent quieter class with $\theta= 0.1[0-0.4]$ [12].
6. When a population's number of deaths is calculated according to its size and expressed as a percentage per unit of time, the result is the mortality rate, also called the death rate. The mortality rate is given as $\xi = 0.0125$ [218].
7. The probability or rate at which exposure results in addiction is indicated by the fraction of exposed people who become members of the addicted class is represented by ν . This can be important for developing approaches to intervention as well as for comprehending the dynamics of addiction spread within a population. Thus, $\nu= 0.7$ [15].
8. The rate at which individuals leave the exposed class is given as $\gamma= 0.25$ [168].
9. We suppose that the Suicidal death rate due to excessive use of social media (by cyberbullying, threats, bad comments, etc) is $\mu = 0.4$.
10. We assume that the mean duration of an individual in the addicted class before experiencing depression is denoted by $\frac{1}{\alpha}$. We approximate this from the observed data as $\alpha= 0.1491$.
11. The effectiveness of treatment for the addict population is the probability of how successful the treatment is, and an individual in $I_1(t)$ will move to $R(t)$. According to a survey, about 59 percent of individuals admitted to recovering from SMA with treatments. Therefore, we assume its value as $r_1 =0.29[0-0.6]$ [265].
12. The rate at which the addict population accepts/rejects treatment is shown by $\frac{1}{\phi_1}$. assuming the average time for treatment like Cognitive Behavioral Therapy (CBT) is about 300 days with $\phi_1 = 0.003$.
13. The rate at which the depressed population accepts/rejects treatment is shown by $\frac{1}{\phi_2}$. The average time for treatment is approximated as $\phi_2 = 0.0069$.
14. The people who leave the treatment in the middle and try to overthink, in such people, due to negative thinking patterns, biological factors, anxiety, or mental health disorders, etc, the addiction symptoms return after some time of improvements, which is called relapse. Thus, the probability of relapsing is approximated as $q = 0.9704$.
15. Some good people instantly take self-control and start to stop or avoid the excessive use of social media. The value of this parameter is $\beta = 0.09[0.01-0.2]$ [15].

According to the available data of Spain, we average the total population between 2010 to 2022, $N(t) = 4.6729420 \times 10^7$. The estimated initial values of all classes up

to five decimal places are given as

$$\begin{aligned} S(0) &= 4.27602 \times 10^7, & E(0) &= 2.3975 \times 10^4, & I_1(0) &= 3.27105 \times 10^6, \\ I_2(0) &= 4.79500 \times 10^5, & R(0) &= 1.43850 \times 10^5, & Q(0) &= 4.7950 \times 10^4, \\ D(0) &= 2.85049 \times 10^3. \end{aligned}$$

TABLE 5.1: Parameters of Model

Parameters	Symbol	Units
Recruitment rate	Γ	Per-capita/year
rate at which addiction spreads to susceptible	λ_1	Per capita/year rate
rate at which depression spreads to susceptible	λ_2	Per capita/year rate
Proportion of recovered individuals susceptible to SMA	π	Dimensionless
Individuals that leave recovered class	θ	1/year
Natural death rate/Mortality rate	ξ	1/year
Proportion of exposed individuals who eventually become addicted	ν	Dimensionless
Individuals that leave exposed class	γ	1/year
The rate at which people commit suicide due to depression	μ	1/year
Treatment rate for the addicted population	ϕ_1	1/day
Treatment rate for the depressed population	ϕ_2	1/day
The fraction of individuals whose treatment is successful	r_1	Dimensionless
Per capita rate of depression due to media influence.	α	1/year
The probability of relapse of recovered Individuals	q	1/year
Susceptible abstaining from social media	β	1/year

Machine learning, especially neural networks, can improve epidemic models' prediction by introducing observed data and parameter heterogeneity. To enable neural networks to provide training samples and discover the relationship between data and system dynamics, randomized changes are introduced around parameters. This improves the precision of the model by enabling the network to capture non-linear interactions and fine-tune predictions that conventional models restricted by fixed parameters would ignore. *Feed Forward neural network* (FNN) over the epidemic model utilizes the observed data with literature-initialized parameters to produce the training samples. The network learns the functional mapping between inputs like time, population compartments, and outputs directly from data, approximating system dynamics and capturing nonlinear relationships that conventional mechanistic models might not be able to capture. FNN is effective in capturing nonlinear interactions, but its dependability may be limited due to its lack of a mechanistic foundation and reliance on a large, high-quality database. *Physics Informed neural network* (PINN) incorporates model equations into training samples, which ensures consistency with the residuals of the differential equations in the model, rather than just minimizing the error between predictions and real or synthetic data. Such networks can refine parameters during training by initializing them from literature and letting them fluctuate within feasible limits. PINN may learn from sparse or noisy information using this dual loss technique, combining data fitting and physical law enforcement, producing predictions that are aligned with established epidemic dynamics. We apply both techniques on model (5.1) to predict the addiction and depression dynamics.

5.4.2 Feed forward neural network

We propose a data-driven strategy that integrates synthetic data generation with real data by utilizing a *Feed Forward Neural Network* (FNN). This approach aims to predict the dynamics of addiction and depression as described in model equation (5.1), allowing for an evaluation of the complex system through the calibration of specific parameters. This approach is based on the general neural network framework. The model (5.1) is described in a compact way

$$\frac{d\mathcal{U}(t)}{dt} = \mathcal{F}(\mathcal{U}(t); \theta),$$

where $\mathcal{U}(t) = [S, E, I_1, I_2, R, Q, D]^\top$ are state vectors and $\theta \in \mathbb{R}^n$ is the vector of biological parameters in which $\pi, \theta, \beta, \zeta, \gamma, \nu, \phi_1, r_1$ assume to be unknown for forecasting model. The FNN is intended for estimating the inverse mapping

$$\mathcal{N}_\omega : (\mathcal{U}(t), t) \mapsto \theta,$$

where ω denotes the network weights and biases. Thus, the network learns.

$$\theta \approx \mathcal{N}_\omega(\mathcal{U}(t), t),$$

eliminating the need for iterative optimization. The parameter identification problem seeks to recover θ from observed state trajectories $\{\mathcal{U}_{\text{obs}}(t_i)\}_{i=1}^N$. The governing ODEs mimic the typical epidemic flow with given parameters in Sec 5.4.1. The baseline mutual addiction and depression dynamics are represented by the fixed true parameter values θ_{true} . We create synthetic training data by using the forward model after introducing random perturbations from the baseline of the true parameters.

$$\theta_j^{(i)} = \theta_{j,\text{true}} \times (\eta + \text{rand}(0, 1)), \quad i = 1, \dots, N_{\text{train}}.$$

For each parameter $\theta^{(i)}$, the trajectory $\mathcal{U}^{(i)}(t_k)$ is generated by integrating $\dot{\mathcal{U}} = f(\mathcal{U}, t; \theta^{(i)})$ using Runge-Kutta method. The training set is defined as

$$\mathcal{T} = \{(\mathcal{U}^{(i)}(t_k), t_k), \theta^{(i)}\},$$

where `mapminmax` transformation is applied to all inputs and target values for training stability. The network parameters are trained by minimizing a combined loss function to balance the synthetic trajectory with actual data, given as

$$\theta^* = \arg \min_{\theta} J(\theta),$$

where

$$J(\theta) = \omega_r J_r(\theta) + \omega_s J_s(\theta),$$

and $\omega_r, \omega_s > 0$ regulate the proper participation of synthetic and actual data. A mean-squared error (MSE) over the relevant dataset is usually used for every aspect of the composite loss. The MSE for the synthetic data is provided by

$$J_s(\omega) = \frac{1}{N_s} \sum_{i=1}^{N_s} \left\| \mathcal{N}_\omega(\mathcal{U}^{(i)}(t), t) - \theta^{(i)} \right\|_2^2,$$

furthermore, for the actual data,

$$J_r(\omega) = \frac{1}{N_r} \sum_{i=1}^{N_r} \left\| \mathcal{N}_\omega(\mathcal{U}_{\text{real}}^{(i)}(t), t) - \theta_{\text{real}}^{(i)} \right\|_2^2.$$

We used the Levenberg-Marquardt approach to optimize the network parameters (ω) as

$$\omega^{(k+1)} = \omega^{(k)} - (J^\top J + \mu I)^{-1} J^\top e,$$

where e is the error vector, J is the Jacobian of the residuals, and μ is a dampening component with identity matrix I . This procedure iteratively updates the network weights to minimize the composite weighted MSE. Let ω represent the set of all network weights and biases. For an input pair $(\mathcal{U}^{(i)}(t_k), t_k)$, the method of forward propagation is executed accordingly. For neural network approximation, we utilize the FFN with $\mathcal{L} + 1$ layers with input vector x , the proposed network computes

- **Input Layer:** here, the current time t is the only input feature utilized by the model, written as

$$x = \begin{bmatrix} \mathcal{U}^{(i)}(t_k) \\ t_k \end{bmatrix} \in \mathbb{R}^{d-1} \times \mathbb{R}.$$

- **Hidden Layers:** The connection between time and the epidemic levels is captured by several fully linked layers with nonlinear activation functions. For $\ell = 1, \dots, L - 1$,

$$\begin{aligned} x^1 &= W^1 x + b^1 \\ x^\ell &= \sigma^{(\ell)} \left(W^{(\ell)} x^{(\ell-1)} + b^{(\ell)} \right) \end{aligned}$$

where $W^{(\ell)}$, $b^{(\ell)}$, and $\sigma^{(\ell)}$ denote the weights, biases, and activation function of layer ℓ .

- **Output Layer:** The outcome is a node that represents the expected value of the epidemic dynamics at time t .

$$\mathcal{N}_w(x) = W^{(L)} x_{(L-1)} + b^{(L)}.$$

Here, $\omega = (W^1, \dots, W^L, b^1, \dots, b^L)$ denotes the complete set of network parameters. The predicted parameters for sample (i, k) are $\hat{\theta}^{(i)}(t_k) = \mathcal{N}_\omega((\mathcal{U}^{(i)}(t_k), t_k))$, and the corresponding residual is

$$e_{i,k} = \hat{\theta}^{(i)}(t_k) - \theta^{(i)}.$$

When $\bar{\theta}$ is substituted into the RK4 model, expected trajectory estimates $\mathcal{U}_{\text{pred}}(t)$ are obtained. These are contrasted with $\mathcal{U}_{\text{true}}(t)$ employing normalized absolute error as

$$\varepsilon_j(t) = \frac{|\mathcal{U}_{\text{pred},j}(t) - \mathcal{U}_{\text{true},j}(t)|}{\mathcal{U}_{\text{true},j}(0)}. \quad (5.24)$$

This framework transforms the classical inverse problem into a supervised learning task, enabling fast and noise-tolerant parameter identification. Once trained, the FFN instantly estimates behavioral transition parameters for new data, facilitating adaptive modeling and control of social media addiction and depression dynamics. FFNs' accuracy in forecasting potential epidemic dynamics is hampered by their incapacity to simulate sequential dependency since they lack memory systems, which

also limits their interpolation efficiency. Due to this reason, we define another technique in the next subsection.

5.4.3 Physics informed neural network

We present a methodology for parameter identification in the model system (5.1) that makes use of *Physics Informed Neural Networks* (PINN) to estimate additional parameters and evaluate prediction models with significant accuracy and precision. Recent studies illustrate the emerging impact of integrating the epidemic models with PINN, particularly in the setting of limited, noisy, and partially missing data, which is a common real-world complexity in various epidemiological surveillance. Theory behind PINN is similar to that of literature-informed learning, in which the absence of high-quality data is addressed by using mathematical models that provide structure and information. They also ensure that the trained dynamics are compatible with biological concepts associated with disease transmission by employing the model equations to drive the neural network instead of relying just on data fitting. Using this paradigm, [255] estimates the incidence and recovery rates of the compartment model using Disease-Informed Neural Network (DINN) based on the concept of PINN from sparse data, including classical epidemic equations within the learning mechanism. A similar study [4] demonstrates how model-driven learning may retrieve hidden information and variables in an SEIR-type model by imposing the relevant ODE restrictions via automated differentiation. These approaches go beyond forecasting disease models may also be used to predict patient addiction behaviors by utilizing theoretical frameworks as priors, especially when empirical data is scarce [36]. More recent study [312] shows adaptation of this framework with limited information by using Caputo fractional derivatives to find biological parameters, including memory effects. These studies motivate that in the case of limited but reliable data, hybrid model-data techniques work best. This is consistent with the current work's goal of using model-specific limitations to improve parameter identification, accurate forecasting, and clarity in model (5.1).

PINN integrates the fundamental system of governing equations directly into the learning process, in contrast to traditional neural networks as in Sec 5.4.2. In our scenario, these equations represent the actual model (5.1), which guarantees that the network forecasts are data-driven and in line with the model dynamics. We use synthetic data from the limited depression class observations to illustrate the efficacy of this technique for the model, demonstrating that PINN can recover the genuine parameters with high accuracy while remaining consistent with the proposed behavior dynamics. A schematic diagram of this approach is shown in Fig 5.2. The model (5.1) is described in a compact way

$$\frac{d\mathcal{U}(t)}{dt} = \mathcal{F}(\mathcal{U}(\cdot, \cdot)),$$

where we assume state trajectory function in vector form as

$$\mathcal{U}(t; \mathcal{E}) = \left[S, E, I_1, I_2, R, Q, D \right]^\top \in \mathbb{R}^7$$

on which we want to apply proposed strategy such that $\mathcal{E} \in \mathbb{R}^n$ where $\mathcal{E}$ is the vector related to chosen parameters for estimation to predict model dynamics. The variables S, E, I_1, I_2, R, Q, D represent the system classes with the assumption that

the total living population is

$$N(t) = S(t) + E(t) + I_1(t) + I_2(t) + R(t) + Q(t) + D(t).$$

This leads to our key idea in PINN formulation, where the solution of a system of differential equations is approximated using a neural network trained by observable data from the Runge-Kutta technique and governing equations. In the proposed strategy setting, the network learns the full dynamics trajectories

$$(S, E, I_1, I_2, R, Q, D) \in \Psi$$

along with assuming unknown parameters $\mathcal{E}$.

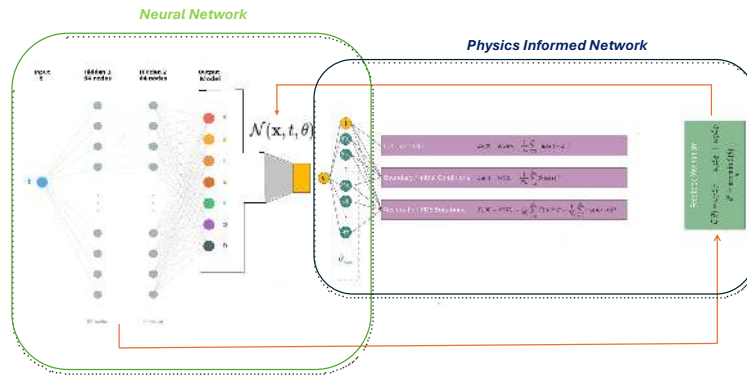


FIGURE 5.2: A representation of the proposed PINN approach to estimating the model (5.1) solution. An input layer with a single input variable—a time coordinate—makes up the trained network. Each of the three hidden layers has n neurons that are connected to the input and output layers. Since the intended response is expressed by a single neuron, the output layer has model state vectors. Using state vectors and their derivatives, this neural network design is used to evaluate the residual of the ordinary differential equation. A complete loss function is created by merging partial loss functions. After that, it is weighted and reduced as necessary.

The discretization of the training data is define $\{t^j, \mathcal{U}^j\}_{j=0}^{N_{data}}$, N_{data} represents the number of training samples. The approximation is shown as $\hat{\mathcal{U}}(t; \mathcal{E}, \theta)$. The neural network approximation process uses a neural network with parameters θ vector of parameter learning specified by the neural network design, to estimate the state trajectory

$$\hat{\mathcal{U}}(t; \mathcal{E}, \theta) = [\hat{S}(t), \hat{E}(t), \hat{I}_1(t), \hat{I}_2(t), \hat{R}(t), \hat{Q}(t), \hat{D}(t)]^\top.$$

The network weights and unknown biological parameters are refined simultaneously in a hybrid process. This technique makes it easier for the model to estimate parameters while reconstructing unidentified states, especially in situations when data is limited. Furthermore, the integration of underlying physics serves as a regulative constraint, preventing over-fitting and enhancing the model's comprehension.

Time derivatives are computed via automatic differentiation:

$$\frac{d}{dt}\hat{\mathcal{U}}(t; \mathcal{E}, \theta) = \left(\frac{d}{dt}\hat{S}(t), \frac{d}{dt}\hat{E}(t), \frac{d}{dt}\hat{I}_1(t), \frac{d}{dt}\hat{I}_2(t), \frac{d}{dt}\hat{R}(t), \frac{d}{dt}\hat{Q}(t), \frac{d}{dt}\hat{D}(t) \right)^\top.$$

As we mentioned earlier, PINN also incorporates a system of model equations (5.1) into its loss function, compared to traditional approaches that just use observable data for training. This combination is accomplished by expecting the neural network outputs to accurately reflect the behavior model's core dynamics. In particular, the derivatives $\frac{d}{dt}\hat{\mathcal{U}}(\cdot, \cdot)$ are added to the right-hand side of the system after being estimated numerically. This mechanism effectively mitigates differences between the neural network forecasts and the proposed behavioral model by making sure that the predicted residuals approach to zero for the true solution. This strengthens the model's coherence with the observed dynamics. The residual vector at time t is

$$\mathcal{R}(t; \mathcal{E}, \theta) = \frac{d}{dt}\hat{\mathcal{U}}(t; \mathcal{E}, \theta) - \mathcal{F}(\hat{\mathcal{U}}(t; \mathcal{E}, \theta); \mathcal{E}), \quad (5.25)$$

such that

$$\begin{aligned} \mathcal{R}_S(t) &= \frac{d}{dt}\hat{S}(t) - \left(\Gamma + \pi\theta\hat{R}(t) - \lambda_1 \frac{\hat{I}_1(t)\hat{S}(t)}{\hat{N}(t)} - \lambda_2 \frac{\hat{I}_2(t)\hat{S}(t)}{\hat{N}(t)} - (\beta + \xi)\hat{S}(t) \right), \\ \mathcal{R}_E(t) &= \frac{d}{dt}\hat{E}(t) - \left(\lambda_1 \frac{\hat{I}_1(t)\hat{S}(t)}{\hat{N}(t)} + \lambda_2 \frac{\hat{I}_2(t)\hat{S}(t)}{\hat{N}(t)} - (\gamma + \xi)\hat{E}(t) \right), \\ \mathcal{R}_{I_1}(t) &= \frac{d}{dt}\hat{I}_1(t) - \left(\nu\gamma\hat{E}(t) - \phi_1\hat{I}_1(t) - (\xi + \alpha)\hat{I}_1(t) \right), \\ \mathcal{R}_{I_2}(t) &= \frac{d}{dt}\hat{I}_2(t) - \left(\alpha\hat{I}_1(t) + \phi_1(1 - r_1)\hat{I}_1(t) + q\hat{R}(t) - \phi_2\hat{I}_2(t) - (\mu + \xi)\hat{I}_2(t) \right), \\ \mathcal{R}_R(t) &= \frac{d}{dt}\hat{R}(t) - \left((1 - \nu)\gamma\hat{E}(t) + \phi_1r_1\hat{I}_1(t) + \phi_2\hat{I}_2(t) - q\hat{R}(t) - (\xi + \theta)\hat{R}(t) \right), \\ \mathcal{R}_Q(t) &= \frac{d}{dt}\hat{Q}(t) - \left(\beta\hat{S}(t) + (1 - \pi)\theta\hat{R}(t) - \xi\hat{Q}(t) \right), \\ \mathcal{R}_D(t) &= \frac{d}{dt}\hat{D}(t) - \left(\mu\hat{I}_2(t) - \xi\hat{D}(t) \right). \end{aligned}$$

The training problem is a joint optimization over network parameters θ and epidemiological parameters $\mathcal{E}$

$$(\hat{\mathcal{E}}, \hat{\theta}) = \arg \min_{\theta, \mathcal{E}} \mathcal{L}(\mathcal{E}, \theta).$$

The weighted loss function combines the components

$$\mathcal{L}_{total}(\hat{\mathcal{E}}, \hat{\theta}) = \Omega_{\text{phy}} \mathcal{L}_{\text{phy}} + \Omega_{\text{ic}} \mathcal{L}_{\text{ic}} + \Omega_{\text{data}} \mathcal{L}_{\text{data}}. \quad (5.26)$$

At collocation points $\{t_j\}_{j=1}^{N_{\text{data}}}$, the physical or data loss is defined as a weighted sum of individual compartment residuals

$$\mathcal{L}_{\text{phy}}(\hat{\mathcal{E}}, \hat{\theta}) = \sum_{i=1}^7 \Omega_{\text{phy},i} \mathcal{L}_i(\hat{\mathcal{E}}, \hat{\theta}). \quad (5.27)$$

Each compartment contribution is given by

$$\mathcal{L}_i(\hat{\mathcal{E}}, \hat{\theta}) = \frac{1}{N_{data}} \sum_{j=1}^{N_{data}} |\mathcal{R}_i(t_j)|^2.$$

where i represents the model compartments. For observational data at time points $\{t_k\}_{k=1}^{N_{data}}$, the data loss is defined as

$$\mathcal{L}_{data}(\hat{\mathcal{E}}, \hat{\theta}) = \sum_{i=1}^7 \Omega_{data,i} \frac{1}{N_{data}} \sum_{k=1}^{N_{data}} |u_k^i - \hat{u}_k^i(\hat{\mathcal{E}}, \hat{\theta})|^2,$$

where u_k^i is the observed value of compartment i at time t_k and $\hat{u}_k^i$ is the neural network prediction. At the initial time t_0 , the initial condition loss is defined as

$$\mathcal{L}_{ic}(\hat{\mathcal{E}}, \hat{\theta}) = \sum_{i=1}^7 \Omega_{ic,i} |u_0^i - \hat{u}_0^i(\hat{\mathcal{E}}, \hat{\theta})|^2.$$

Altogether, this formulation provides a robust biological and data-driven foundation for the subsequent studies, ensuring that our predictions and validation procedures are based on a well-established and extensively verified epidemiological paradigm.

5.5 Global sensitivity analysis

Sensitivity analysis is the critical component in epidemiological dynamics that precisely describes how variation in biological parameters effectively changes the evaluation of disease prediction and stability. All types of epidemic models rely on certain parameters that describe the contact heterogeneity, pathogens' characteristics, host interaction, disease transmission, etc. Model's reliability and inter-predictability depend on these parameters, which are mostly estimated from uncertain data. Sensitivity analysis aims to identify key parameters, assess uncertainty, guide interventions, and validate the model. Local sensitivity quantifies the variation in model prediction with small perturbations around baseline in parameter values, which did not capture the nonlinear behavior within a broad range of parameter values. Mathematically, we express them as a partial derivative or elastic incidence as

$$\mathcal{S}_j = \frac{\partial \mathcal{Y}}{\partial \mathcal{X}_j} \frac{\mathcal{X}_j}{\mathcal{Y}},$$

where $\mathcal{Y}$ represents model output with considered parameters shown as $\mathcal{X}_j$. Global sensitivity examines how the uncertainty in the model result correlates to the input parameter values when these parameters fluctuate concurrently across their entire feasible ranges. It investigates the whole input space and seeks to capture the impact of non-linearities and interactions in model data. Some popular techniques include the Sobol variation decomposition [264, 254], Latin Hypercube Sampling (LHS), Partial Rank Correlation Coefficient (PRCC) [54, 198], and Mirror screening methods [206]. Among these techniques, Latin Hypercube Sampling combined with Partial Rank Correlation Coefficient (LHS-PRCC) convinced us due to their reliable computational efficiency with the ability to accurately describe the monotonic relation among parameters with model variables.

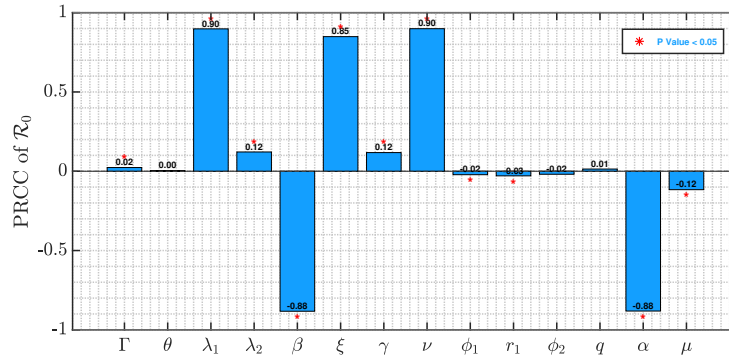


FIGURE 5.3: Evaluation of the global sensitivity of basic reproduction number given in (5.4) of behavioral dynamics proposed in model (5.1). The output shows that $\Lambda, \lambda_1, \xi, \gamma, \nu$ are significant parameters with positive magnitude while $\beta, \phi_1, r_1, \alpha, \mu$ are significant parameters with negative magnitude. Particularly parameters $\lambda_1, \beta, \xi, \nu, \alpha$ are strongly influence the disease state. To decrease the addition and depression transmission in infected individuals, λ_1, ξ, ν should be decreased in a positive direction while β, α increase in a negative direction. For the simulation of the outputs, we take a sample size $N = 10000$ with $t \in [0, 1000]$ having step size $dt = 1$ and time point of interest 500.

LHS-PRCC is especially well adapted to epidemic models, where outputs are frequently determined by several interdependent variables expressed in a complex way. The parameter space sampled utilizing LHS with the assumption that the epidemic model contains n uncertain parameters, with each parameter value lying in a specific interval or may be distributed using the cumulative distribution function (CDF). Particularly, for each parameter, we specify a corresponding probability space into $\mathcal{N}$ intervals of equal distribution, like

$$\left[0, \frac{1}{\mathcal{N}}\right), \left[\frac{1}{\mathcal{N}}, \frac{2}{\mathcal{N}}\right), \dots, \left[\frac{\mathcal{N}-1}{\mathcal{N}}, 1\right).$$

Thus, from each interval, we sample a uniform random value with $\mathcal{U}_j^{(i)} \sim \text{Uniform}\left(\frac{i-1}{\mathcal{N}}, \frac{i}{\mathcal{N}}\right)$ where $i = 1, \dots, \mathcal{N}$. To make sure correct space-filling qualities and avoid incorrect correlations, randomly permute the sampled values for each parameter separately, leading to the final form of the LHS sample matrix, given as

$$\mathbf{X} = \begin{pmatrix} \mathcal{X}_1^{(\psi_1(1))} & \mathcal{X}_2^{(\psi_2(1))} & \dots & \mathcal{X}_n^{(\psi_n(1))} \\ \mathcal{X}_1^{(\psi_1(2))} & \mathcal{X}_2^{(\psi_2(2))} & \dots & \mathcal{X}_n^{(\psi_n(2))} \\ \vdots & \vdots & \ddots & \vdots \\ \mathcal{X}_1^{(\psi_1(\mathcal{N}))} & \mathcal{X}_2^{(\psi_2(\mathcal{N}))} & \dots & \mathcal{X}_n^{(\psi_n(\mathcal{N}))} \end{pmatrix}, \quad (5.28)$$

where ψ_j represents the random permutation for each parameter j . To extract the result of interest for each parameter set $\mathbf{X}^{(i)}$, compute the epidemic model

$$Y^{(i)} = \mathcal{F}\left(X_1^{(i)}, X_2^{(i)}, \dots, X_n^{(i)}\right),$$

where $\mathcal{F}(\cdot)$ indicates the model computation and output collection. This leads to the output vector

$$\mathbf{Y} = \left(Y^{(1)}, Y^{(2)}, \dots, Y^{(N)} \right)^T.$$

Notably, for reliable sensitivity estimates, the sample size N must be extreme. PRCC is calculated to determine parameter sensitivity while considering uncertainties after LHS generates the sample matrix $\mathbf{X}$ and the model outputs $\mathbf{Y}$. To identify a monotonic relation, change both parameter samples and outputs to ranks, such as

$$\tilde{X}_j^{(i)} = \text{rank} \left(X_j^{(i)} \right), \quad \tilde{Y}^{(i)} = \text{rank} \left(Y^{(i)} \right),$$

After considering all other factors, the PRCC consequently quantifies the distinct, partial relationship between X_j and Y . Thus, supposing the variance and covariance formulation without normalization, we define

$$\begin{aligned} \text{Cov}(\mathfrak{R}_{X_j}, \mathfrak{R}_Y) &= \sum_{i=1}^N \left(\mathfrak{R}_{X_j}^{(i)} - \bar{\mathfrak{R}}_{X_j} \right) \left(\mathfrak{R}_Y^{(i)} - \bar{\mathfrak{R}}_Y \right), \\ \text{Var}(\mathfrak{R}_{X_j}) &= \sum_{i=1}^N \left(\mathfrak{R}_{X_j}^{(i)} - \bar{\mathfrak{R}}_{X_j} \right)^2, \quad \text{Var}(\mathfrak{R}_Y) = \sum_{i=1}^N \left(\mathfrak{R}_Y^{(i)} - \bar{\mathfrak{R}}_Y \right)^2, \end{aligned}$$

PRCC between parameter X_j and output Y as the Pearson correlation coefficient of their residuals within the case of $\bar{\mathfrak{R}}_{X_j} = \bar{\mathfrak{R}}_Y = 0$ implies ordinary least square residual has zero mean when an intercept is included, we write it as

$$\text{PRCC}(X_j, Y) = \frac{\sum_{i=1}^N \mathfrak{R}_{X_j}^{(i)} \cdot \mathfrak{R}_Y^{(i)}}{\sqrt{\sum_{i=1}^N \left(\mathfrak{R}_{X_j}^{(i)} \right)^2} \sqrt{\sum_{i=1}^N \left(\mathfrak{R}_Y^{(i)} \right)^2}}, \quad (5.29)$$

where the residuals of parameter X_j and output Y that cannot be clarified using the other parameters are represented by $\mathfrak{R}_{X_j}$ and $\mathfrak{R}_Y$. To determine the individual contribution of every parameter X_j to the result Y , two ordinary least squares regressions are computed as

$$\tilde{X}_j = \alpha_j + \sum_{\substack{\ell=1 \\ \ell \neq j}}^k \beta_{j\ell} \tilde{X}_\ell + \varepsilon_{X_j}, \quad \mathfrak{R}_{X_j} = \varepsilon_{X_j} = \tilde{X}_j - \hat{\tilde{X}}_j,$$

shows how the ranked parameter $\tilde{X}_j$ is regressed on all other ranked parameters with residual $\mathfrak{R}_{X_j}$ where $\hat{\tilde{X}}_j$ as predicted values during regression. The ranked output $\tilde{Y}$ is simultaneously regressed on all ranked parameters except X_j as follows

$$\tilde{Y} = \alpha_0 + \sum_{\substack{\ell=1 \\ \ell \neq j}}^k \gamma_\ell \tilde{X}_\ell + \varepsilon_Y, \quad \mathfrak{R}_Y = \varepsilon_Y = \tilde{Y} - \hat{\tilde{Y}},$$

possessing the residual $\mathfrak{R}_Y$ for the model output. Finally, to make the desired sensitivity of model output with respect to parameters, we may apply the T-test, making sure the observed PRCC is statistically significant through corresponding p-values.

We compute the sensitivity of the basic reproduction number $\mathbb{R}_0$ and the system of order differential equations of model (5.1) by the proposed LHS-PRCC techniques,

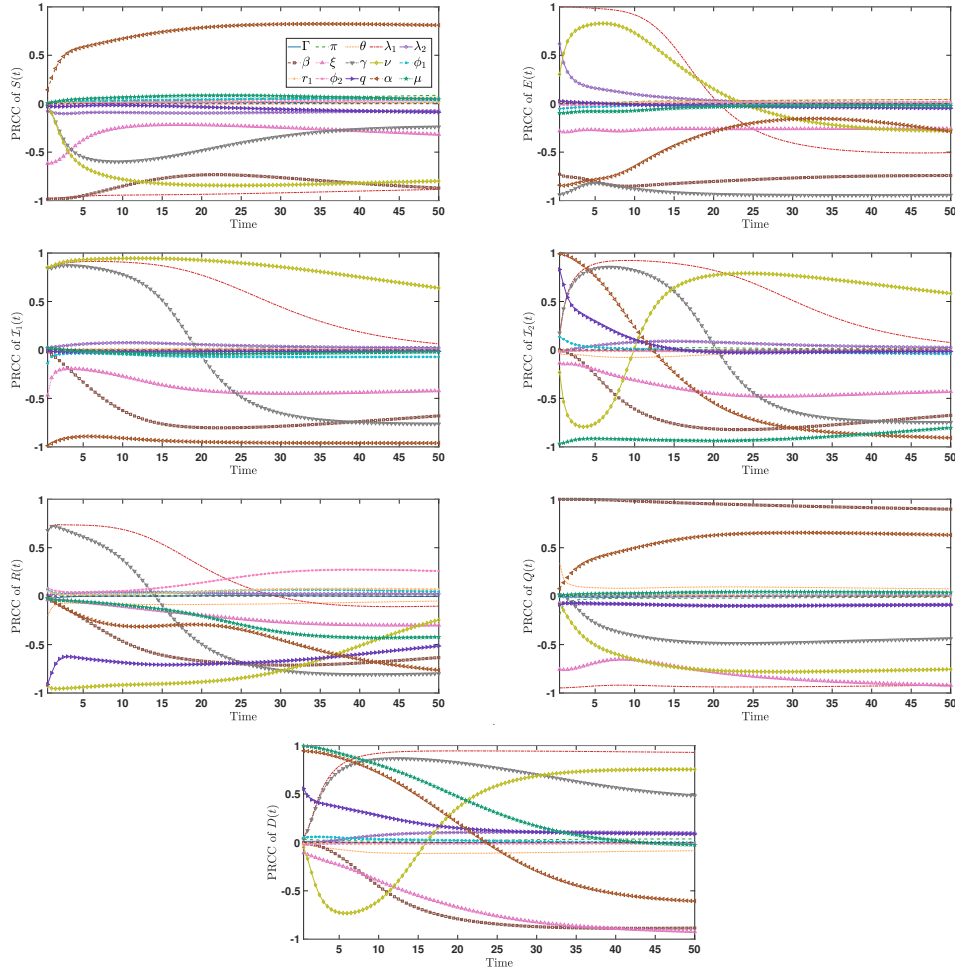


FIGURE 5.4: Time series evaluation of the global sensitivity description of proposed model (5.1) to represent the nonlinear correlation of all the biological parameters having specific interval with $\pm 50\%$ from their baseline values in Table 5.1 with the compartments $S(t)$, $E(t)$, $I_1(t)$, $I_2(t)$, $R(t)$, $Q(t)$, $D(t)$ row wise from left to right respectively. The simulation was done with sample size $N = 10000$ and time interval $t \in [0, 50]$ of step size $dt = 0.5$. The precise investigation of addition state $I_1(t)$ indicates that parameter $\nu, \lambda_1, \gamma, \xi, \beta, \alpha$ strongly influence the disease state of this compartment individuals while depression state $I_2(t)$ effectively compromised through $\alpha, \beta, \lambda_1, \gamma, \xi, \mu, q$. This statistical simulation became a strong turning point for the health care centers and policymakers to effectively implement the precaution measures through media awareness, increasing physical therapy centers as first aid.

considering biological parameters defined in Table 5.1. The results interpretation, defined as PRCC values having magnitude +1, confirms the strong positive impact of input parameters with output model equations while having a magnitude -1 express the strong negative influence, and those with magnitude 0 negligibly affect the outputs. The biological significance is precisely defined through the expression $p\text{-values} < 0.05$, which indicates that statistically a parameter is significant if its corresponding $p\text{-value}$ lies within the above constraint. Fig 5.3 displays the sensitivity analysis of the threshold $\mathbb{R}_0$ given in (5.4) of model (5.1) to describe the monotonic behavior of the proposed dynamics disease state. we choose sample size $N = 10000$ with time interval $t \in [0, 1000]$ and assuming step size $dt = 1$. Effectively analyzing the complexity of the behavior dynamics, we consider the time point of interest 500. For Latin Hypercube Sampling, we specified the interval with $\pm 50\%$ for all parameters from their baselines defined in Table 5.1. The results describe that parameter $\lambda_1, \beta, \xi, \nu, \alpha$ has a strong influence on $\mathbb{R}_0$ for long-term behavior regardless of the magnitude direction. This indicates that for taking precautionary measures and controlling the persistence of the disease outbreak, one must effectively manage the influential parameters. The monotonic relation of the biological parameters with the compartments of the behavioral dynamics is shown in Fig 5.4. This time series visualization defines the long-term evaluation of model state variables' behavior row-wise from left to right, describing all the parameters given in Table 5.1 with initial data briefly written in Sec 5.4.1. For the simulation, we take the sample size $N = 10000$ and time interval $[0, 50]$ with step size $dt = 0.5$. The addiction and depression states of individuals are effectively influenced by the excessive use of social media, particularly increasing the depression dynamic with the connection of susceptibility and pre-pressure from the media. The PRCC findings highlight how crucial it is for legislators to implement several types of measures to address social media addiction, which is correlated with depression. It also emphasizes the importance of taking preliminary measures like social awareness, physical therapies, and affordable treatment to control and lower the fatality rate associated with these physiological states.

5.6 Formulation of optimal control model

In the model, we observe that social media addiction can easily become a source of depression, especially for adults, which affects mental health badly. To minimize the negative impact of social media addiction and depression on society, government prevention measures are essential. Thanks to optimal control theory, that applied to effectively reduce the individuals who face addiction and depression due to social media use. These affected individuals may be precisely targeted and diminished by creating time-dependent measures like treatment plans and awareness campaigns. This method assists in determining the most effective means of limiting the impact and spread of these conditions while taking execution and financial limitations.

We introduce the following time-dependent control variables to this dynamics:

- $u_1(t)$: Awareness/Education through social media services, promoting a preventive lifestyle via social sports activities, exercise, reducing mobile screen time, and proper sleep.
- $u_2(t)$: Treatment boost by affordable rehabilitation programs and government-funded counseling services.

First control u_1 is applied to optimize the addiction and depression rate. This can be done by replacing the current λ_1 and λ_2 by $\lambda_1(1 - u_1)$ and $\lambda_2(1 - u_1)$ respectively. This made biological sense, that when the control input is zero, no effect on the addiction and depression rate. Once u_1 increases from 0 to 1, the term $(1 - u_1)$ will become smaller than 1, decreasing the transmission. Similarly second control u_2 is utilized to increase the treatment success rate by replacing the current ϕ_1 and ϕ_2 by $\phi_1 + u_2$ and $\phi_2 + u_2$ respectively. The modified system with time-dependent controls, becomes:

$$\begin{cases} \frac{dS}{dt} = \Gamma + \pi \theta R - \lambda_1(1 - u_1) \frac{I_1 S}{N} - \lambda_2(1 - u_1) \frac{I_2 S}{N} - (u_1 + \beta + \xi) S, \\ \frac{dE}{dt} = \lambda_1(1 - u_1) \frac{I_1 S}{N} + \lambda_2(1 - u_1) \frac{I_2 S}{N} - (\gamma + \xi) E, \\ \frac{dI_1}{dt} = \nu \gamma E - (\phi_1 + u_2) I_1 - (\xi + \alpha) I_1, \\ \frac{dI_2}{dt} = \alpha I_1 + (\phi_1 + u_2)(1 - r_1) I_1 + q R - (\phi_2 + u_2) I_2 - (\mu + \xi) I_2, \\ \frac{dR}{dt} = (1 - \nu) \gamma E + (\phi_1 + u_2) r_1 I_1 + (\phi_2 + u_2) I_2 - q R - (\xi + \theta) R, \\ \frac{dQ}{dt} = (u_1 + \beta) S + (1 - \pi) \theta R - \xi Q, \\ \frac{dD}{dt} = \mu I_2 - \xi D. \end{cases} \quad (5.30)$$

with initial conditions

$$\begin{aligned} S(0) &= 0, \quad E(0) = 0, \quad I_1(0) = 0, \quad I_2(0) = 0 \\ R(0) &= 0, \quad Q(0) = 0, \quad D(0) = 0. \end{aligned} \quad (5.31)$$

while

$$0 \leq u_1 \leq u_{1\max} \quad 0 \leq u_2 \leq u_{2\max} \quad (5.32)$$

The objective of this control problem is to minimize the addiction I_1 and depression I_2 populations along with cost associated with the awareness programs and treatment boost strategies within the interval $[0 \ T]$ by control variables $u_1 : [0 \ T] \rightarrow u_{1\max}$ and $u_2 : [0 \ T] \rightarrow u_{2\max}$ align with studies [173, 90, 275, 222]. The objective functional is defined as

$$J(u_1, u_2) = \min_{u_1, u_2 \in \Omega} \int_0^T \left(\kappa_1 I_1 + \kappa_2 I_2 + \frac{1}{2} (\kappa_3 u_1^2 + \kappa_4 u_2^2) \right) dt, \quad (5.33)$$

subject to system (5.30) and Ψ . The set Ω of admissible controls is defined as

$$\Omega = \left\{ (u_1(t), u_2(t)) \mid \begin{aligned} &0 \leq u_1(t) \leq u_{1\max}, \quad 0 \leq u_2(t) \leq u_{2\max}, \\ &u_1(t), u_2(t) \text{ are measurable } \forall t \in [0, T] \end{aligned} \right\} \quad (5.34)$$

The terms $\kappa_1 I_1, \kappa_2 I_2$ are the percentage of people who are addicted and depressed, respectively, to minimize them in the optimization function. The optimization function is made more balanced by the positive weight components κ_1 and κ_1 . In particular, these factors explain the relative importance of the percentage of addicted and depressed people vs the expenses of medical treatment. The goal is to save those who experience severe symptoms when weight factors κ_1 and κ_1 are high.

Conversely, a low optimum control results from the emphasis on limiting the cost of intervention policies when the weight factors κ_1 and κ_1 are low. Similarly, κ_3 and κ_4 are the positive weight constants given to the control $\kappa_3 u_1$ and $\kappa_4 u_2$ representing the marginal cost of making awareness programs and affordable treatment strategies for addicted and depressed individuals. Additionally, in line with other research, we consider the quadratic cost on the control variables $u_1(t)$ and $u_2(t)$, which is the most straightforward and popular nonlinear description of control cost in social/epidemic dynamics [55, 225, 2, 257].

Any optimal strategy needs to be mathematically validated by ensuring that the admissible control set is nonempty, bounded, and closed, and that the state equations satisfy conventional regularity conditions. The existence of the proposed optimal control model (5.30) with admissible control set Ω over the objective function (5.33) subject to (5.30) and Ψ with initial data (5.31)-(5.32) can be describe using the studies of Fleming and Rishel [115] and Carathodory's existence theorem [191] which will ensure that our control model has a unique solution under admissible control pair u_1, u_2 .

Theorem 5.10. *Given the objective functional (5.33) stated on the admissible control functions $(u_1, u_2) \in \Omega$, determined by the dynamical system (5.30) with initial conditions (5.31)-(5.32), an optimal control pair exists that minimizes the objective functional.*

Proof. By utilizing the results of [115, 191], we define the following conditions to prove the statement.

1. Since the solutions to the state equations are a prior bounded, the admissible control-state set is not empty. Each admissible control in Ω has a unique solution due to the equations' continuous nature and Lipschitz in the state variables.
2. By definition, the control set Ω is convex and closed.
3. By considering the linear dependency of the state equations on the controls u_1 and u_2 , the right-hand side of the system (5.30) may be expressed as the sum of linear functions in the control and state variables.
4. As the controls are quadratic, the integrand of the objective functional is convex concerning the control variables.
5. The integrand of the objective function is bounded below

$$\begin{aligned} \mathcal{M}(t, I_1, I_2, u_1, u_2) &= \kappa_1 I_1 + \kappa_2 I_2 + \frac{1}{2}(\kappa_3 u_1^2 + \kappa_4 u_2^2) \\ &\geq \max \{ \kappa_3, \kappa_4 \} (u_1^2 + u_2^2) \\ &\geq -g_3 \left(\sum_{j=1}^2 |u_j|^2 \right)^\eta - g_4 \end{aligned}$$

where $g_3 = \max \{ \kappa_3, \kappa_4 \}$, $\eta = 1$, $g_4 = \kappa_1 g_1 + \kappa_2 g_2$ assuring $I_1 \geq -g_1, I_2 \geq -g_2$.

Finally, these results ensure that there exists an optimal control pair $(u_1, u_2) \in \Omega$ that minimizes the objective functional $J(u_1, u_2)$. $\square$

Since this is a constrained optimization problem, we apply Pontryagin's maximum principle. This technique turns the control model dynamics (5.30) and objective function (5.33) into a new problem where we aim to minimize the Hamiltonian

at each point of time using the desired control variables u_1 and u_2 . Here, first, we derive the Hamiltonian function. After that, the adjoint variables system is created. Finally, we define the optimal control variable values. We establish the adjoint system, transversality criteria, and evaluation of optimum controls of the proposed control model (5.30) with the following result.

Theorem 5.11. *For a given optimal control pair (u_1, u_2) that yields the optimal state solution $S^*, E^*, I_1^*, I_2^*, R^*, Q^*$ and D^* which minimizing the objection functional $J(u_1, u_2)$ then there exist a set of adjoint variable $\lambda_i, i \in \{S, E, I_1, I_2, R, Q, D\}$ satisfy:*

$$\begin{aligned}
\frac{d\lambda_S}{dt} &= -\frac{\partial H}{\partial S} = -\left\{ \lambda_Q(u_1 + \beta) - \lambda_S(u_1 + \beta + \xi) - \lambda_S \left(I_1 \lambda_1(1 - u_1) \right) \left(\frac{N - S}{N^2} \right) \right. \\
&\quad \left. - \lambda_S \left(I_2 \lambda_2(1 - u_1) \right) \left(\frac{N - S}{N^2} \right) + \lambda_E \left(I_1 \lambda_1(1 - u_1) \right) \left(\frac{N - S}{N^2} \right) \right. \\
&\quad \left. + \lambda_E \left(I_2 \lambda_2(1 - u_1) \right) \left(\frac{N - S}{N^2} \right) \right\} \\
\frac{d\lambda_E}{dt} &= -\frac{\partial H}{\partial E} = -\left\{ \lambda_{I_1} \gamma v - \gamma \lambda_R(v - 1) - \lambda_E(\gamma + \xi) \right\} \\
\frac{d\lambda_{I_1}}{dt} &= -\frac{\partial H}{\partial I_1} = -\left\{ \kappa_1 + \lambda_{I_2}(\alpha + (\phi_1 + u_2)(1 - r_1)) - \lambda_{I_1}(\alpha + (\phi_1 + u_2) + \xi) \right. \\
&\quad \left. + S \lambda_1(1 - u_1) \lambda_E \left(\frac{N - I_1}{N^2} \right) - S \lambda_1(1 - u_1) \lambda_S \left(\frac{N - I_1}{N^2} \right) \right. \\
&\quad \left. + \lambda_R(\phi_1 + u_2)r_1 \right\} \\
\frac{d\lambda_{I_2}}{dt} &= -\frac{\partial H}{\partial I_2} = -\left\{ \kappa_2 + \lambda_D \mu + \lambda_R(\phi_2 + u_2) - \lambda_{I_2}(\mu + (\phi_2 + u_2) + \xi) \right. \\
&\quad \left. + S \lambda_2(1 - u_1) \lambda_E \left(\frac{N - I_2}{N^2} \right) - S \lambda_2(1 - u_1) \lambda_S \left(\frac{N - I_2}{N^2} \right) \right\} \\
\frac{d\lambda_R}{dt} &= -\frac{\partial H}{\partial R} = -\left\{ \lambda_{I_2} q - \lambda_R(q + \theta + \xi) + \lambda_Q \theta(1 - \pi) + \lambda_S \pi \theta \right\} \\
\frac{d\lambda_Q}{dt} &= -\frac{\partial H}{\partial Q} = -\left\{ -\lambda_Q \xi \right\} \\
\frac{d\lambda_D}{dt} &= -\frac{\partial H}{\partial D} = -\left\{ -\lambda_D \xi \right\}
\end{aligned} \tag{5.35}$$

with transversality conditions as the objective function, they do not have a terminal cost term

$$\lambda_i(T) = 0 \text{ for } i \in \{S, E, I_1, I_2, R, Q, D\}.$$

Furthermore, the optimal control functions u_1^*, u_2^* characterize as

$$\begin{aligned}
u_1^* &= \max \left\{ 0, \min \left\{ \frac{(\lambda_S - \lambda_Q)S/N + (\lambda_E - \lambda_S)S(\lambda_1 I_1 + \lambda_2 I_2)}{N \kappa_3} \right\} \right\} \\
u_2^* &= \max \left\{ 0, \min \left\{ \frac{(\lambda_{I_2} - \lambda_R)(I_2 + I_1 r_1) + (\lambda_{I_1} - \lambda_{I_2})I_1}{\kappa_4} \right\} \right\}
\end{aligned}$$

Proof. Follows the Pontryagin's maximum principle [237], the Hamiltonian $\mathcal{H}$ is give as

$$\begin{aligned} \mathcal{H} = & \kappa_1 I_1 + \kappa_2 I_2 + \frac{1}{2}(\kappa_3 u_1^2 + \kappa_4 u_2^2) + \lambda_S \frac{dS}{dt} + \lambda_E \frac{dE}{dt} + \lambda_{I_1} \frac{dI_1}{dt} + \lambda_{I_2} \frac{dI_2}{dt} \\ & + \lambda_R \frac{dR}{dt} + \lambda_Q \frac{dQ}{dt} + \lambda_D \frac{dD}{dt}. \end{aligned} \quad (5.36)$$

We differentiate the Hamiltonian $\mathcal{H}$ with each adjoin variable λ_i where $i = S, E, I_1, I_2, R, Q, D$ to get the adjoin system as

$$\begin{aligned} \frac{d\lambda_S}{dt} &= -\frac{\partial \mathcal{H}}{\partial S}, & \frac{d\lambda_E}{dt} &= -\frac{\partial \mathcal{H}}{\partial E}, & \frac{d\lambda_{I_1}}{dt} &= -\frac{\partial \mathcal{H}}{\partial I_1}, & \frac{d\lambda_{I_2}}{dt} &= -\frac{\partial \mathcal{H}}{\partial I_2}, \\ \frac{d\lambda_R}{dt} &= -\frac{\partial \mathcal{H}}{\partial R}, & \frac{d\lambda_Q}{dt} &= -\frac{\partial \mathcal{H}}{\partial Q}, & \frac{d\lambda_D}{dt} &= -\frac{\partial \mathcal{H}}{\partial D}. \end{aligned}$$

Differentiating the Hamiltonian $\mathcal{H}$ with respect to control variables and setting them equal to zero, we get

$$\begin{aligned} \frac{\partial \mathcal{H}}{\partial u_1} &= \kappa_3 u_1 - (\lambda_S - \lambda_Q)S - \lambda_E \frac{S(\lambda_1 I_1 + \lambda_2 I_2)}{N} + \lambda_S \frac{S(\lambda_1 I_1 + \lambda_2 I_2)}{N}, \\ u_1 &= \frac{(\lambda_S - \lambda_Q)S/N + (\lambda_E - \lambda_S) S(\lambda_1 I_1 + \lambda_2 I_2)}{\kappa_3 N}. \end{aligned} \quad (5.37)$$

$$\begin{aligned} \frac{\partial \mathcal{H}}{\partial u_2} &= \kappa_4 u_2 - \lambda_{I_1} I_1 - \lambda_{I_2} (I_2 + I_1 (r_1 - 1)) + \lambda_R (I_2 + I_1 r_1) \\ u_2 &= \frac{\lambda_{I_1} I_1 + \lambda_{I_2} (I_2 + I_1 (r_1 - 1)) - \lambda_R (I_2 + I_1 r_1)}{\kappa_4} \end{aligned} \quad (5.38)$$

Using the bond on the control variable u_1 and u_2 , we get

$$u_1^* = \begin{cases} 0 & \text{if } \frac{\partial \mathcal{H}}{\partial u_1} < 0 \\ \frac{(\lambda_S - \lambda_Q)S/N + (\lambda_E - \lambda_S) S(\lambda_1 I_1 + \lambda_2 I_2)}{N \kappa_3} & \text{if } 0 < \frac{\partial \mathcal{H}}{\partial u_1} < u_{1 \max} \\ u_{1 \max} & \text{if } \frac{\partial \mathcal{H}}{\partial u_1} > u_{1 \max} \end{cases} \quad (5.39)$$

$$u_2^* = \begin{cases} 0 & \text{if } \frac{\partial \mathcal{H}}{\partial u_2} < 0 \\ \frac{(\lambda_{I_2} - \lambda_R) (I_2 + I_1 r_1) + (\lambda_{I_1} - \lambda_{I_2}) I_1}{\kappa_4} & \text{if } 0 < \frac{\partial \mathcal{H}}{\partial u_2} < u_{2 \max} \\ u_{2 \max} & \text{if } \frac{\partial \mathcal{H}}{\partial u_2} > u_{2 \max} \end{cases} \quad (5.40)$$

leads to a compact form of terminal conditions and ended proof. $\square$

By integrating the defined control set within initial and transversal conditions, the optimality system is produced from the state system, and the adjoint variable system describes the optimal control system of model (5.1)

State Equations :

$$\begin{cases}
\frac{dS}{dt} = \Gamma + \pi \theta R - \lambda_1(1 - u_1^*) \frac{I_1 S}{N} - \lambda_2(1 - u_1^*) \frac{I_2 S}{N} - (u_1^* + \beta + \xi) S, \\
\frac{dE}{dt} = \lambda_1(1 - u_1^*) \frac{I_1 S}{N} + \lambda_2(1 - u_1^*) \frac{I_2 S}{N} - (\gamma + \xi) E, \\
\frac{dI_1}{dt} = \nu \gamma E - (\phi_1 + u_2^*) I_1 - (\xi + \alpha) I_1, \\
\frac{dI_2}{dt} = \alpha I_1 + (\phi_1 + u_2^*)(1 - r_1) I_1 + q R - (\phi_2 + u_2^*) I_2 - (\mu + \xi) I_2, \\
\frac{dR}{dt} = (1 - \nu) \gamma E + (\phi_1 + u_2^*) r_1 I_1 + (\phi_2 + u_2^*) I_2 - q R - (\xi + \theta) R, \\
\frac{dQ}{dt} = (u_1^* + \beta) S + (1 - \pi) \theta R - \xi Q, \\
\frac{dD}{dt} = \mu I_2 - \xi D, \\
S(0) = 0, E(0) = 0, I_1(0) = 0, I_2(0) = 0, \\
R(0) = 0, Q(0) = 0, D(0) = 0.
\end{cases} \quad (5.41)$$

Adjoint Equations :

$$\begin{cases}
\frac{d\lambda_S}{dt} = -\lambda_Q(u_1^* + \beta) + \lambda_S((u_1^* + \beta) + \xi) - (\lambda_E - \lambda_S)(I_1 \lambda_1(1 - u_1^*) \\
\quad + I_2 \lambda_2(1 - u_1^*)) \left(\frac{E + I_1 + I_2 + R + Q + D}{N^2} \right) \\
\frac{d\lambda_E}{dt} = -\lambda_{I_1} \gamma \nu + \gamma \lambda_R (\nu - 1) + \lambda_E (\gamma + \xi) \\
\frac{d\lambda_{I_1}}{dt} = -\kappa_1 - (\lambda_{I_2} - \lambda_{I_1})(\alpha + (\phi_1 + u_2^*)) - (\lambda_R - \lambda_{I_2})(\phi_1 + u_2^*) r_1 \\
\quad - (\lambda_E - \lambda_S) S \lambda_1(1 - u_1^*) \left(\frac{S + E + I_2 + R + Q + D}{N^2} \right) + \lambda_{I_1} \xi \\
\frac{d\lambda_{I_2}}{dt} = -\kappa_2 - (\lambda_D - \lambda_{I_2}) \mu - (\lambda_R - \lambda_{I_2})(\phi_2 + u_2^*) - (\lambda_E \\
\quad - \lambda_S) S \lambda_2(1 - u_1^*) \left(\frac{S + E + I_1 + R + Q + D}{N^2} \right) + \lambda_{I_2} \xi \\
\frac{d\lambda_R}{dt} = -(\lambda_{I_2} - \lambda_R) q - (\lambda_Q - \lambda_R) \theta - (\lambda_S - \lambda_Q) \pi \theta + \lambda_R \xi \\
\frac{d\lambda_Q}{dt} = \lambda_Q \xi \\
\frac{d\lambda_D}{dt} = \lambda_D \xi \\
\lambda_i(T) = 0, \text{ where } i = \{S, E, I_1, I_2, R, Q, D\}
\end{cases} \quad (5.42)$$

5.7 Numerical analysis

In this section, we describe a comprehensive framework for the evaluation of behavior dynamics in coupled social media addiction and depression (5.1), their parameter identification using modern machine learning tools, and possible prevention strategies using an optimal control problem (5.41)-(5.42). The numerical investigation is defined using the actual depression data [204] and a synthetic simulation. The estimation of parameters is performed with the minimization problem proposed in Sec 5.4.1. As the actual data is limited, validation of the parameters chosen from sources

is captured using the literature learning process through Feed Forward Neural Network and Physics Informed Neural Network. The FNN-based strategy initiates the simulation by training a network to estimate the target parameters over the actual data trajectories with randomness to observe the dynamic relation between the estimated parameters and state variables whereas the PINN paradigm naturally blends physical consistency with data learning, yielding more reliable and precise parameter identification. The data-driven techniques formulated in Table 1-2. Mathematical analysis of the model (5.1) indicates that even with the threshold values less than one, the addition and depression dynamics will persist due to the geographical factors, relapse phenomena, and lack of medical facilities. The proposed optimal control strategies (5.41)-(5.42) illustrate the effective use of media awareness programs and increasing the initial medical facility can control the behavioral dynamics. The simulation of the optimal control model was performed using the most famous technique, called the forward and backward sweep method, with Runge-Kutta of order four. The algorithm of this technique is defined in Table 3.

Algorithm 1 Parameter Identification of model (5.1) with FNN

Input: Define the Initial data y_0 , actual parameters θ_{true} , time interval and number of training samples

Output: Estimated parameters $\bar{\theta}$ and predicted model compartments Y_{pred}

Synthetic data generation:

Integrate $\dot{Y}(t) = \mathcal{F}(Y(t), \theta_{\text{true}})$ applying RK4 to get $\mathcal{Y}_{\text{actual}}(t)$ and add Gaussian noise for $\mathcal{Y}_{\text{synth}}$

Training data formation:

for $i = 1 : N_s$ **do**

Randomly perturb δ_i the parameters like $\tilde{\theta}^{(i)} = \theta_{\text{actual}} \odot (1 + \delta_i)$,

Simulate $\mathcal{Y}^{(i)}(t_j) = \text{RK4Solver}(y_0, \tilde{\theta}^{(i)}, t_j)$

for t_j **do**

Define input-output pairs: $\mathcal{X}_{ij} = [\mathcal{Y}^{(i)}(t_j), t_j]$, $\mathcal{Y}_{ij} = \tilde{\theta}^{(i)}$

end for

end for

Normalize training inputs and outputs to $[-1, 1]$

FNN structure:

Write feedforward network $\mathcal{N}_\omega$ with 3 hidden layers [10–10–10], satlins activation and linear output. Train network with Levenberg–Marquardt algorithm and stop when epochs = 200 or gradient $< 10^{-7}$, using 80/10/10 train/val/test split

Parameter Identification:

Define network input from synthetic data: $\mathcal{X}_{\text{synth}} = [\mathcal{Y}_{\text{synth}}, t_{\text{synth}}]$

Forecast parameters at each time: $\hat{\theta}(t_j) = \mathcal{N}_\omega(X_{\text{synth},j})$ and compute mean parameter estimates $\bar{\theta}$ to simulate predicted state vectors: $\mathcal{Y}_{\text{pred}}(t) = \text{RK4Solver}(y_0, \bar{\theta}, t)$

5.7.1 Evaluation of data driven model

The model (5.1) precisely describe the overlapping of social media addiction and depression by complex structure, emphasizing how sensitive biological parameters like incidence rate of the addiction and depression state, the pressure of media over the addictions individuals leads to depression, relapse in depressed individual and treatment success ration during the behavioral dynamics which closely relate with

Algorithm 2 Parameter Identification of model (5.1) with PINN

Input: Start with given model $\mathcal{Y}'(t) = \mathbf{f}(t, y, \theta^{\text{true}})$ having initial data in time frame and true parameters θ^{true} , and ensure estimated parameters θ^*
 Simulate model using Runge-Kutta to generate reference solution. For synthetic data, add Gaussian noise to the reference solution and make the training dataset $\mathcal{D}_t = \{t_i, \mathcal{Y}_{\text{synth}}(t_i)\}_{i=0}^{N_d}$
Initialize PINN training
 Initialize neural network $\mathcal{N}_\omega : \mathbb{R} \rightarrow \mathbb{R}^m$ with multiple layers, some hidden units, network weights ω and parameter variables $\{\xi_j\}_{j=1}^k$ randomly
Adam Optimization
for $n = 1$ to I_{Adam} **do**
 Sample collocation points: $\{t_i^c\}_{i=1}^{N_c} \sim \text{Uniform}(0, T)$
 Forward pass: $\hat{\mathcal{Y}}_i = \mathcal{N}_\omega(t_i^c)$ and compute derivatives $\frac{\partial \hat{\mathcal{Y}}_i}{\partial t}$ using auto-grad
 Transform parameters: $\theta_j = \theta_j^{\text{nom}} \cdot \left[\frac{\beta_{\max} - \beta_{\min}}{2} \tanh(\xi_j) + \frac{\beta_{\max} + \beta_{\min}}{2} \right]$
 Compute loss: $\mathcal{L}_{\text{total}} = \lambda_1 \mathcal{L}_{\text{odes}} + \lambda_2 \mathcal{L}_{\text{IC}} + \lambda_3 \mathcal{L}_{\text{data}}$
 Compute gradients: $\nabla_\omega \mathcal{L}_{\text{total}}, \nabla_{\{\xi_j\}} \mathcal{L}_{\text{total}}$ and update system.
end for
L-BFGS Refinement
for $n = 1$ to I_{LBFGS} **do**
 Compute $\mathcal{L}_{\text{total}}$ and gradients
 Update network, $\{\xi_j\}$ using L-BFGS with line search
 if $\|\nabla \mathcal{L}_{\text{total}}\| < \epsilon$ **then break**
 end if
end for
Evaluation and Validation
 Compute PINN predictions: $\hat{\mathcal{Y}}(t) = \mathcal{N}_{\omega^*}(t)$ for $t \in [0, T]$
 Calculate state variables prediction error and parameter error
Output: Estimated parameters $\theta^* = \{\theta_j(\xi_j^*)\}_{j=1}^k$ and state variables prediction $\hat{\mathcal{Y}}(t)$

Algorithm 3 Forward-Backward Sweep Method using RK-4 for model (5.30)

Input: Start with state equations of model $\dot{x} = f(x, u, t)$ with initial state for control $u^{(0)}(t)$ having adjoint dynamics $\dot{\lambda} = -\frac{\partial H}{\partial x}$ and initial state $x(0)$
 Initialize iteration $k = 0$ and control $u^{(0)}(t)$
repeat
 For forward integration of state equation use RK-4 by integrate $\dot{x} = f(x, u^{(k)}, t)$ from $t = 0$ to $t = T$ to get $x^{(k)}(t)$
 For backward integration of adjoint system, use RK-4 by integrate $\dot{\lambda} = -\frac{\partial H}{\partial x}$ from $t = T$ to $t = 0$ with terminal condition $\lambda(T)$ to get $\lambda^{(k)}(t)$
 Update control using optimality condition: $u^{(k+1)}(t) = \arg \min_u H(x^{(k)}(t), u, \lambda^{(k)}(t), t)$
 Convergence check through compute $\Delta u = \|u^{(k+1)} - u^{(k)}\|_\infty$
 $k \leftarrow k + 1$
until $\Delta u < \epsilon$ or $k \geq K_{\max}$
Output: Optimal state trajectory $x^*(t) = x^{(k)}(t)$, control $u^*(t) = u^{(k)}(t)$, adjoint state trajectory $\lambda^*(t) = \lambda^{(k)}(t)$

the suicide rate. Key parameters such as λ_1 , λ_2 , α , q , and ϕ_2 , whose descriptions are given in Table 5.1 enhance the model reliability, estimated from actual data. Remaining parameters sourced from literature significantly impact the treatment efficiency and relapse prediction, thereby affecting the suicide rate in depressed individuals. The model's biological feasibility and the ambiguity concerning these parameters are essential for its validation, establishing it as a critical tool for policymakers dealing with behavioral dynamics related to internet, social media, and gaming addictions, as well as their potential relation to depression within communities. Even with such limited or partially absent observations, neural networks may robustly estimate parameters by extracting latent patterns from sparse, noisy, or incomplete data. We are using Feed Forward Neural Network and Physics Informed Neural Network to estimate the parameters from the random synthetic data collected from the reference solution of model (5.1) using the Runge Kutta method.

In the initial setting, the formation of the FNN for model (5.1) is described, utilizing the fourth-order Runge-Kutta scheme. Key parameters such as λ_1 , λ_2 , α , q , and ϕ_2 are fixed, while other parameters $\pi, \theta, \beta, \xi, \gamma, \nu, \phi_1, r_1$ are selected for estimation based on literature baselines. Empirical realism is achieved by including comparative Gaussian noise into the reference solution. We choose sample size $N = 1000$ to generate an effective training parameters set, with each set being equally disturbed within $\pm 25\%$ of their actual values, keeping other parameters fixed. At each time point, different trajectories of the state variables were generated with the RK-4 solver for synthetic variability. These trajectories are taken as distinct training samples, enabling the network to learn the mapping from stochastic state variables and time to latent parameter values. The network consists of three hidden layers with ten neurons in each layer. The training is defined using the Levenberg-Marquardt back propagation technique with 200 epochs. For each synthetic time point, the trained FNN produces a separate mean estimate for each selected parameter, which is used to predict the compartments' trajectories of the model (5.1). Fig 5.5 visualizes the prediction of the model with the estimated parameters using the proposed FNN data-driven technique. Synthetic data shows the error between prediction and observed data, while FNN prediction defines the error between the reference and the prediction solution. The predicted results show the validity of the complex parameters taken from literature, indicating that the model behavior is biologically meaningful. The comparison of the estimated and true parameters over time with epochs is shown in Fig 5.6. The technique provides for the parallel pointwise estimate of selected parameters and allows for the more precise forecast of behavioral dynamics. This configuration is intended to investigate simple neural network-based parameter estimation while quantifying errors in state vectors and parameter estimations.

In the second setting, we define the PINN-based strategy to predict the state variables of model (5.1) with the estimated parameters. Our approach initiates by solving the model compartments with the Runge Kutta technique for generating a reference solution with actual parameters, which serve as ground truth for validation. Synthetic data is being defined using Gaussian noise in the reference solution, mimicking the stochastic behavior in realistic epidemic models. The central component of our approach is a deep neural network to learn unidentified model parameters through a constrained transformation approach while approximating the temporal progression of model state variables. PINN formulation embedding the governing differential equation of the model explicitly into the loss function through automatic differentiation. This strategy reduces the demand for complete knowledge of all parameters by enabling the neural network to naturally follow the physical limits related to the epidemic model, which is typically essential for FNN approaches. The

deep neural network structure consists of an input layer with three hidden layers, each with 64 neurons and ReLU activation after each linear transformation. The output layer is defined using element-wise absolute operation to ensure non-negative predictions, making the solution physically meaningful. This descriptive structure is highly computational and has enough capacity to mimic the dynamics of the system. The training procedure employs a hybrid optimization approach, with an initial Adam phase for quick parameter search and L-BFGS refinement for exact matching to optimum solutions. Fig 5.7 illustrates the perdition of model (5.1) compartments row-wise from left to right with the estimated parameters. The graphics display the biological meaningful state variable prediction with reliable parameter inference, even with uncertain data. The comparison of estimated parameters with actual values is presented in Fig 5.8 over selected epochs for the training network. Complete analysis of the proposed data-driven strategies output is given in Table 5.2

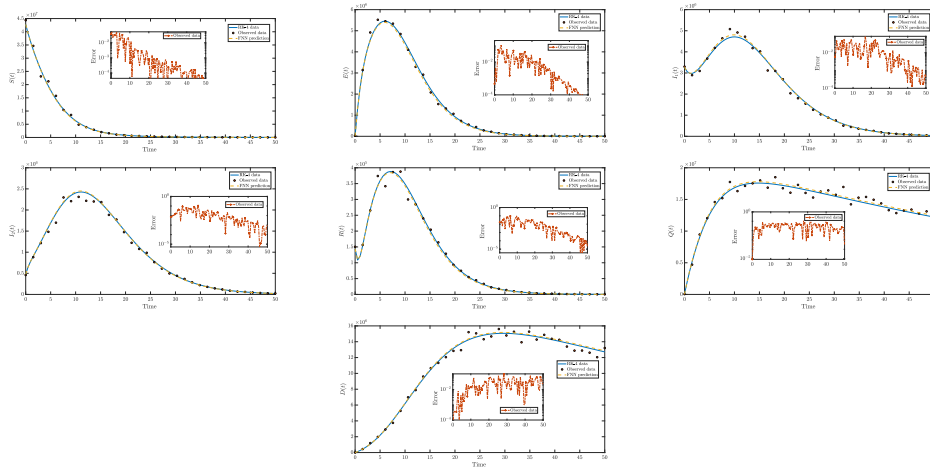


FIGURE 5.5: Evaluation of state variables row-wise from left to right of the behavior model (5.1) with estimated parameters and error estimation. The simulation done using the FNN base neural network structure consists of three layers, with each layer having 10 neurons. The hidden activation is performed through linear saturating satlins, while for output activation we use a linear function purelin. The network was trained using Levenberg Marquardt back propagation `trainlm` with epochs 200 and a data division ratio of train: 80%, val: 10%, test: 10%. The reference solution was generated using the RK-4 method with $t \in [0, 50]$ and a step size of 0.5. The forecasting trajectories of each state variable are biologically meaningful and collectively capture the monotonic relation between social media addiction and depression in individuals.

5.7.2 Transition flow and media influence

The model (5.1) precisely describes the partial bilateral relation of social media addiction and depression with respective incidence rates λ_1 and λ_2 . Their mutual interaction is primarily governed by infected individuals' movement between these two states. This relation defines the disease persistence phases during the evaluation of behavioral dynamics. A significant decrease in addiction and depression dynamics comes with effective medical precautions and positive outcomes of media awareness programs. We explicitly investigate the complex relation of these behavioral states in different settings using the higher-order Runge-Kutta method.

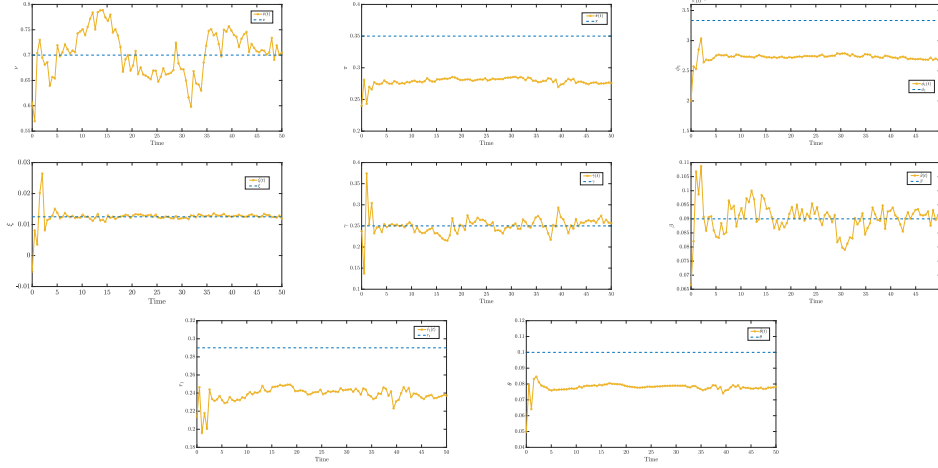


FIGURE 5.6: Comparison of the estimated parameters ν , π , ϕ_1 , ξ , γ , β , r_1 , θ , and corresponding true values row-wise from left to right over time with FNN over 200 epochs. The reference solution was generated using the RK-4 method with $t \in [0, 50]$ and a step size of 0.5.

TABLE 5.2: Representation of estimated parameters with FNN & PINN.

Parameter	True Value	FNN Estimate	FNN Error	PINN Estimate	PINN Error
π	0.350 000	0.278 504	0.071 496	0.004 704	0.375 296
θ	0.100 000	0.077 790	0.022 210	0.171 770	0.071 770
β	0.090 000	0.090 299	0.000 299	0.086 375	0.003 625
ξ	0.012 500	0.012 482	0.000 018	0.011 910	0.000 590
γ	0.250 000	0.250 599	0.000 599	0.254 267	0.004 267
ν	0.700 000	0.703 807	0.003 807	0.681 782	0.018 218
ϕ_1	0.003 333	0.002 724	0.000 609	0.000 003	0.002 997
r_1	0.290 000	0.238 418	0.051 582	0.520 553	0.230 553

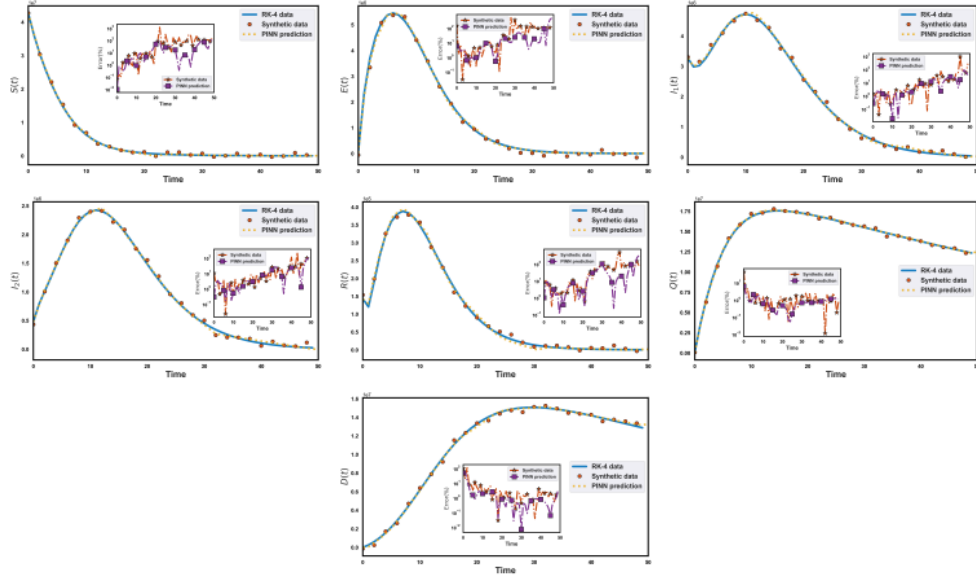


FIGURE 5.7: Evaluation of state variables row-wise from left to right of the behavior model (5.1) with estimated parameters and error estimation. The simulation defined with PINN-based deep neural network structure consists of one input layer and three hidden layers, with each layer having 64 neurons with ReLU (Rectified Linear Unit) activation functions to describe the monotonic behavior of the model. The reference solution was generated using the RK-4 method with $t \in [0, 50]$ and a step size of 0.5. The forecasting trajectories of each state variable are biologically meaningful and collectively capture the complex interaction between social media addiction and depression in individuals.

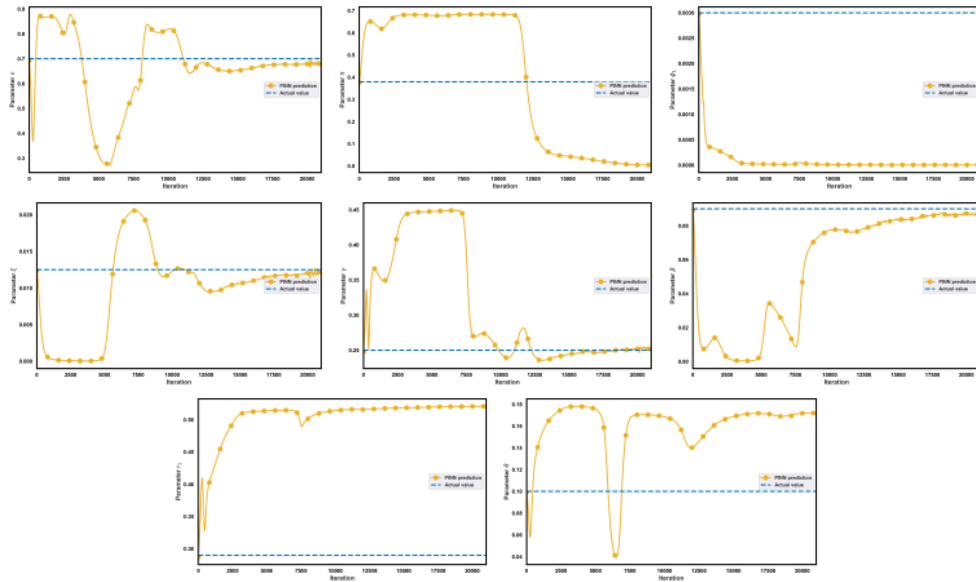


FIGURE 5.8: Comparison of the estimated parameters ν , π , ϕ_1 , ξ , γ , β , r_1 , θ , and corresponding true values row-wise from left to right over time with FNN over 20000 epochs. The reference solution was generated using the RK-4 method with $t \in [0, 50]$ and a step size of 0.5.

In Setting 1, we describe the evaluation of model (5.1) using the actual data and estimated parameters through data-driven models defined in Sec 5.7.1. This qualitative comparison describes the consistency of mathematical output with proposed data-driven strategies, making the model's reliability and validation more efficient. Fig 5.9 shows the Setting 1 evaluation, by presenting the comparison of all compartments' predictions through actual data, FNN estimated data, and PINN estimated data. The total infection emerges when an infected individual comes in contact with a susceptible individual, due to which individual showing infection symptoms during the incubation period despite self-precautions. Generally, individuals leave the treatment process after some improvements, increase the relapse may lead to severe depression state causing suicide attempts.

Better analysis of behavioral dynamics defined by measures not merely the infected cases, but also the date of the epidemic's peak and overall severity across time. Therefore, evaluation of epidemic factors like total peak infection, time to peaks, and cumulative infection signifies the model's predictability. Setting 2-3 precisely explains these epidemic concepts in behavioral dynamics, which provide complementary information about the mutual interaction of addiction and depression dynamics. The highest percentage of individuals who were actively infected across the simulation period justifies the total peak infection. As model (5.1) contains two infected compartments $I_1(t)$ as addicted individuals, while $I_2(t)$ is depression individuals. Thus total infection $I(t) = I_1(t) + I_2(t)$ leads to formulation of peak size of infection as

$$I_{\text{peak}} = \max_{t \in [0, T]} [I_1(t) + I_2(t)],$$

quantify the maximum instantaneous epidemic burden on the population. This quantity indicates the time to the peak of behavior dynamics, describing the growth of epidemic severity by indicating the moments where the total epidemic infection approaches its maximum limit. Its mathematical description is given as

$$t_{\text{peak}} = \arg \max_{t \in [0, T]} [I_1(t) + I_2(t)],$$

assessing key information about the initial public health response. The cumulative infections capture the overall magnitude of the infection spread throughout the outbreak, written as

$$I_{\text{cuml}} = \int_0^T [I_1(t) + I_2(t)] dt.$$

Fig 5.10 display the Setting 2 scenario which examine the model (5.1) behavior trough the complex relation of incidence rate of addiction and depression with receptive treatment strategies by evaluating peak size and moment of peak level of the addiction $I_1(t)$ individuals in first row and depression $I_2(t)$ individuals in second row in any community. The first column expresses that the addiction state grows instantly due to large initial cases decreasing the treatment effectiveness, while depressed individuals can be managed by effective treatment and decreasing relapse. The time to peak visualization shows the explosive start of addiction dynamics with $\phi < 1.5 \times 10^{-3}$. This means the individual addiction rate is high and hospitals are overloaded, while with $\phi > 1.5 \times 10^{-3}$, the peak time delay and initial health steps become significant to increase the successful recovery of addicted individuals. The depression state show critical transition occurrence at $\phi = 6 \times 10^{-3}$ differentiate the rapid depression growth $\phi < 6 \times 10^{-3}$ from delay peak $\phi > 6 \times 10^{-3}$. Setting 3 justifies the impact of pre-pressure and self precautions over the total behavior

dynamics through defining the epidemic factors with respect to biological parameters ν, q, α, β defined in Table 5.1. Fig 5.11 precisely evaluates the time to peak (first row), peak infection (second row), and cumulative infections (third row) over estimated parameters ν, q, α, β by perturbing $\pm 50\%$ from their baselines. The simulation results present that the pre-pressure α of media rapidly approaches the epidemic outbreak as compared to relapse individuals q . With passing time, parameters q, ν maximum infected the individuals, indicating that addiction and depression states are marginally affected by these factors. The cumulative infection leads to the conclusion that pre pressure of media impact decreases while the relapse phenomena serve the disease outbreak over time.

Basic reproduction number $\mathbb{R}_0$ has a central role in defining the epidemic trajectory in the population. The average number of secondary infections in model (5.1) through single infected individuals is represented by the reproduction number (5.4) in the vulnerable population. The infection can grow during an outbreak when $\mathbb{R}_0$ is greater than one, which makes sense that each infected individual generates more than one new infected case. while $\mathbb{R}_0$ is less than one, show that the infection eventually dies out in the population. In Setting 4, we briefly describe the analysis of reproduction number $\mathbb{R}_0$ (5.4) with respect to addiction and depression transmission, pre-pressure of media, self-precautions to quit social media, etc. This guidance is effective the public health planning and model forecasting strategies. Fig 5.12 follows Setting 4, effectively illustrating the simulation of $\mathbb{R}_0$ with multiple pairs dependency to define the healthy and endemic phase in behavior dynamics. The simulation suggests that parameters q, ξ, γ, ν are more sensitive to $\mathbb{R}_0$, with respective parameters β, α justify the assumption that pre pressure of media over addicted individuals and the increase of relapse loop make the epidemic persistence due to suicidal cases and slow treatment process. The first stereogram plot quantifies the correlation of addiction transmission, respective treatment rate, and individual full shown addicted symptoms in the incubation period. The second captures the correlation of depression transmission, respective treatment rate, and relapse phenomena, while the third defines the relation of media pre-pressure, suicide rate, and self-reflective individuals.

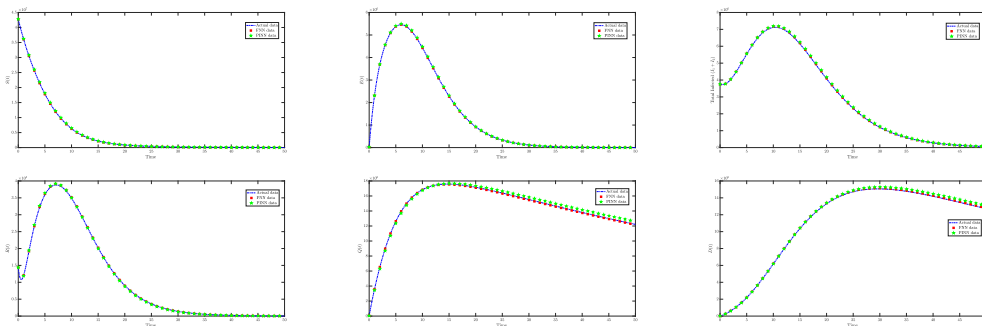


FIGURE 5.9: Setting 1: Comparison of actual data given in Table 5.1 and estimated FNN and PNN data simulated in Sec 5.7.1 over each compartment $S(t), E(t), I(t), R(t), Q(t), D(t)$ of model (5.1) row wise from left to right with initial condition written in Sec 5.4.1. The simulation perfectly describes the alignment of data-driven models with epidemic trajectories over time. The simulation run over $t \in [0, 50]$ with step size $dt = 0.05$ using higher order RK-4 method.

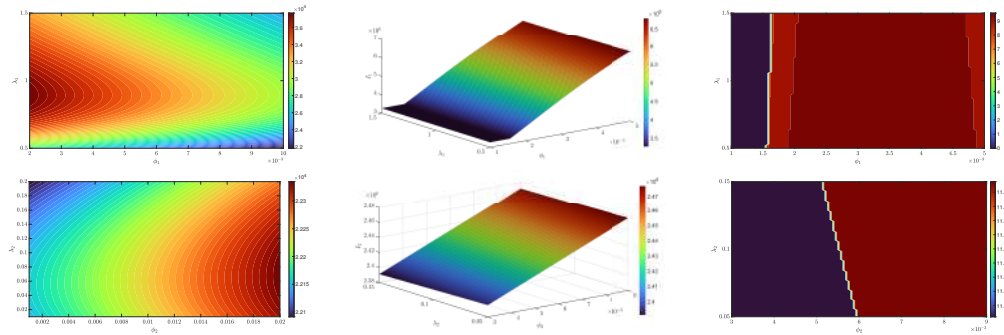


FIGURE 5.10: Setting 2: Evaluation of transition flow and forecasting of addiction and depression states on individuals with respective transmission rates and treatment process. The first row describes the addiction dynamics while the second row shows depression dynamics using model (5.1). The first column explains the variation of addiction and depression variables, peak size of addiction and depression defined in the second column, and time to peak with respect to parameter pairs $\lambda_1, \phi_1, \lambda_2, \phi_2$ of addiction and depression given in the third column. For simulation, we assume $t \in [0, 50]$ and $dt = 0.1$.

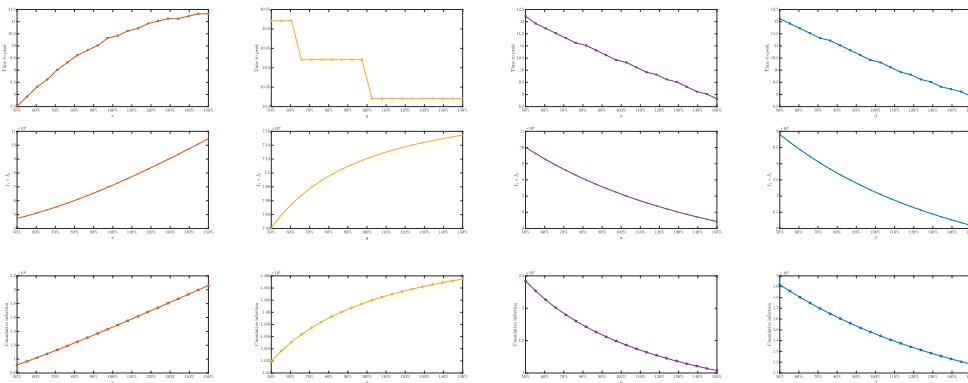


FIGURE 5.11: Setting 3: Simulation of total infection time to peak (first row), total infection peak size (second row) and cumulative infection (third row) with respective parameters cases ν (first column), q (second column), α (third column) and β (fourth column) precisely describe in Table 5.1. The evaluation is defined by perturbing $\pm 50\%$ each parameter from baseline and taking $t \in [0, 50]$ with $dt = 1/10$.

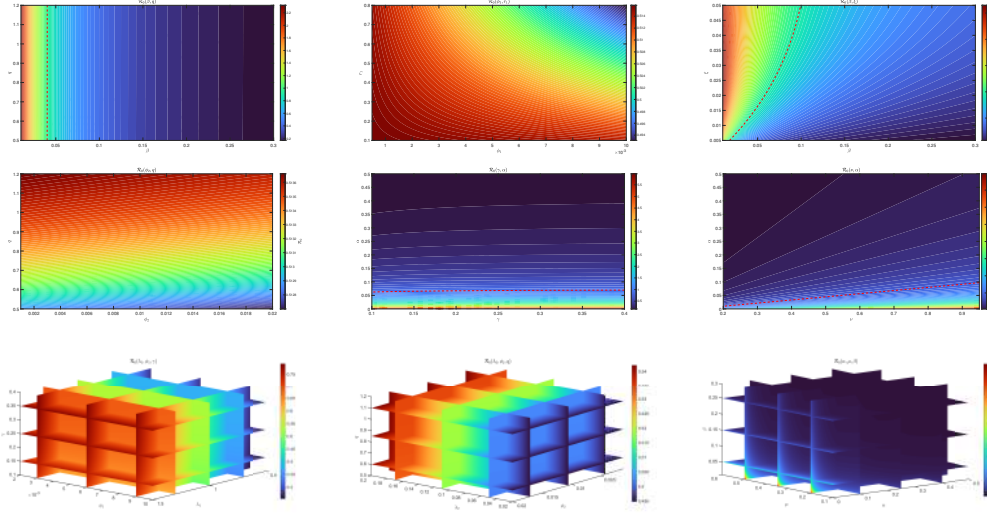


FIGURE 5.12: Setting 4: Impact of basic reproduction number R_0 (5.4) of model (5.1) on the behavior epidemic spread. Analysis of biological parameters pairs (β, q) , (ϕ_1, r_1) , (β, ξ) , (ϕ_2, q) , (γ, α) , (v, α) over R_0 given in row one and two from left to right respectively. The dotted red line defines the value $R_0 = 1$. In third row, we presents the 3D visualization of pairs $(\lambda_1, \phi_1, \gamma)$, (λ_2, ϕ_2, q) and (α, μ, β) over R_0 . All parameter descriptions are given in Table 5.1.

5.7.3 Simulation of control model

In this section, we focus on capturing the analysis of the optimal control strategies derived from the behavioral dynamics in social media addiction and depression. The optimality system consists of state system (5.41) and adjoint system (5.42) with terminal conditions of the control pair (u_1^*, u_2^*) . The systems (5.41)-(5.42) were solved using the Forward-Backward Sweep Method defined in Table 3 with initial data given in Sec 5.6. The simulation was performed over a prescribed control horizon with estimated parameters from actual data given in Table 5.1 to evaluate various state variables of model (5.30) under optimal intervention strategies. The controls are refined using a convex combination of the prior controls and the data obtained through the characterisation procedure. The systems (5.41)-(5.42) solutions are repeated by the updated controls, and iteration continues until the desired convergence limit is met. To precisely examine the optimal control model, we consider multiple Tests with different strategies given as follows with social cost $\kappa_1 = 1, \kappa_2 = 2$ and weighted parameters $\kappa_3 = \kappa_4 = 10$.

Test 1 presents the educational programs, awareness campaigns, and designs digital literacy strategies to prevent the individual from the negative impact of social media ($u_1 \neq 0, u_2 = 0$). These initiatives reduce the susceptible individuals' exposure to addicted and depressed contacts. Fig 5.13 illustrates the strategy with setting $u_2 = 0$ while the objective function (5.33) optimizes only with $u_1 \neq 0$, showing a clear reduction of exposed, addictive, depressed, and suicide attempt populations and an increase in the quitter individuals as compared to the no control solution. The recovered population decreases due to relapse in the depressed population, making the setting partially identical.

Test 2 describes the counseling services, therapeutic interventions, and rehabilitation programs to increase the opportunity of successful and reliable treatments

for new individuals and those who already suffer from addiction and depression ($u_1 = 0, u_2 \neq 0$). These treatment strategies urge the infected individuals to freely participate in government health programs, effectively increase the recovery process, and decrease the suicide rate. Fig 5.14 displays this scenario with setting $u_2 \neq 0$ while $u_1 = 0$, evident that addicted and depressed individuals effectively decrease and subsequently recover, while quitter individuals show a sudden jump in solution as compared to the situation with no control. Due to the effective treatment control, the suicide rate has decreased significantly. Hence, this strategy seems effective in controlling the spread of behavioral dynamics, making a suitable choice to manage the burden of social media addiction and depression issues.

Test 3 truly captures the effectiveness of combining the impact of both control strategies to die out the social media addiction and depression dynamics with prevention and treatment interventions ($u_1 = u_2 \neq 0$). Fig 5.15 shows the combined control strategies to optimize the objective function J . From the simulation results, one can easily see that the exposed, addicted, depressed, and suicidal population significantly decreases while the recovered and quitter population suddenly increases compared to the no control solution. This intervention strategy is efficient in bringing down the infected population in the initial years compared to other Tests, making it a more ideal choice for policymakers to manage the behavioral dynamics.

To examine the epidemiological impact of the proposed optimal control strategies, efficiency analysis is critical for all Tests simulated in this study. Efficiency analysis identifies the control intervention that prevents a large quantity of infection, regardless of considering the importance of relevant economic costs during an epidemic. In the context of model (5.30), an intervention with a high efficiency index is more effective for suppressing the media-driven addiction and depression prevalence in the community. Thus efficiency index ($\mathbb{E}$) used for efficiency analysis of control strategies is defined as

$$\mathbb{E} = \frac{\text{Total infection averted}}{\text{Total infection without control}} \times 100. \quad (5.43)$$

Considering total infection $I(t) = I_1(t) + I_2(t)$ in model (5.30), the total infection averted given as

$$\text{Total infection averted} = \int_0^T I(t) dt - \int_0^T \tilde{I}(t) dt,$$

where $\tilde{I}(t)$ shows the infection with control while $I(t)$ without control. Using formulation (5.43), efficiency indices of each control strategy are simulated and given in Table 5.3. Cost-effectiveness analysis, also referred to as economic analysis, has

TABLE 5.3: Efficiency analysis of model (5.30) with $\mathbb{E}$.

Control strategy	Infection baseline	Infection averted	$\mathbb{E}$ (%)
u_1 only	1.4617×10^8	1.0865×10^8	74.332 138
u_2 only	1.4617×10^8	1.2307×10^8	84.194 179
u_1, u_2 combine	1.4617×10^8	1.3322×10^8	91.139 679

particular significance to compare the economic cost of various control strategies assumed in the model (5.30) setting. However, the biological significance of control strategies is critical; it is equally important to determine whether the financial assets required for their execution are worth the health advantages. This analysis gives

an empirical approach that assesses strategies based on their infection suppression compared to the costs of execution. It determines the best epidemiological approach as well as the most practicable and financially advantageous options for execution. We implemented three different techniques to describe the cost-effectiveness analysis, named as Infection averted ratio (IAR), Average cost-effectiveness ratio (ACER), and Incremental cost-effectiveness ratio (ICER). The IAR is adapted to simulate the demographic advantage of each intervention by comparing the quantity of infection occurrences avoided to the number of victims who recover as a consequence of the intervention. It can be formulated as

$$\text{IAR} = \frac{\text{Total infection averted due to specific control strategy}}{\text{Total recovered individuals due to specific control strategy}}, \quad (5.44)$$

where recovered population due to control is written as $\int_0^T \tilde{R}(t) dt$. Any control strategy with the highest IAR value is assumed to be the most cost-effective intervention defined in the optimality system. The precise IAR analysis given in Table 5.4. The ACER used to measure the economic efficiency of each intervention by con-

TABLE 5.4: Cost Effective analysis of model (5.30) with IAR.

Control strategy	Infection averted	Total recovered	IAR (%)
u_1 only	1.0865×10^8	6.1372×10^5	177.035 985
u_2 only	1.2307×10^8	1.5053×10^7	8.175 621
u_1, u_2 combine	1.3322×10^8	7.9042×10^6	16.853 988

trasting its overall execution cost to the entire amount of infection cases mitigated. Mathematically, it is expressed as

$$\text{ACER} = \frac{\text{Total intervention cost for particular control strategy}}{\text{Total infection averted due to particular control strategy}}. \quad (5.45)$$

The total intervention cost (T) define through objective function (5.33) regarding control pairs (u_1, u_2) for model (5.30) given as

$$T = \frac{1}{2} \int_0^T [\kappa_3 u_1^2(t) + \kappa_4 u_2^2(t)] dt,$$

where κ_3, κ_4 are weighted parameters associated with specific control. Furthermore, any control strategy considered to be most cost-effective has least ACER value. Simulation of ACER measurement written in Table 5.5. The ICER is introduced as an in-

TABLE 5.5: Cost Effective analysis of model (5.30) with ACER.

Control strategy	Infection averted	Total cost (\$)	ACER (%)
u_1 only	1.0865×10^8	4.1473×10^1	3.8171×10^{-7}
u_2 only	1.2307×10^8	1.5992×10^2	1.2995×10^{-6}
u_1, u_2 combine	1.3322×10^8	1.9088×10^2	1.4329×10^{-6}

teresting tool for comparing and assessing two different approaches to intervention. By measuring the additional expenditure required to avert an additional infection, it can assist executives in determining if the higher expenses of a more effective approach are worthwhile when compared to other strategies. Assuming $\mathcal{H}_1$ and $\mathcal{H}_2$

are two different control interventions with the same intervention cost, the ICER can be computed with the following expression

$$\text{ICER} = \frac{\text{Differnec in total intervation cost for } \mathcal{H}_1 \text{ and } \mathcal{H}_2 \text{ stratigies}}{\text{Differnec in total infection averted by } \mathcal{H}_1 \text{ and } \mathcal{H}_2 \text{ stratigies}} \quad (5.46)$$

Using (5.46), we capture the comparison of all strategies given in Table 5.6. In the medical sector, along with other domains where cost-effectiveness is crucial, this study is crucial for directing the best possible distribution of resources. The analysis

TABLE 5.6: Cost Effective analysis of model (5.30) with ICER.

Control strategy	Infection averted	Total cost (\$)	ICER (%)
$u_1 \neq 0$	1.0865×10^8	4.1473×10^1	3.8168×10^{-7}
$u_1 \neq 0, u_2 \neq 0$	1.442×10^7	1.18447×10^2	8.214×10^{-6}
$u_1 \neq 0, u_2 \neq 0, u_1 = u_2 \neq 0$	1.015×10^7	3.096×10^1	3.051×10^{-6}

of interventions reveals a distinct hierarchy in the performance of behavioral control strategies in light of efficiency and economic cost indicators. Test 3 with both controls reveals the highest mitigation in disease burden of model (5.1) combining exposed, addicted, depressed, and suicide populations. This case is more effective than Test 2, which utilizes only u_2 control, while Test 1, with only u_1 control, is less dominant relative to Test 2. The economic assessments, which are captured by the ACER and ICER, show that the combining controls u_1, u_2 strategy is still remarkably cost-effective regardless of the extra expenses related to execution of these controls. This approach optimizes the number of addiction and depression prevented per unit expenditure, but only the u_2 control strategy shows a moderate level of economic feasibility and efficacy. On the other side, even if only the u_1 control strategy is less expensive, it is not as financially attractive due to its small influence on disease burden reduction. Essentially combined control innervation shown in Test 3 provides the best possible balance between economic efficiency and medical advantages. Comparison of all strategies with respective controls profile behavior is shown in Fig 5.16, clearly indicating the die out of addiction and depression dynamics in the community.

Remark 5.12. It is crucial to recall that the findings are supported by actual data, enhancing the legitimacy and applicability of the proposed model. The model successfully reflects real-world behavioral dynamics patterns and responds to therapies by integrating actual diagnostic and epidemiological features from the French population. Overall, the study emphasizes the relevance of mathematical modeling, supported by actual data, in aiding cost-effective decision-making targeted at limiting the burden of addiction and depression while boosting long-term public health benefits. Mental health care authorities and policy makers can use these study findings to:

- Distribute modest health care expenditures to finance highly effective intervention.
- Improve the availability of behavioral health services, particularly in regions with a substantial prevalence.
- Construct effective preventative and awareness campaigns based on economical and effective facts.

- Enhance strategic thinking while assessing combined medical advantages and commercial viability.

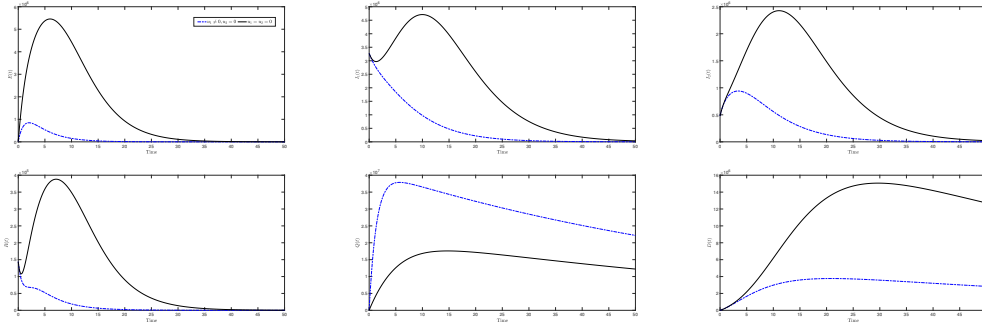


FIGURE 5.13: Test 1: Simulation of the optimality system (5.41)-(5.42) using numerical scheme expressed in Algorithm 3 for evaluation of state variables E, I_1, I_2, R, Q, D row wise from left to right by applying only u_1 control with $u_2 = 0$. We assume social cost $\kappa_1 = 1, \kappa_2 = 2$ and weighted parameters $\kappa_3 = \kappa_4 = 10$ of controls having upper bound $u_{1\max} = 0.6$ and $u_{2\max} = 0$ with $u_i \in [0, 1]$. The simulation is defined with time in years $t \in [0, 50]$ of 1000 discrete time points with step size $0.05/2$ and convergence tolerance 1×10^{-5} .

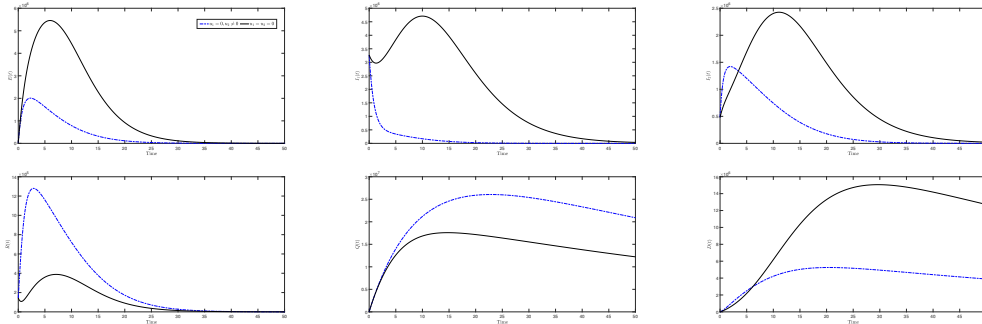


FIGURE 5.14: Test 2: Simulation of the optimality system (5.41)-(5.42) using numerical scheme given in Algorithm 3 for evaluation of state variables E, I_1, I_2, R, Q, D row wise from left to right by applying only u_2 control with $u_1 = 0$. We assume social cost $\kappa_1 = 1, \kappa_2 = 2$ and weighted parameters $\kappa_3 = \kappa_4 = 10$ of controls having upper bound $u_{1\max} = 0$ and $u_{2\max} = 0.8$ with $u_i \in [0, 1]$. The simulation is defined with time in years $t \in [0, 50]$ of 1000 discrete time points with step size 0.025 and convergence tolerance 1×10^{-5} .

5.8 Conclusion

In this study, a novel modeling approach was implemented to describe the coupled addiction and depression states badly affected due to negative social media impact, emphasizing the behavior dynamics in the community. This rigorous perspective provides further insight into how social media, through false information, cyberbullying, and social comparison, shifts the physiological states of individuals. Thus,

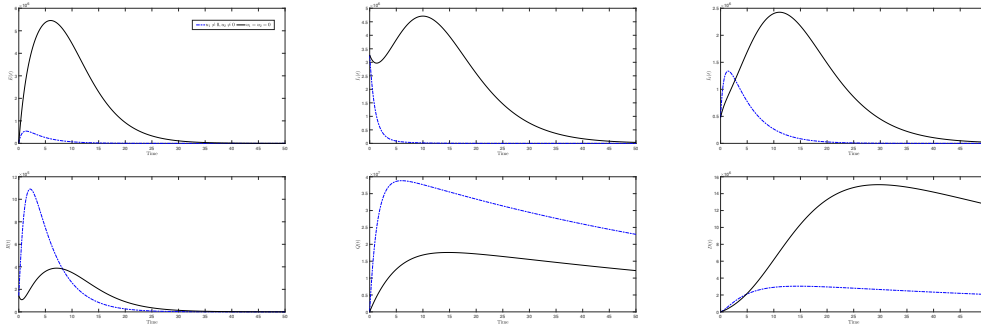


FIGURE 5.15: Test 3: Simulation of the optimality system (5.41)-(5.42) using numerical scheme written in Algorithm 3 for evaluation of state variables E, I_1, I_2, R, Q, D row wise from left to right by applying combine controls $u_1 = u_2 \neq 0$. We assume social cost $\kappa_1 = 1, \kappa_2 = 2$ and weighted parameters $\kappa_3 = \kappa_4 = 10$ of controls having upper bound $u_{1\max} = 0.6$ and $u_{2\max} = 0.8$ with $u_i \in [0, 1]$. The simulation is defined with time in years $t \in [0, 50]$ of 1000 discrete time points with step size 0.025 and convergence tolerance 1×10^{-5} .

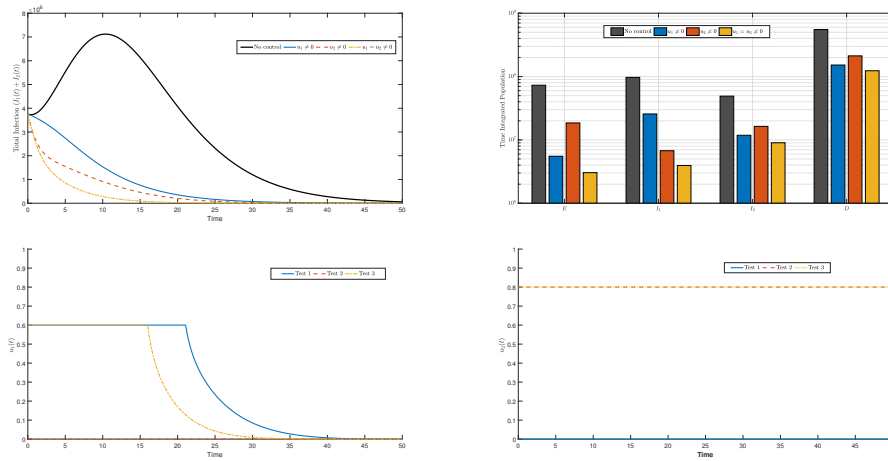


FIGURE 5.16: Comparison of all Tests simulation for optimality system (5.41)-(5.42) using numerical scheme define in Algorithm 3. In the first row, the total infection (right) shows a significant reduction in addition and depression population, while the cumulative population (right) of disease burden presents a comparative relation of interventions. The second row shows both control profiles $u_1(t), u_2(t)$ for all intervention Tests.

an addictive individual with demographic impacts and pre-pressure from the media leads to depressive disorder. By modeling the social behavior structure, we can identify the complex pattern among individuals with addicted and depressed behavior states. The model formulation is defined through a deterministic approach, dividing the total population into susceptible, exposed, addicted, depressed, recovered, quitter, and suicide compartments. After that, data data-driven model was constructed to estimate the biological parameters through actual data and forecast the mutual impact of addiction and depression dynamics over the population using Feed Forward Neural Network (FNN) and Physics Informed Neural Network (PINN) to increase model validity and reliability. The mathematical analysis shows the positivity and boundedness of the model solution. Well-posedness of the model proves the existence and uniqueness of the solution. The long-term behavior of the model presented through basic reproduction number $\mathbb{R}_0$ justifies that the DFF is locally asymptotically stable if $\mathbb{R}_0 < 1$; otherwise EEP when $\mathbb{R}_0 > 1$. The global stability of steady states is described through the Castillo-Chavez theorem and the Lyapunov method, respectively. The global sensitivity analysis captures biological relevance and uncertain behavior of various parameters over the model compartments using the Partial Rank Correlation Coefficient (PRCC). Dynamical analysis reveals that perturbing the key influential parameters, addiction and depression dynamics persist, which motivates to suitable intervention objectives mitigate disease severity. Next, we derive the optimal control model of behavior dynamics using awareness campaigns as prevention control and increasing the medical facility as treatment control to effectively mitigate the behavioral dynamics in the community. The optimal control analysis was performed to evaluate the three strategies: utilization of prevention control u_1 only, implementation of therapeutic control u_2 only, and combined execution of u_1, u_2 . Simulation results prove that all interventions reduce the disease burden, but the combined strategy delivered the highest reduction across addiction and depression populations. The efficiency and cost-effective analysis using E, IAR, ACER, and ICER values showed that despite having a relatively higher economic cost, the $u_1 = u_2 \neq 0$ approach largely reduces addiction and depression prevalence, while the relevant lowest ICER value, indicating the best possible health benefits per unit of expense. The findings support the proposal of integrated control strategies as a feasible prospective health care intervention, particularly in environments where financial limitations must be matched with psychological objectives. Since addiction and depression dynamics emerge from the prior state of individuals. To analyze the memory effect in behavior dynamics, we will use Caputo fractional derivation through complete actual data of any region. Although uncertainty in diagnosis delays, intervention reactions, and behavioral structure influenced the depression dynamics. A stochastic modeling approach will be the best way to calculate these features.

Part III

Boltzmann Type and Mean Field Modeling of Social & Disease Dynamics

Chapter 6

Coupled opinion-epidemic dynamics on graphon-based networks

Understanding the mutual influence between social behavior and physical health is of paramount importance for designing effective epidemic mitigation strategies. In this work, a mathematical framework is developed to analyze the coupled dynamics of opinion formation and disease transmission using graphon-based networks. This approach captures the heterogeneous connectivity patterns found in real social structures. By deriving a kinetic compartmental epidemic model through a mean-field limit, the study investigates the joint evolution of disease states and opinion distributions, focusing on long-term behavior in static and evolving networks. Numerical experiments show that this graphon-based method offers a unified view of opinion-epidemic dynamics, highlighting new strategies for controlling disease spread by influencing population opinions.

6.1 Introduction

As the number of COVID-19 cases in 2020 spiked, it became evident that wearing masks and avoiding social gatherings were crucial safety precautions to safeguard public health, particularly in the absence of medications or vaccinations [45, 49, 308]. On the other hand, it is clear that the effectiveness of lockdown and preventive measures is strongly tied to individual opinions regarding protective behaviors, which themselves depend on personal situational awareness. Indeed, following recent studies [103, 281, 89], social effects and opinion formation phenomena play a significant role in adherence to safety standards, since individuals respond differently to rising case numbers. Opinion formation models have been extensively studied over the past decades, reflecting a broad and growing interest in the behavior of large-scale interacting agent systems across diverse disciplines [40, 80, 207, 118]. Classical models rely on the assumption that individual opinions evolve through binary interactions and external influences such as social media. Most of these models focus on individual-level dynamics, which collectively lead to large and complex systems [87, 141, 148, 128, 153]. In many such settings, microscopic interaction rules give rise to complex patterns and emergent collective behavior. At the same time, it has been shown in recent years [277, 7, 105, 229, 228, 307, 120, 57] that kinetic theory can provide a very powerful framework for understanding the driving mechanisms and properties of macroscopic opinion dynamics starting from simple microscopic rules and through suitable scaling limits.

On the other hand, network theory is crucial for understanding complex dynamics in many fields, including epidemiology [84, 188]. Real-world contact networks, characterized by heterogeneous interaction strengths, clustering, and degree distributions, shape how information and opinions spread. Through the use of such methods, opinion formation models explain how social interactions generate consensus, polarization, or clustered opinions [8, 111, 6, 85, 179] and network topology strongly influences these dynamics. In addition, the increasing availability of data from social platforms has advanced the study of social influence on voting behavior and vaccination campaigns [8, 6], on opinion control [11], polarization [172, 24], and influence strategies [278].

The above arguments suggest that coupling epidemic dynamics with network structure and opinion formation is essential to adequately capture mutual influence effects. Classical epidemic models assume homogeneous mixing among individuals [163, 162, 44], yet real epidemics arise from repeated interactions shaped by both disease features and social behavior [190, 197, 98]. Network-based compartmental models extend this perspective by linking transmission to structured contacts. Network topology strongly affects disease spread: highly connected networks exhibit low epidemic thresholds and rapid transmission [234, 270, 253], while small-world networks slow spread, allowing more time for intervention [292, 150, 252]. Social behavior further shapes epidemic trajectories, as opinions on vaccination, distancing, and disease severity influence protective actions. In this setting, the large size of modern networks, often comprising millions of vertices, motivates the study of their asymptotic topological properties. Graph theory, and graphons in particular, provides an effective framework for describing such large-scale structures and their emergent collective behaviors [71, 286]. Graphons treat large networks as continuous objects, replacing the discrete adjacency matrix with a continuum connectivity function. Building on this idea, the works [43, 223] investigated the asymptotic behavior of network dynamics through graphon-based kinetic and mean-field equations.

In this paper, we develop a mathematical framework to analyze in detail the interplay between opinion formation and disease dynamics through network structures. The framework captures the coupled phenomena of social networks, governing opinion exchange, and physical interactions, driving disease transmission. Opinion dynamics are first described microscopically and, then in the continuum limit, through graphons acting as interaction kernels. This setting reflects quite in detail real-world behaviors where individuals act in a protective manner as a consequence of their own opinion.

The rest of the paper is organized as follows. Section 6.2 presents the coupled compartmental model incorporating opinion exchange over a social network, driven by binary interactions and indirect influence through graphon-based connectivity. In the same section, we derive a Fokker–Planck asymptotics for opinion dynamics on social networks, obtaining the temporal evolution of the joint density of opinions and graphon-encoded connections. We also analyze macroscopic observables of the corresponding surrogate kinetic epidemic model. Section 6.3 discusses graphon estimation from real network data. Section 6.4 provides numerical simulations showing how different scaling regimes and model parameters influence disease outcomes. A last section is devoted to conclusions where we outline perspectives for future research.

6.2 A new model of opinion-driven epidemic dynamics with graphon based connectivity structure

Recent COVID-19 outbreaks have demonstrated that individuals' opinions about the efficacy of mask protections and social distancing have a significant impact on shaping the trajectory of the disease. To capture this complex interaction, we propose a new compartmental model that integrates significant factors that might change the dynamics of an epidemic, including public perceptions toward preventive measures and the influence of prominent agents on a substantial portion of the population. In the following, we take inspiration from [307, 10] to integrate mechanisms of consensus formation into an epidemic model.

6.2.1 Graphon based social connectivity structure

Since we aim to examine patterns and interactions among individuals through network structures, graph theory is well known to provide basic frameworks for modeling such connections. As data grow in scale, graphons emerge as a continuous counterpart of finite graphs, enabling the study of large-scale interaction patterns among individuals; see, for example, [127, 189, 82, 104]. We start then our discussion by introducing an unweighted graph $\mathcal{G}$ defined as a pair $(\mathcal{V}, \mathcal{E})$, where $\mathcal{V}(\mathcal{G})$ is a set of vertices or nodes and $\mathcal{E}(\mathcal{G})$ is a set of edges connecting nodes, pairwise distinct and with the same weight. For the graph $\mathcal{G}$, the so-called adjacency matrix $\mathcal{A}(\mathcal{G}) \in \{0, 1\}^{n \times n}$ is defined by its entries $\mathcal{A}_{ij}(\mathcal{G})$ as

$$\mathcal{A}_{ij}(\mathcal{G}) = \begin{cases} 1 & \text{if } (i, j) \in \mathcal{E}(\mathcal{G}), \\ 0 & \text{otherwise.} \end{cases}$$

These matrices are typically represented by their pixel pictures: discretized unit squares $[0, 1] \times [0, 1]$ with grids of $n \times n$ squares of size $\frac{1}{n} \times \frac{1}{n}$, with n the number of nodes. Given the above definition, the corresponding pixel image at location $(\frac{i-1}{n}, \frac{j-1}{n})$ encoded through the adjacency matrix $\mathcal{A}_{ij}(\mathcal{G})$ is

$$\text{Pixel}(x, y) = \begin{cases} \text{Black} & \text{if } x \in [\frac{i-1}{n}, \frac{i}{n}), y \in [\frac{j-1}{n}, \frac{j}{n}) \text{ and } \mathcal{A}_{ij}(\mathcal{G}) = 1, \\ \text{White} & \text{otherwise.} \end{cases}$$

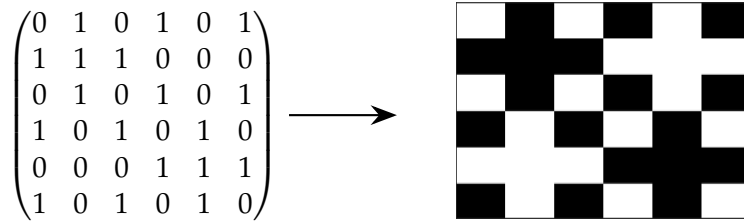


FIGURE 6.1: 6×6 adjacency matrix and corresponding pixel visualization.

In this framework, the concept of a graphon formalizes the convergence of sequences of graphs to a smooth, continuous function, as it represents the exact mathematical limit of a series of large, dense graphs; see Figure 6.2 for a graphical perspective.

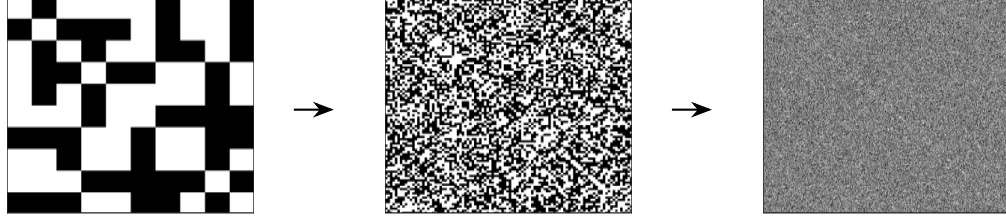


FIGURE 6.2: Pixel representations of graphs with $n = 10, 100, 1000$.

Thus, a graphon $\mathcal{B}$ can be defined as a bounded, measurable, and symmetric function $\mathcal{B} : \Psi \times \Psi \rightarrow [0, 1]$, $\Psi \subset [0, 1]$. To see the space of graphons as the completion of the space of finite graphs and make graphon convergence precise, one can define the distance $D(\mathcal{B}, \mathcal{C})$ between two graphons $\mathcal{B}$ and $\mathcal{C}$ by (see [127])

$$D(\mathcal{B}, \mathcal{C}) = \inf_{\varphi, \psi} \sup_{S, T} \left| \int_{S \times T} \mathcal{B}(\varphi(x), \varphi(y)) - \mathcal{C}(\psi(x), \psi(y)) dx dy \right|,$$

where the infimum is taken over all re-labelings φ of $\mathcal{B}$ and ψ of $\mathcal{C}$, and the supremum is taken over all measurable subsets S and T of $[0, 1]$. This distance measures the maximum discrepancy between the integrals of two labeled graphons over measurable boxes of $[0, 1]^2$, then minimizes that maximum discrepancy over all possible re-labelings. Under the above definitions, it is possible to prove that every graphon is the D -limit of a sequence of finite graphs.

6.2.2 A new model of opinion-driven epidemic dynamics model

We assume now $x \in \Psi = [0, 1]$ to define the network position of individuals (connections) over a given graphon $\mathcal{B}$, while $w \in \Phi = [-1, 1]$ encodes the individual opinion on protection measures, i.e., lockdown, social distancing, and vaccinations. Here, $w = 1$ corresponds to fully endorsing the protection behavior, while $w = -1$ corresponds to strongly rejecting such measures (such as social distancing, wearing masks, or vaccinations) due to misinformation or ideological beliefs. To describe the disease dynamics, we adopt a kinetic compartmental approach based on the Susceptible–Exposed–Infected–Recovered (SEIR) model [142], coupled with the evolution of individuals' opinion distributions over a graphon-based social structure. Although other compartmental epidemic models could be considered depending on the desired level of detail, the SEIR paradigm provides a suitable framework for our purposes.

Let $f_{\mathcal{J}}(x, w, t)$, $\mathcal{J} \in \mathcal{C} = \{S, E, I, R\}$, denote the density of agents at time $t \geq 0$ with opinion w and network position x (as defined by the graphon $\mathcal{B}$) belonging to compartment $\mathcal{J}$. The total density of agents is then

$$\sum_{\mathcal{J} \in \mathcal{C}} f_{\mathcal{J}}(x, w, t) = f(x, w, t), \quad \int_{\Psi \times \Phi} f_{\mathcal{J}}(x, w, t) dx dw = 1, \quad \forall t \geq 0.$$

In this setting, opinion dynamics and graphon-driven social interactions are integrated with the SEIR structure through the kinetic system

$$\partial_t \mathbf{f}(x, w, t) = \mathcal{K}(x, w, \mathbf{f}(x, w, t)) + \frac{1}{\tau} \mathcal{Q}(\mathbf{f}(x, w, t)), \quad (6.1)$$

where $\mathbf{f} = (f_{\mathcal{J}})_{\mathcal{J} \in \mathcal{C}}$. The vector $\mathcal{K}$ describes disease transmission and compartmental transitions, with each component depending on the individual opinion w and network position x . The operator $\mathcal{Q}$ is a integral-type term encoding opinion variation due to social interactions mediated by the graphon structure $\mathcal{B}(x, y)$. The parameter $\tau > 0$ represents the relative time scale of opinion dynamics with respect to the epidemic evolution. Under this framework, the SEIR model within a graphon structure is governed by the following set of kinetic equations:

$$\begin{cases} \partial_t f_S = -\mathcal{K}(f_I, f_S)(x, w, t) + \frac{1}{\tau} \sum_{\mathcal{J} \in \mathcal{C}} \mathcal{Q}(f_S, f_{\mathcal{J}})(x, w, t), \\ \partial_t f_E = \mathcal{K}(f_I, f_S)(x, w, t) - \sigma_E f_E + \frac{1}{\tau} \sum_{\mathcal{J} \in \mathcal{C}} \mathcal{Q}(f_E, f_{\mathcal{J}})(x, w, t), \\ \partial_t f_I = \sigma_E f_E - \gamma f_I + \frac{1}{\tau} \sum_{\mathcal{J} \in \mathcal{C}} \mathcal{Q}(f_I, f_{\mathcal{J}})(x, w, t), \\ \partial_t f_R = \gamma f_I + \frac{1}{\tau} \sum_{\mathcal{J} \in \mathcal{C}} \mathcal{Q}(f_R, f_{\mathcal{J}})(x, w, t). \end{cases} \quad (6.2)$$

Here, $1/\sigma_E$ denotes the average latent period and $1/\gamma$ the mean infectious period. The operator $\mathcal{Q}(\cdot, \cdot)$ models opinion interactions and will be specified later. The disease transmission is characterized by the local incidence rate

$$\mathcal{K}(f_I, f_S)(x, w, t) = f_S(x, w, t) \int_{\Psi \times \Phi} \kappa(w, w_*; x, y) f_I(y, w_*, t) dy dw_*, \quad (6.3)$$

where $0 \leq \kappa \leq 1$ represents the probability of infection modulated by opinions and, indirectly, by network connectivity. Following [307, 10], a possible choice is

$$\kappa(w, w_*; x, y) = \frac{\beta}{4^\eta} (1 - w)^\eta (1 - w_*)^\eta, \quad (6.4)$$

with β the baseline transmission rate and $\eta > 0$ the parameter encoding the effect of protective behavior. Individuals with negative opinions ($w \approx -1$) exhibit higher susceptibility. Conversely, when either $w \approx 1$ or $w_* \approx 1$, the transmission probability decreases substantially. A visualization of (6.4) is provided in Figure 6.5. Note that the kernel (6.4) does not explicitly depend on network positions (x, y) ; however, the opinion dynamics driven by the graphon $\mathcal{B}(x, y)$ influences the distribution of opinions over time, thereby indirectly affecting epidemic spread as specified later. In particular, highly connected nodes and the connectivity patterns encoded by $\mathcal{B}$ play a central role in shaping collective behavior.

6.2.3 Opinion formation under graphon structures

We detail now the opinion model. To that aim, we consider a binary interaction system where two agents with opinions w, w_* and network positions x, y (in the

graphon $\mathcal{B}$) update their post-interaction opinions according to

$$\begin{aligned} w' &= w + \alpha_{\mathcal{H}} P(w, w_*, x, y) (w_* - w) + \zeta_{\mathcal{H}\mathcal{J}} \mathcal{D}(x, w), \\ w'_* &= w_* + \alpha_{\mathcal{J}} P(w_*, w, y, x) (w - w_*) + \zeta_{\mathcal{J}\mathcal{H}} \mathcal{D}(x, w_*), \end{aligned} \quad (6.5)$$

where $\alpha_{\mathcal{J}} \in [0, \frac{1}{2}]$ is the compromise parameter and $P \in [0, 1]$ modulates interaction strength based on the agents' positions on the graphon. The function $\mathcal{D}(x, w)$ represents local diffusion (self-thinking or access to information), and the random variables $\zeta_{\mathcal{H}\mathcal{J}}, \zeta_{\mathcal{J}\mathcal{H}}$ are centered, independent and identically distributed:

$$\langle \zeta_{\mathcal{H}\mathcal{J}} \rangle = \langle \zeta_{\mathcal{J}\mathcal{H}} \rangle = 0, \quad \langle \zeta_{\mathcal{H}\mathcal{J}}^2 \rangle = \langle \zeta_{\mathcal{J}\mathcal{H}}^2 \rangle = \sigma_{\mathcal{J}\mathcal{H}}^2 > 0.$$

A common choice is $\mathcal{D}(w)$ vanishing at the boundary of $\Phi = [-1, 1]$, ensuring opinions remain admissible. The update rule thus combines a deterministic compromise term and a stochastic fluctuation modulated by $\mathcal{D}$. We discuss now different interaction operators models.

Opinion-based interaction. If the interaction kernel depends only on opinion similarity, one may use non-increasing functions such as $P(|w|)$ [277] or $P(|w - w_*|)$, the latter corresponding to the bounded-confidence framework [141]. A standard choice is

$$P(w, w_*) = \chi(|w - w_*| \leq \Delta), \quad w, w_* \in [-1, 1], \quad (6.6)$$

where $\Delta \in [0, 2]$ is the confidence threshold [230]. Here, agents interact only when their opinions are sufficiently close, producing nonlocal effects such as clustering or fragmentation. This formulation neglects network heterogeneity, as interactions depend solely on opinion proximity.

Network-based interaction. If the interaction kernel depends only on network positions, following [104], we define the so-called in-degree

$$d_i(x) = \int_{\Psi} \mathcal{B}(x, y) dy, \quad d_o(y) = \int_{\Psi} \mathcal{B}(x, y) dx,$$

with $\mathcal{B}$ the graphon. Thus, possible choices in this setting include

$$P(x, y) = \exp\left(-\gamma \frac{d_i(x)}{d_i(y)}\right), \quad P(x, y) = \left(1 + \frac{d_i(x)}{d_i(y)}\right)^{-\gamma}, \quad \gamma > 0, \quad (6.7)$$

where the ratio $\frac{d_i(x)}{d_i(y)}$ determines how connectivity shapes opinion: highly connected agents are more resistant to external persuasion, while weakly connected nodes tend to be more affected by interactions.

Coupled opinion–network interaction. Following [10], a more realistic kernel depends on both opinion similarity and network connectivity:

$$P(w, w_*, x, y) = \chi(|w - w_*| \leq \Delta(x, y)) \mathcal{B}(x, y), \quad (6.8)$$

where $\Delta(x, y) \in [0, 2]$ is a position-dependent confidence threshold and $\mathcal{B}(x, y) \in [0, 1]$ encodes connection intensity. This formulation couples social structure and opinion proximity, producing richer dynamics capable of capturing both behavioral similarity and network topology effects. In the numerical section, we will make use of this latter kernel.

Independently on the kernel, since the opinion variable w is defined withing bounded domain $[-1, 1]$, its worth to assume only the interactions that do not result in values beyond these domains. The following statement provides a sufficient condition to maintain such property, see [278, 6, 104] for details.

Proposition 6.1. *Assuming for any $x \in [0, 1]$, $\mathcal{D}(x, \pm 1) = 0$ with pre interaction $w, w' \in [-1, 1]$, the binary interaction (6.5) preserve the bound i.e $w_*, w'_* \in [-1, 1]$ provided*

$$0 < P(w, w_*, x, y) \leq 1, \quad 0 < \alpha_{\mathcal{J}} \leq \frac{1}{2}, \quad |\xi_{\mathcal{JH}}| \leq (1 - \lambda^*)d, \quad (6.9)$$

where

$$\lambda^* = \min_{\substack{\mathcal{J} \in \mathcal{C} \\ w, w_* \in [-1, 1] \\ x, y \in [0, 1]}} \alpha_{\mathcal{J}} P(w, w_*, x, y), \quad d = \min_{\substack{w \in [-1, 1] \\ x \in [0, 1]}} \left\{ \frac{1 - |w|}{\mathcal{D}(x, w)} : \mathcal{D}(x, w) \neq 0 \right\}. \quad (6.10)$$

Moreover, under the same conditions, if additionally the interaction function P is symmetric, one can show that the mean opinion is preserved during an interaction while the mean energy in absence of stochastic terms is dissipated, meaning that a compromise is likely reached.

A continuous description of the microscopic binary interaction (6.5) is provided by a Boltzmann-type equation, where $f_{\mathcal{J}}(x, w, t)$ denotes the probability distribution of opinions and $\mathcal{Q}_{\mathcal{J}}(f_{\mathcal{J}}, f_{\mathcal{H}})(x, w, t)$ is the collision operator accounting for the effect of interactions between opinion distributions. The resulting kinetic equation reads

$$\partial_t f_{\mathcal{J}}(x, w, t) = \frac{1}{\tau} \sum_{\mathcal{H} \in \mathcal{C}} \mathcal{Q}_{\mathcal{J}}(f_{\mathcal{J}}, f_{\mathcal{H}})(x, w, t), \quad (6.11)$$

where $\tau > 0$ denotes the interaction frequency. The weak formulation of (6.11), tested against a smooth function $\varphi(x, w)$, is given by

$$\begin{aligned} \frac{d}{dt} \int_{\Psi \times \Phi} f_{\mathcal{J}}(x, w, t) \varphi(x, w) dx dw &= \int_{\Psi \times \Phi} \mathcal{Q}_{\mathcal{J}}(f_{\mathcal{J}}, f_{\mathcal{H}})(x, w, t) \varphi(x, w) dx dw = \\ &= \frac{1}{2} \int_{\Psi^2 \times \Phi^2} \mathcal{B}(x, y) \langle \varphi(x, w') + \varphi(y, w'_*) - \varphi(x, w) - \varphi(y, w_*) \rangle \\ &\quad \times f_{\mathcal{J}}(x, w, t) f_{\mathcal{H}}(y, w_*, t) dx dy dw dw_*. \end{aligned} \quad (6.12)$$

In (6.12), the operator $\langle \cdot \rangle$ denotes the expectation with respect to the random variables $\zeta_{\mathcal{JH}}$ and $\zeta_{\mathcal{HJ}}$. The parameter τ acts as a scaling factor reflecting the fact that opinion formation and disease diffusion may occur on different time scales: smaller values of τ correspond to faster opinion dynamics relative to the epidemic process. Finally, we emphasize that the interaction operator $\mathcal{Q}_{\mathcal{J}}(\cdot, \cdot)$ does not depend explicitly on the underlying network realization. Indeed, the social structure is assumed to be stationary in time, while the graphon $\mathcal{B}(\cdot, \cdot)$ characterizes the connectivity pattern of the population by modulating the interaction intensity between agents located at positions $x, y \in [0, 1]$.

The qualitative behavior of the kinetic model (6.11) is analytically challenging due to its intrinsic complexity. To investigate its behavior, we consider a Fokker-Planck-type approximation obtained through a so-called quasi-invariant opinion limit [277]. This procedure relies on a suitable rescaling of the interaction parameters, leading to a regime of frequent but weak interactions, in analogy with the grazing collision limit for classical Boltzmann equations. This corresponds to a physical

situation in which the opinions are shaped through a long series of small interactions. In this regime, the kinetic description converges to a mean-field Fokker-Planck equation [230], which preserves the essential microscopic interaction mechanisms while allowing for a more tractable analysis of equilibria and asymptotic behavior. In order to get this limit model, we introduce a small parameter $\varepsilon > 0$ and consider the quasi-invariant scaling

$$\alpha_{\mathcal{J}} \mapsto \varepsilon \alpha_{\mathcal{J}}, \quad \sigma_{\mathcal{J}\mathcal{H}}^2 \mapsto \varepsilon \sigma_{\mathcal{J}\mathcal{H}}^2. \quad (6.13)$$

The rescaled random variables $\zeta_{\varepsilon}^{\mathcal{J}\mathcal{H}}$ and $\zeta_{\varepsilon}^{\mathcal{H}\mathcal{J}}$ are assumed independent with

$$\langle \zeta_{\varepsilon}^{\mathcal{J}\mathcal{H}} \rangle = \langle \zeta_{\varepsilon}^{\mathcal{H}\mathcal{J}} \rangle = 0, \quad \langle (\zeta_{\varepsilon}^{\mathcal{J}\mathcal{H}})^2 \rangle = \langle (\zeta_{\varepsilon}^{\mathcal{H}\mathcal{J}})^2 \rangle = \varepsilon \sigma_{\mathcal{J}\mathcal{H}}^2, \quad \langle (\zeta_{\varepsilon}^{\mathcal{J}\mathcal{H}})^3 \rangle = \langle (\zeta_{\varepsilon}^{\mathcal{H}\mathcal{J}})^3 \rangle < \infty. \quad (6.14)$$

Introducing the slow time scale $t_{\varepsilon} = \varepsilon t$, the weak form of (6.11) becomes

$$\frac{d}{dt_{\varepsilon}} \int_{\Psi \times \Phi} f_{\mathcal{J},\varepsilon}(x, w, t_{\varepsilon}) \varphi(x, w) dx dw = \frac{1}{\varepsilon \tau} \sum_{\mathcal{H} \in \mathcal{C}} \mathcal{Q}_{\mathcal{J}}(f_{\mathcal{J},\varepsilon}, f_{\mathcal{H},\varepsilon}). \quad (6.15)$$

To derive the grazing limit, we perform a Taylor expansion of $\varphi(x, w')$ around w :

$$\begin{aligned} \langle \varphi(x, w') - \varphi(x, w) \rangle &= \partial_w \varphi(x, w) \langle w' - w \rangle + \frac{1}{2} \partial_w^2 \varphi(x, w) \langle (w' - w)^2 \rangle \\ &\quad + \frac{1}{6} \partial_w^3 \varphi(x, \tilde{w}) \langle (w' - w)^3 \rangle, \end{aligned}$$

for some $\tilde{w}$ between w and w' . Using the scaling (6.13)–(6.14) and the interaction rule (6.2.3), we obtain

$$\begin{aligned} \langle \varphi(x, w') - \varphi(x, w) \rangle &= \varepsilon \alpha_{\mathcal{J}} \partial_w \varphi(x, w) P(w, w_*, x, y) (w_* - w) \\ &\quad + \frac{\varepsilon}{2} \partial_w^2 \varphi(x, w) \left(\sigma_{\mathcal{J}\mathcal{H}}^2 \mathcal{D}^2(x, w) + \varepsilon \alpha_{\mathcal{J}}^2 P^2(w, w_*, x, y) (w_* - w)^2 \right) \\ &\quad + \varepsilon^2 \mathcal{R}_{\varepsilon}(\varphi), \end{aligned} \quad (6.16)$$

where the remainder $\mathcal{R}_{\varepsilon}(\varphi) \rightarrow 0$ as $\varepsilon \rightarrow 0$. Substituting (6.16) into (6.15) and letting $\varepsilon \rightarrow 0$, we formally obtain the limiting Fokker-Planck operator

$$\begin{aligned} \overline{\mathcal{Q}}_{\mathcal{J}}(f_{\mathcal{J}}, f_{\mathcal{H}}) &= -\alpha_{\mathcal{J}} \partial_w (\mathcal{K}[f_{\mathcal{H}}](x, w, t) f_{\mathcal{J}}(x, w, t)) \\ &\quad + \frac{\sigma_{\mathcal{J}\mathcal{H}}^2}{2} \partial_w^2 (\mathcal{L}[f_{\mathcal{H}}](x, t) \mathcal{D}^2(x, w) f_{\mathcal{J}}(x, w, t)), \end{aligned} \quad (6.17)$$

where

$$\begin{aligned} \mathcal{K}[f_{\mathcal{H}}](x, w, t) &= \int_{\Psi \times \Phi} \mathcal{B}(x, y) P(w, w_*, x, y) (w_* - w) f_{\mathcal{H}}(y, w_*, t) dy dw_*, \\ \mathcal{L}[f_{\mathcal{H}}](x, t) &= \int_{\Psi \times \Phi} \mathcal{B}(x, y) f_{\mathcal{H}}(y, w_*, t) dy dw_*. \end{aligned} \quad (6.18)$$

The resulting Fokker-Planck model associated with (6.11) reads then

$$\partial_t f_{\mathcal{J}}(x, w, t) = \frac{1}{\tau} \sum_{\mathcal{H} \in \mathcal{C}} \overline{\mathcal{Q}}_{\mathcal{J}}(f_{\mathcal{J}}, f_{\mathcal{H}})(x, w, t), \quad (6.19)$$

supplemented with zero-flux boundary conditions at $w = \pm 1$.

We now discuss the equilibrium structure of the Fokker-Planck approximation

(6.19). In general, the stationary states associated with the operator $\overline{Q}_{\mathcal{J}}(\cdot, \cdot)$ are not available in closed form. However, under suitable assumptions on the diffusion coefficient $\mathcal{D}$, the interaction kernel P , and the graphon $\mathcal{B}$, one can explicitly characterize some steady states. Here we focus on the homogeneous interaction case, i.e. $P(\cdot) \equiv 1$, and on the local diffusion

$$\mathcal{D}(w) = \sqrt{1 - w^2}.$$

Moreover, we assume a separable graphon

$$\mathcal{B}(x, y) = \mathcal{B}_1(x) \mathcal{B}_2(y),$$

a structure consistent with several network models, including power-law graphons; for instance [60] suggests

$$\mathcal{B}(x, y) = (xy)^{-\lambda_g}, \quad 0 < \lambda_g < 1,$$

which yields fat-tailed degree distributions often used to model highly connected agents in social dynamics. Let now $f_{\mathcal{J}}^{\infty}(x, w)$ denote a steady state. From (6.17)-(6.19) we obtain, for each $\mathcal{J} \in \mathcal{C}$,

$$\sum_{\mathcal{H} \in \mathcal{C}} \left[-\alpha_{\mathcal{J}} \partial_w (\mathcal{K}[f_{\mathcal{H}}^{\infty}](x, w) f_{\mathcal{J}}^{\infty}(x, w)) + \frac{\sigma_{\mathcal{J}\mathcal{H}}^2}{2} \partial_w^2 (\mathcal{L}[f_{\mathcal{H}}^{\infty}](x) \mathcal{D}^2(w) f_{\mathcal{J}}^{\infty}(x, w)) \right] = 0, \quad (6.20)$$

where, in the homogeneous case $P \equiv 1$,

$$\begin{aligned} \mathcal{K}[f_{\mathcal{H}}^{\infty}](x, w) &= \int_{\Psi \times \Phi} \mathcal{B}(x, y) (w_* - w) f_{\mathcal{H}}^{\infty}(y, w_*) \, dy \, dw_*, \\ \mathcal{L}[f_{\mathcal{H}}^{\infty}](x) &= \int_{\Psi \times \Phi} \mathcal{B}(x, y) f_{\mathcal{H}}^{\infty}(y, w_*) \, dy \, dw_*. \end{aligned} \quad (6.21)$$

Using the separability of $\mathcal{B}$, the contributions of all classes can be expressed in terms of two weighted quantities

$$m_1 := \sum_{\mathcal{H} \in \mathcal{C}} \int_{\Psi \times \Phi} \mathcal{B}_2(y) f_{\mathcal{H}}^{\infty}(y, w_*) \, dy \, dw_*, \quad m_2 := \sum_{\mathcal{H} \in \mathcal{C}} \int_{\Psi \times \Phi} \mathcal{B}_2(y) w_* f_{\mathcal{H}}^{\infty}(y, w_*) \, dy \, dw_*,$$

which are conserved in time. In particular,

$$\sum_{\mathcal{H} \in \mathcal{C}} \mathcal{B}_1(x) \int_{\Psi \times \Phi} \mathcal{B}_2(y) (w - w_*) f_{\mathcal{H}}^{\infty}(y, w_*) \, dy \, dw_* = \mathcal{B}_1(x) (m_1 w - m_2),$$

$$\sum_{\mathcal{H} \in \mathcal{C}} \mathcal{B}_1(x) \int_{\Psi \times \Phi} \mathcal{B}_2(y) f_{\mathcal{H}}^{\infty}(y, w_*) \, dy \, dw_* = \mathcal{B}_1(x) m_1.$$

The stationary solution in w is then of Beta type. Indeed, introducing

$$\lambda := \frac{\sigma_{\mathcal{J}\mathcal{H}}^2}{2\alpha_{\mathcal{J}}}, \quad a := \frac{1 + \frac{m_2}{m_1}}{\lambda}, \quad b := \frac{1 - \frac{m_2}{m_1}}{\lambda},$$

one obtains the normalized profile

$$f_{\mathcal{J}}^{\infty}(w) = \frac{(1+w)^{a-1} (1-w)^{b-1}}{2^{a+b-1} \mathcal{B}(a, b)}, \quad w \in [-1, 1], \quad (6.22)$$

while the full steady state can be written as

$$f_{\mathcal{J}}^{\infty}(x, w) = C(x) f_{\mathcal{J}}^{\infty}(w), \quad C(x) = \frac{\mathcal{B}_1(x)}{d_i(x) C_{\mathcal{B}}}, \quad (6.23)$$

where $d_i(x)$ denotes the in-degree function and $C_{\mathcal{B}} > 0$ is a normalization constant ensuring $\int_{\Psi \times \Phi} f_{\mathcal{J}}^{\infty}(x, w) dx dw = 1$. Hence, the graphon affects the spatial distribution in x through $\mathcal{B}_1$ (and d_{in}), while the marginal in w remains Beta-type.

6.2.4 Macroscopic contacts and opinion-based epidemic dynamics

Under the quasi-invariant limit derived in Section 6.2.3, the opinion interaction operator $\mathcal{Q}_{\mathcal{J}}(\cdot, \cdot)$ in (6.11) is replaced by the Fokker–Planck-type operator $\overline{\mathcal{Q}}_{\mathcal{J}}(\cdot, \cdot)$ (6.17). This asymptotic description allows us to link the microscopic dynamics of opinion exchange with a macroscopic description of an epidemic, in which the infection rate is modulated by the evolving opinion distribution shaped by the graphon-based social structure. Accordingly, we modify the kinetic SEIR system (6.2) and obtain

$$\begin{cases} \partial_t f_S = -\mathcal{K}(f_I, f_S)(x, w, t) + \frac{1}{\tau} \sum_{\mathcal{J} \in \mathcal{C}} \overline{\mathcal{Q}}(f_S, f_{\mathcal{J}})(x, w, t), \\ \partial_t f_E = \mathcal{K}(f_I, f_S)(x, w, t) - \sigma_E f_E + \frac{1}{\tau} \sum_{\mathcal{J} \in \mathcal{C}} \overline{\mathcal{Q}}(f_E, f_{\mathcal{J}})(x, w, t), \\ \partial_t f_I = \sigma_E f_E - \gamma f_I + \frac{1}{\tau} \sum_{\mathcal{J} \in \mathcal{C}} \overline{\mathcal{Q}}(f_I, f_{\mathcal{J}})(x, w, t), \\ \partial_t f_R = \gamma f_I + \frac{1}{\tau} \sum_{\mathcal{J} \in \mathcal{C}} \overline{\mathcal{Q}}(f_R, f_{\mathcal{J}})(x, w, t), \end{cases} \quad (6.24)$$

System (6.24) describes the epidemic evolution while implicitly accounting for socio-behavioral effects through the opinion variable $w \in [-1, 1]$ and the network position $x \in [0, 1]$ encoded by the graphon $\mathcal{B}(x, y)$. We define now the total masses

$$\rho_{\mathcal{J}}(t) := \int_{\Psi \times \Phi} f_{\mathcal{J}}(x, w, t) dx dw, \quad \mathcal{J} \in \mathcal{C},$$

and the corresponding mean opinions

$$m_{\mathcal{J},w}(t) := \frac{1}{\rho_{\mathcal{J}}(t)} \int_{\Psi \times \Phi} w f_{\mathcal{J}}(x, w, t) dx dw, \quad \mathcal{J} \in \mathcal{C}.$$

In the hypothesis in which the transmission rate $\kappa(w, w_*; x, y)$ is given by (6.4) with $\eta = 1$, integrating (6.24) over $(x, w) \in \Psi \times \Phi$ yields the macroscopic mass dynamics

$$\begin{aligned} \frac{d}{dt} \rho_S(t) &= -\frac{\beta}{4} (1 - m_{S,w}(t)) (1 - m_{I,w}(t)) \rho_S(t) \rho_I(t), \\ \frac{d}{dt} \rho_E(t) &= \frac{\beta}{4} (1 - m_{S,w}(t)) (1 - m_{I,w}(t)) \rho_S(t) \rho_I(t) - \sigma_E \rho_E(t), \\ \frac{d}{dt} \rho_I(t) &= \sigma_E \rho_E(t) - \gamma \rho_I(t), \\ \frac{d}{dt} \rho_R(t) &= \gamma \rho_I(t). \end{aligned} \quad (6.25)$$

Compared to the classical SEIR system, (6.25) is not closed, since the incidence term depends on the mean opinions $m_{S,w}(t)$ and $m_{I,w}(t)$. To obtain evolution equations for the mean opinions, we multiply (6.24) by w and integrate over $\Psi \times \Phi$. This gives

$$\begin{aligned} \frac{d}{dt}(\rho_S(t)m_{S,w}(t)) &= -\frac{\beta}{4}\rho_S(t)\rho_I(t)(1-m_{I,w}(t))\left[m_{S,w}(t) - \int_0^1 \int_{-1}^1 w^2 f_S(x,w,t) dx dw\right] + \\ &\quad + \frac{1}{\tau} \sum_{\mathcal{J} \in \mathcal{C}} \rho_S(t)\rho_{\mathcal{J}}(t)(m_{\mathcal{J},w}(t) - m_{S,w}(t)), \\ \frac{d}{dt}(\rho_E(t)m_{E,w}(t)) &= \frac{\beta}{4}\rho_S(t)\rho_I(t)(1-m_{I,w}(t))\left[m_{S,w}(t) - \int_0^1 \int_{-1}^1 w^2 f_S(x,w,t) dx dw\right] + \\ &\quad - \sigma_E \rho_E(t)m_{E,w}(t) + \frac{1}{\tau} \sum_{\mathcal{J} \in \mathcal{C}} \rho_E(t)\rho_{\mathcal{J}}(t)(m_{\mathcal{J},w}(t) - m_{E,w}(t)), \\ \frac{d}{dt}(\rho_I(t)m_{I,w}(t)) &= \sigma_E \rho_E(t)m_{E,w}(t) - \gamma \rho_I(t)m_{I,w}(t) + \\ &\quad + \frac{1}{\tau} \sum_{\mathcal{J} \in \mathcal{C}} \rho_I(t)\rho_{\mathcal{J}}(t)(m_{\mathcal{J},w}(t) - m_{I,w}(t)), \\ \frac{d}{dt}(\rho_R(t)m_{R,w}(t)) &= \gamma \rho_I(t)m_{I,w}(t) + \frac{1}{\tau} \sum_{\mathcal{J} \in \mathcal{C}} \rho_R(t)\rho_{\mathcal{J}}(t)(m_{\mathcal{J},w}(t) - m_{R,w}(t)). \end{aligned} \tag{6.26}$$

System (6.26) is still not closed due to the second moment of the susceptible opinion distribution,

$$m_{S,w}^{(2)}(t) := \frac{1}{\rho_S(t)} \int_{\Psi \times \Phi} w^2 f_S(x,w,t) dx dw.$$

To close the system we adopt an equilibrium closure based on the Beta-type steady state (6.23). In particular, we approximate

$$\int_{\Psi \times \Phi} w^2 f_S(x,w,t) dx dw \approx \int_{\Psi \times \Phi} w^2 f_S^\infty(x,w) dx dw,$$

which yields

$$m_{S,w}^{(2)}(t) \approx \frac{\lambda_S + 2m_{S,w}^2(t)}{2 + \lambda_S} \int_{\Psi} C(x) dx,$$

for a suitable parameter λ_S associated with the susceptible class. Combining (6.25)–(6.26), we obtain a closed system for the evolution of the mean opinions:

$$\begin{aligned}
\frac{d}{dt}m_{S,w}(t) &= -\frac{\beta}{4}(1 - m_{I,w}(t))\rho_I(t)\left(m_{S,w}^2(t) - m_{S,w}^{(2)}(t)\right) \\
&\quad + \frac{1}{\tau} \sum_{\mathcal{J} \in \mathcal{C}} \rho_{\mathcal{J}}(t)(m_{\mathcal{J},w}(t) - m_{S,w}(t)), \\
\frac{d}{dt}m_{E,w}(t) &= \frac{\beta}{4} \frac{\rho_S(t)\rho_I(t)}{\rho_E(t)}(1 - m_{I,w}(t)) \left[m_{S,w}(t) - m_{S,w}^{(2)}(t) - m_{E,w}(t)(1 - m_{S,w}(t)) \right] \\
&\quad + \frac{1}{\tau} \sum_{\mathcal{J} \in \mathcal{C}} \rho_{\mathcal{J}}(t)(m_{\mathcal{J},w}(t) - m_{E,w}(t)), \\
\frac{d}{dt}m_{I,w}(t) &= \sigma_E \frac{\rho_E(t)}{\rho_I(t)}(m_{E,w}(t) - m_{I,w}(t)) + \frac{1}{\tau} \sum_{\mathcal{J} \in \mathcal{C}} \rho_{\mathcal{J}}(t)(m_{\mathcal{J},w}(t) - m_{I,w}(t)), \\
\frac{d}{dt}m_{R,w}(t) &= \gamma \frac{\rho_I(t)}{\rho_R(t)}(m_{I,w}(t) - m_{R,w}(t)) + \frac{1}{\tau} \sum_{\mathcal{J} \in \mathcal{C}} \rho_{\mathcal{J}}(t)(m_{\mathcal{J},w}(t) - m_{R,w}(t)).
\end{aligned} \tag{6.27}$$

The coupled system (6.25)–(6.27) has the same compartmental structure as the classical SEIR model, but with an opinion-dependent incidence rate. In particular, transmission may significantly decrease (and possibly vanish) when susceptible and infectious individuals adopt protective behaviors (e.g., mask usage and social distancing), which correspond to larger mean positive opinions.

6.3 Graphon Estimation from observed network data

The main difficulty in network modeling is to forecast node interaction probabilities accurately without imposing restrictive functional forms. Over the last decade, machine learning and statistics have advanced significantly through the development of statistical models for network data [294, 221, 5, 144, 267]. Nevertheless, fitting a given parametric family becomes increasingly challenging as network complexity grows. We adopt the exchangeable graph model framework to estimate the graphon from observed network data. Specifically, we rely on the Sorting-And-Smoothing (SAS) algorithm [75], a histogram-based approach that consists of sorting empirical node degrees, rearranging the adjacency matrix accordingly, and constructing a histogram representation, which is subsequently smoothed via total variation minimization. The main steps of the Sorting-And-Smoothing procedure are illustrated in Figure 6.3. In the first step, the rows and columns of the observed graph $G \in \{0, 1\}^{n \times n}$ are reordered based on empirical degrees, yielding the sorted graph $\hat{A} \in \{0, 1\}^{n \times n}$. In the second step, a local histogram $\hat{\mathcal{H}} \in [0, 1]^{k \times k}$ is computed from $\hat{A}$, and the final graphon estimate $\hat{\mathcal{B}}^{\text{tv}} \in [0, 1]^{k \times k}$ is obtained by applying total variation minimization to smooth the histogram.

Thus, the sorting step aims to reorder the observed graph G such that the corresponding empirical degrees form a monotonically non-decreasing sequence. Specifically, the empirical degree of node i is defined as

$$d_i = \sum_{j=1}^n G_{ij}.$$

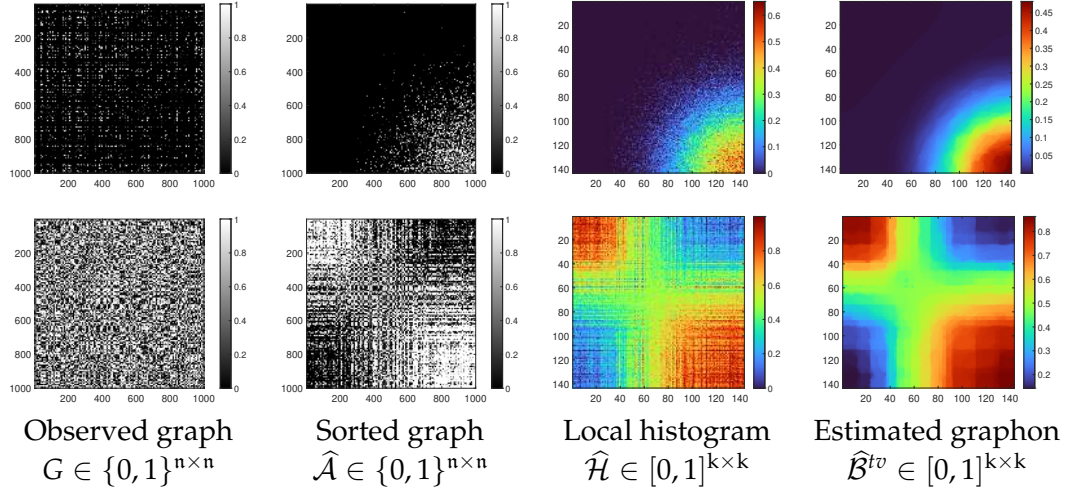


FIGURE 6.3: Illustration of the SAS algorithm for two different scenarios.

We then introduce a permutation $\hat{\theta}$ such that

$$d_{\hat{\theta}(1)} \leq d_{\hat{\theta}(2)} \leq \dots \leq d_{\hat{\theta}(n)},$$

and define the sorted graph as

$$\hat{\mathcal{A}}_{ij} = G_{\hat{\theta}(i)\hat{\theta}(j)}.$$

It is important to note that, the true permutation that determines the canonical graphon may differ from the empirical permutation $\hat{\theta}$ obtained from degree sorting. We therefore denote by θ the oracle permutation that orders the latent variables $\mathcal{U}_i$ associated to the nodes i such that

$$\mathcal{U}_{\theta(1)} \leq \mathcal{U}_{\theta(2)} \leq \dots \leq \mathcal{U}_{\theta(n)}.$$

The corresponding oracle-ordered graph is given by

$$\mathcal{A}_{ij} = G_{\theta(i)\theta(j)}.$$

In the smoothing step, following the canonical representation, graphon estimation reduces to identifying a smooth surface that best approximates the sorted adjacency matrix $\hat{\mathcal{A}}$. To this end, we employ a simplified stochastic block model approximation, in which the continuous graphon is represented by a piecewise-constant function. Specifically, the histogram estimator is defined as

$$\hat{\mathcal{H}}_{ij} = \frac{1}{h^2} \sum_{i_1=1}^h \sum_{j_1=1}^h \hat{\mathcal{A}}_{ih+i_1,jh+j_1},$$

and, analogously, the oracle histogram is given by

$$\mathcal{H}_{ij} = \frac{1}{h^2} \sum_{i_1=1}^h \sum_{j_1=1}^h \mathcal{A}_{ih+i_1,jh+j_1},$$

where $h > 0$ denotes the block size. These expressions show that $\hat{\mathcal{H}}_{ij}$ and $\mathcal{H}_{ij}$ correspond to histogram representations of $\hat{\mathcal{A}}$ and $\mathcal{A}$, respectively. Since the function is constant within each block, the dimensionality of the estimation problem is significantly reduced: instead of estimating n^2 parameters, only k^2 parameters are required, where the number of blocks is $k = \lfloor n/h \rfloor$.

Although the network histogram estimator is consistent, incorporating a total variation (TV) regularization step can significantly improve the error decay rate. This refinement relies on a sparsity assumption on the underlying graphon $\mathcal{B}$. In particular, we assume that graphons are gradient-sparse, in analogy with natural images. Under this assumption, the discretized graphon $\mathcal{B}$, represented on a $k \times k$ grid, is expected to exhibit bounded total variation. The total variation of $\mathcal{B}$ is defined as

$$\|\mathcal{B}\|_{\text{TV}} = \sum_{i=1}^k \sum_{j=1}^k \sqrt{\left(\frac{\partial \mathcal{B}}{\partial x}\right)_{ij}^2 + \left(\frac{\partial \mathcal{B}}{\partial y}\right)_{ij}^2},$$

where $\frac{\partial \mathcal{B}}{\partial x}$ and $\frac{\partial \mathcal{B}}{\partial y}$ denote the horizontal and vertical finite differences of the discretized graphon, respectively. By exploiting this total variation concept, the smoothing stage can be formulated as the following constrained optimization problem:

$$\hat{\mathcal{B}}^{\text{tv}} = \arg \min_{\hat{\mathcal{R}}} \|\hat{\mathcal{R}}\|_{\text{TV}}, \quad \text{subject to } \|\hat{\mathcal{R}} - \hat{\mathcal{H}}\|_2 \leq \varepsilon,$$

where $\varepsilon > 0$ controls the fidelity between the TV-regularized solution $\hat{\mathcal{R}}$ and the histogram estimator $\hat{\mathcal{H}}$, and $\|\cdot\|_2$ denotes the Frobenius norm. The SAS algorithm's total computational complexity is $O(n \log n + k^2 \log k^2)$ multiplications and $O(n^2)$ additions while the magnitude of the final estimate $\hat{\mathcal{B}}^{\text{est}}$ is $n \times n$.

In the following, we consider two real network datasets: the collaboration network of arXiv astrophysics (ca-AstroPh) and the who-trusts-whom network from Epinions.com (soc-Epinions-1). Both datasets are obtained from the Stanford Large Network Dataset Collection (SNAP)¹. The soc-Epinions-1 dataset corresponds to a directed (asymmetric) binary graph with approximately 7.5×10^4 nodes and 5.1×10^5 edges, whereas the ca-AstroPh dataset represents an undirected (symmetric) binary graph with about 1.8×10^4 nodes and 3.9×10^5 edges. To emulate a raw-data scenario in which node labels are intrinsically unordered, we randomly permute the rows and columns of the adjacency matrices for both networks. Figure 6.4 displays the visualizations of the two networks using different bin sizes in order to efficiently highlight their structures. The estimated graphon for the ca-AstroPh network reveals tightly connected collaborations and relatively small communities. In contrast, the soc-Epinions-1 network exhibits recurring interaction patterns driven by influential nodes, which is reflected in the repeated structures observed in the corresponding graphon.

6.4 Numerical Tests

In this section, we present several numerical experiments aimed at illustrating the qualitative behavior of the proposed model. To this end, we adopt an operator-splitting strategy that decouples the opinion dynamics from the epidemiological evolution. Specifically, we introduce the opinion update step $f_\ell^* = \mathcal{I}_{\Delta t}(f_\ell^n)$, defined

¹<http://www.cise.ufl.edu/research/sparse/matrices/SNAP/>

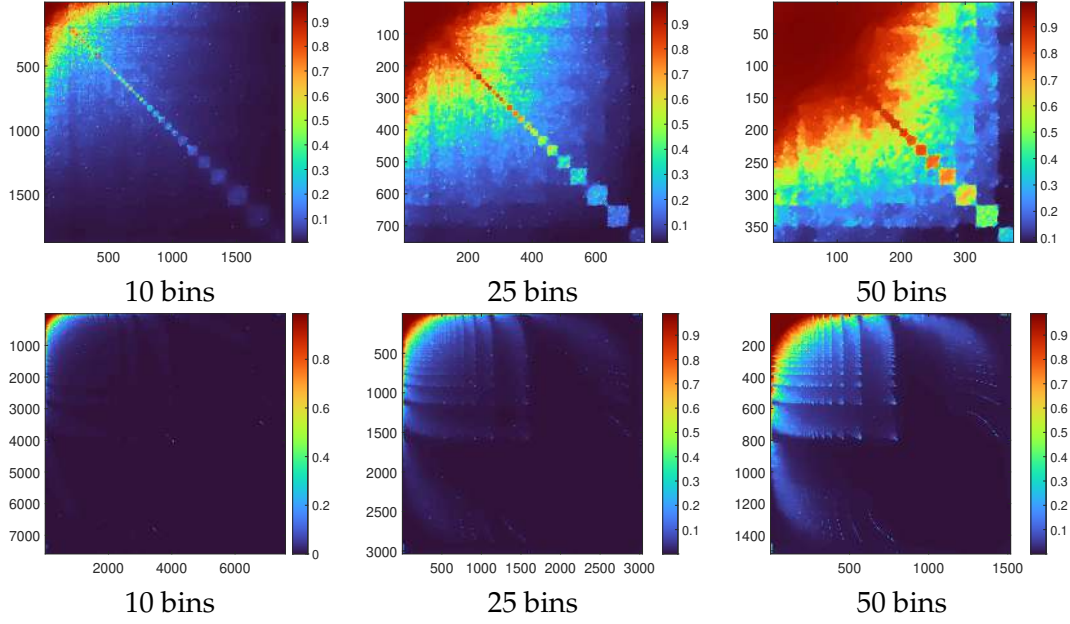


FIGURE 6.4: Estimated graphons for real networks. First row: Collaboration network of arXiv astro physics (ca-AstroPh) $n = 1.8 \times 10^4$. Second row: who-trusts-whom network of Epinions.com (soc-Epinions1) $n = 7.5 \times 10^4$.

as

$$\begin{cases} \partial_t f_\ell^* = \frac{1}{\tau} \sum_{\ell \in \mathcal{C}} \overline{\mathcal{Q}}_\ell(f_\ell^*, f_\ell^*)(x, w, t), \\ f_\ell^*(x, w, 0) = f_\ell^n(x, w), \quad \forall \ell \in \mathcal{C}, \end{cases} \quad (6.28)$$

followed by the epidemiological step $f_\ell^{**} = \mathcal{E}_{\Delta t}(f_\ell^*)$, which reads

$$\begin{cases} \partial_t f_S^{**} = \mathcal{K}(f_S^{**}, f_I^{**})(x, w, t), \\ \partial_t f_E^{**} = \mathcal{K}(f_S^{**}, f_I^{**})(x, w, t) - \sigma_E f_E^{**}, \\ \partial_t f_I^{**} = \sigma_E f_E^{**} - \gamma f_I^{**}, \\ \partial_t f_R^{**} = \gamma f_I^{**}, \\ f_\ell^{**}(x, w, 0) = f_\ell^*(x, w, \Delta t). \end{cases} \quad (6.29)$$

The solution at time t^{n+1} is then obtained by combining the two evolution steps. In particular, a first-order operator-splitting scheme is given by

$$f_\ell^{n+1}(w) = \mathcal{E}_{\Delta t}(\mathcal{I}_{\Delta t}(f_\ell^n(w))),$$

where $\mathcal{I}_{\Delta t}$ and $\mathcal{E}_{\Delta t}$ denote the opinion consensus and epidemiological evolution operators, respectively. The epidemiological system (6.29) is integrated in time using a fourth-order Runge–Kutta (RK4) scheme. For the opinion consensus step (6.28), we employ a first-order semi-implicit, structure-preserving numerical method [231, 41] applied to

$$\partial_t f(x, w, t) = -\alpha \partial_w (\mathcal{K}[f](x, w, t) f(x, w, t)) + \frac{\sigma^2}{2} \partial_w^2 (\mathcal{H}[f](x, t) D^2(x, w) f(x, w, t)),$$

which guarantees the preservation of quasi-steady states. In Figures 6.5 and 6.6, we illustrate the shape of the interaction function $\kappa(w, w_*)$ defined in (6.4) and the equilibrium distributions (6.23) $f_{\mathcal{J}}^{\infty}$ for different choices of the model parameters, respectively.

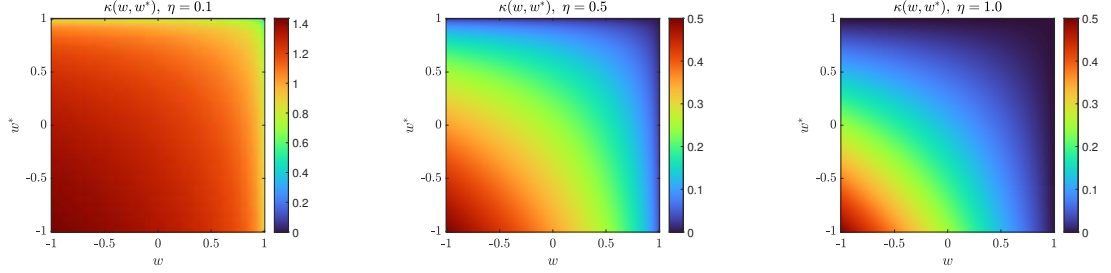


FIGURE 6.5: Function $\kappa(w, w_*)$ in (6.4) for $\eta = 0.1$ (left), $\eta = 0.5$ (middle) and $\eta = 1$ (right). For all cases, $\beta = 0.5$.

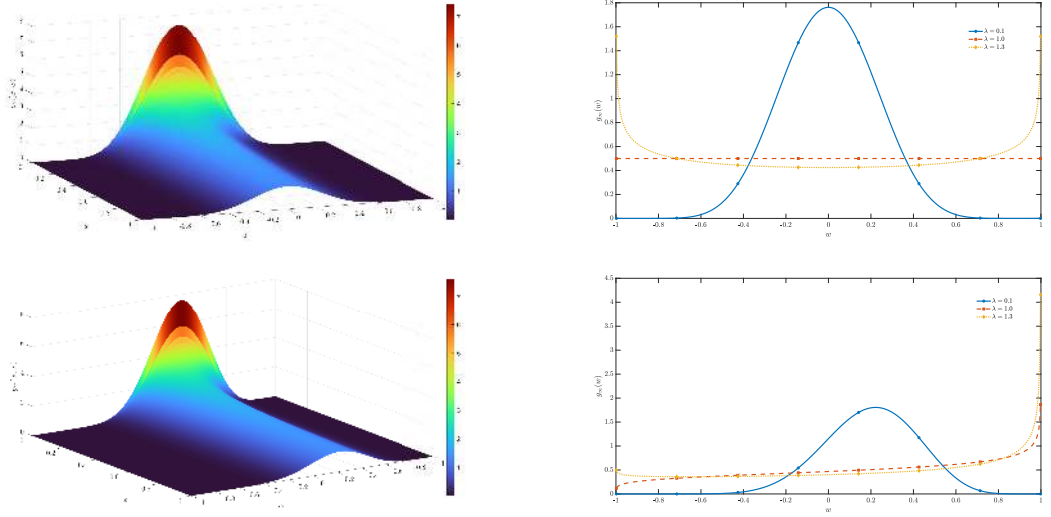


FIGURE 6.6: Equilibrium distribution (6.23) with $\mathcal{B}(x, y) = \frac{9}{16}(xy)^{-1/4}$ for several choices of the parameter $\lambda > 0$, $m_1 = 0$ (first row) and $m_1 = 0.2$ (second row), with $m_2 = 1$. We assume $\lambda = 0.1$. Opinion polarization is observed for $m_1 > 1$, whereas consensus formation corresponds to $m_1 < 1$.

6.4.1 Numerical experiments in homogeneous and graphon-based settings

We first consider a homogeneous network setting and employ Direct Simulation Monte Carlo (DSMC) methods to verify that the time-dependent solution of the Boltzmann-type model (6.12) is consistent with its Fokker–Planck approximation (6.19) in the quasi-invariant limit. To this end, we assume constant interaction kernels, namely the graphon

$$\mathcal{B}(x, y) = 1$$

and the opinion interaction function

$$P(w, w_*, x, y) = 1.$$

Under these assumptions, the steady-state solution (6.23) reduces to a Beta distribution.

Within this framework, we investigate the effect of the initial opinion distribution, chosen as a truncated Gaussian with mean $u_0 = 0$ and variance $\sigma_0^2 = 0.1$, supported on $w \in [-1, 1]$ and uniform in $x \in [0, 1]$. Its probability density function is given by

$$g_0(w) = \begin{cases} \frac{1}{Z} \exp\left(-\frac{(w - u_0)^2}{2\sigma_0^2}\right), & \text{if } -1 \leq w \leq 1, \\ 0, & \text{otherwise,} \end{cases} \quad (6.30)$$

where $Z > 0$ denotes the normalization constant.

We consider a system of $N = 10^5$ particles with relaxation parameter $\tau = 1$, and we adopt the quasi-invariant scaling $\epsilon = 10^{-1}, 10^{-2}, 3 \times 10^{-4}$. Figure 6.7 reports the marginal opinion densities reconstructed via the DSMC method at time $T = 5$, assuming $\epsilon = \Delta t$ and starting from the initial condition (6.30). As expected, the numerical results confirm that the Fokker-Planck approximation converges to the Boltzmann dynamics as $\epsilon \rightarrow 0^+$.

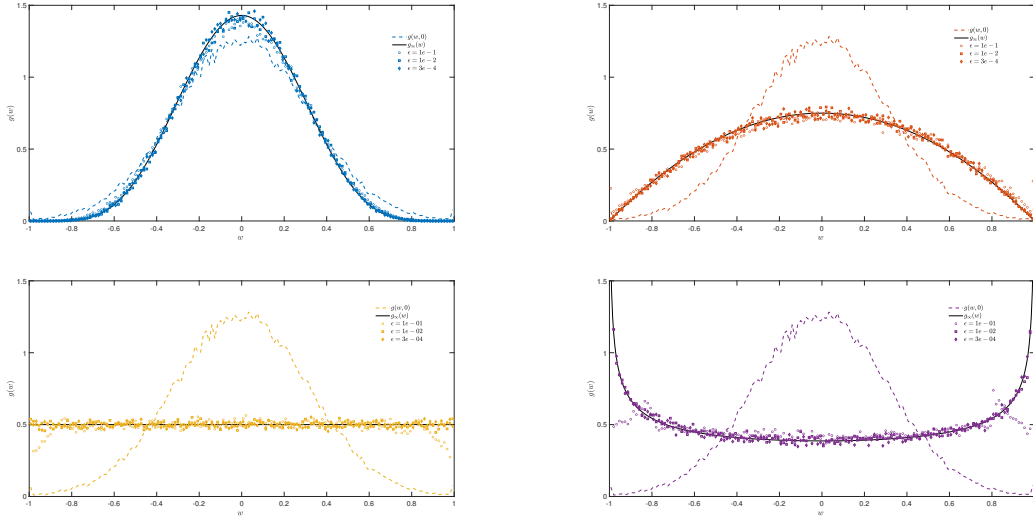


FIGURE 6.7: Comparison between the Boltzmann model (6.19) and the Fokker-Planck equation (6.12), computed via DSMC for $\lambda = 0.15, 0.5, 1.0, 1.5$ (row-wise), with $m_1 = 0$, $m_2 = 1$, and initial distribution (6.30). Simulations use $N = 10^5$ particles over $T \in [0, 5]$ and $\epsilon = \Delta t = 10^{-1}, 10^{-2}, 3 \times 10^{-4}$.

We next investigate the opinion dynamics governed by

$$\partial_t f(x, w, t) = \frac{1}{\tau} \mathcal{Q}(f, f)(x, w, t),$$

using a structure-preserving numerical scheme [231]. The same initial condition (6.30) is adopted. We consider the separable graphon

$$\mathcal{B}_1(u, v) = \exp(-(u^{0.7} + v^{0.7})).$$

The computational domain is discretized using $N_x = 60$ grid points for the connectivity variable and $N_w = 30$ grid points for the opinion variable, with $\tau = 1$. In

all simulations, we fix $\alpha = 1$ and set $\sigma^2 = \epsilon/4$, with $\epsilon = 10^{-1}$, 10^{-2} and time step $\Delta t = \epsilon$. The interaction kernel is assumed constant, $P(w, w_*) = c \in (0, 1)$.

Figure 6.8 illustrates the resulting dynamics. The first column shows snapshots of $f(x, w, t)$ at times $t = 0, 16, 32, 50$ over the interval $T = [0, 50]$. The second column reports the marginal opinion distribution $\int_0^1 f(x, w, t) dx$, while the third column displays the surface plot of $f(x, w, t)$ at the final time.

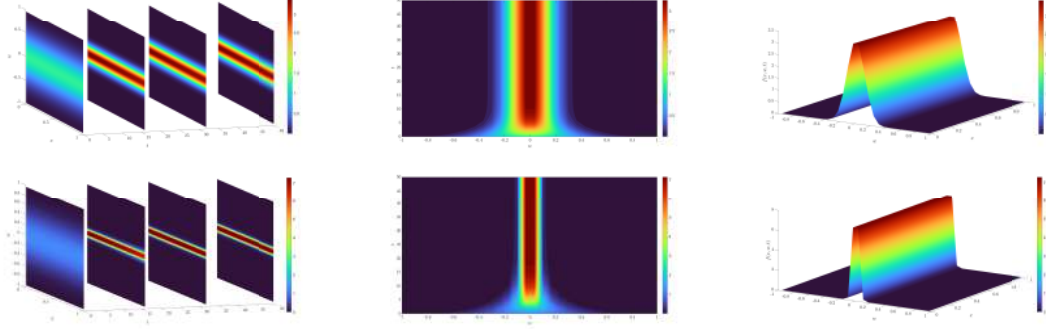


FIGURE 6.8: Evolution of $f(x, w, t)$ for the separable graphon $\mathcal{B}_1$. Rows correspond to $\epsilon = 10^{-1}$ and 10^{-2} , respectively.

We then consider a non-separable interaction structure defined by

$$\mathcal{B}_2(u, v) = 1 - \frac{1}{1 + \exp(-15(0.8|u - v|)^{4/5} - 0.1)},$$

while keeping the same numerical parameters as in the previous case. Figure 6.9 reports the corresponding dynamics for $\epsilon = 10^{-1}$ (first row) and $\epsilon = 10^{-2}$ (second row).

In both settings, the opinion interactions are predominantly attractive. As ϵ decreases, diffusion effects become negligible and the system converges toward a global consensus. Conversely, for larger values of ϵ , diffusion dominates the dynamics, resulting in a broader distribution of opinions without consensus formation.

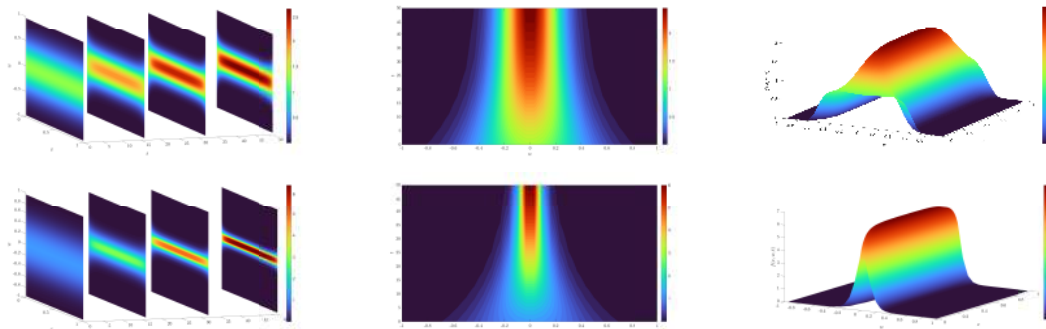


FIGURE 6.9: Evolution of $f(x, w, t)$ for the non-separable graphon $\mathcal{B}_2$. Rows correspond to $\epsilon = 10^{-1}$ and 10^{-2} , respectively.

6.4.2 Opinion formation model with real network data

We investigate opinion dynamics over real social networks by exploiting graphons estimated from empirical data. This framework captures heterogeneous interaction

patterns and allows us to analyze how network structure influences opinion evolution, consensus formation, and fragmentation over time. We assess the applicability of the proposed graphon-based methodology using the two datasets previously described in Section 6.3: the arXiv astrophysics collaboration network (ca-AstroPh) and the who-trusts-whom network from Epinions.com (soc-Epinions-1). We recall that the estimated graphon for the ca-AstroPh network reflects tightly connected collaborations and relatively small communities, whereas the soc-Epinions-1 graphon exhibits repeated interaction patterns driven by influential agents. In the following, we adopt an interaction function that depends explicitly on agents' opinions,

$$P(w, w_*) = \chi(|w - w_*| \leq \Delta), \quad w, w_* \in [-1, 1],$$

which corresponds to the classical bounded confidence model. Under this assumption, two agents influence each other only if their opinions differ by less than Δ . This mechanism introduces nonlocal effects in the opinion dynamics and naturally leads to opinion clustering or fragmentation, rather than global consensus. The simulations are performed with the initial distribution $f(x, w, 0)$ given by (6.30) and model parameters as specified in Section 6.4.1. We discretize the domain $[0, 1] \times [-1, 1]$ using $N_x = 40$ grid points for the connectivity variable and $N_w = 20$ grid points for the opinion variable, and set $\tau = 1$.

Figure 6.10 illustrates the evolution of $f(x, w, t)$ using the graphon from the ca-AstroPh network, assuming $\alpha = 1$, $\sigma^2 = 10^{-1}/4$, and time step $\Delta t = 0.1$. For the bounded confidence model, we consider two confidence thresholds, $\Delta = 0.2$ and $\Delta = 0.8$, shown in the first and second rows, respectively. The first column displays snapshots of $f(x, w, t)$ at times $t = 0, 10, 20, 30$ over the interval $T = [0, 30]$. The second column shows the marginal opinion distribution $\int_0^1 f(x, w, t) dx$, while the third column presents the surface plot of $f(x, w, t)$ at the final time. Figure 6.11 re-

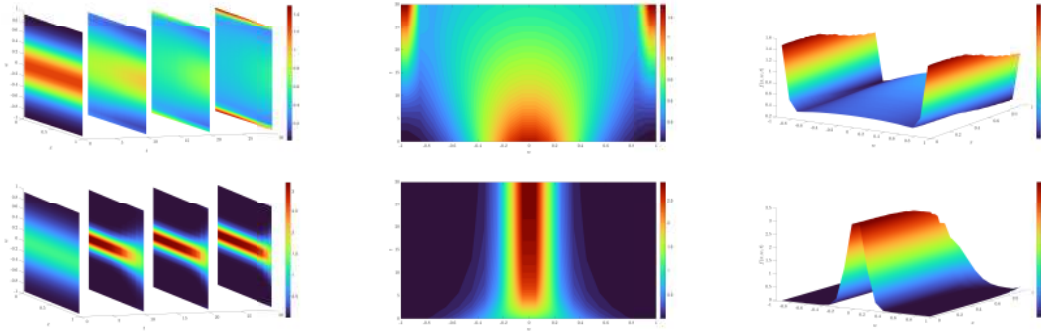


FIGURE 6.10: Opinion dynamics on the ca-AstroPh network. Rows correspond to confidence thresholds $\Delta = 0.2$ and $\Delta = 0.8$. The first column shows snapshots of $f(x, w, t)$ at $t = 0, 10, 20, 30$, the second column reports the marginal opinion distribution, and the third column displays the final-time surface of $f(x, w, t)$.

ports analogous results for the soc-Epinions-1 network, using the same parameter settings. Again, we consider $\Delta = 0.2$ and $\Delta = 0.8$ in the first and second rows, respectively. The first column shows the evolution of $f(x, w, t)$ at times $t = 0, 10, 20, 30$ over $T = [0, 30]$, the second column displays the marginal opinion distribution, and the third column presents the final-time surface.

In both network settings, the estimated graphons generate complex opinion dynamics that cannot be easily reproduced using simple functional graphon models.

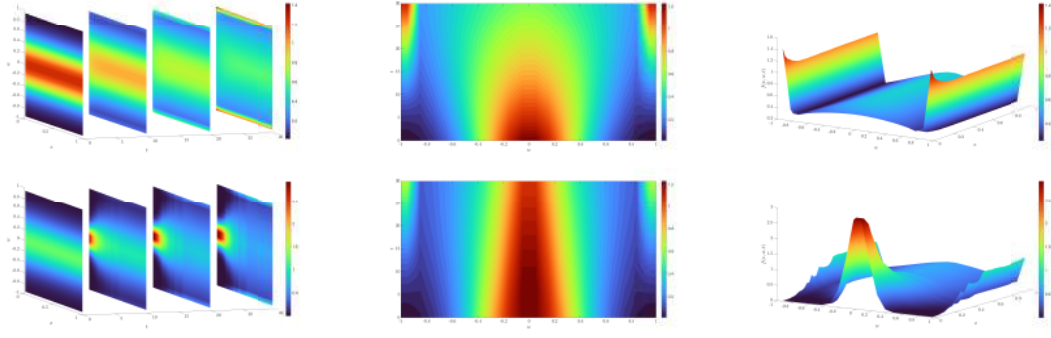


FIGURE 6.11: Opinion dynamics on the soc-Epinions-1 network. Rows correspond to confidence thresholds $\Delta = 0.2$ and $\Delta = 0.8$. The first column shows snapshots of $f(x, w, t)$, the second column reports the marginal opinion distribution, and the third column displays the final-time surface.

In the ca-AstroPh network, a small confidence threshold leads to strong opinion fragmentation, whereas increasing Δ promotes convergence toward consensus. In contrast, the soc-Epinions-1 network exhibits persistent opinion fragmentation and clustering even for large confidence thresholds, highlighting the importance of the underlying social structure in shaping opinions.

6.4.3 Epidemic model simulation with networks

We now focus on the joint epidemic–opinion dynamics described by the SEIR Fokker–Planck model (6.24). The initial conditions are prescribed as

$$f_{\mathcal{J}}(x, w, 0) = \rho_{\mathcal{J}}(0) \chi_{\mathcal{J}}(w) \mathcal{Z}(x),$$

where the opinion distributions $\chi_{\mathcal{J}}(w)$ are defined by

$$\chi_{\mathcal{J}}(w) = \begin{cases} 1, & w \in [-1, 0], \quad \text{for } \mathcal{J} \in \{S, E\}, \\ 1, & w \in [0, 1], \quad \text{for } \mathcal{J} \in \{I, R\}, \\ 0, & \text{otherwise.} \end{cases}$$

The network connectivity profile $\mathcal{Z}(x)$ is assumed to be uniform for $x \in (0, 1)$. The initial mass fractions are set as

$$\rho_E(0) = \rho_I(0) = \rho_R(0) = 10^{-2}, \quad \rho_S(0) = 1 - \rho_E(0) - \rho_I(0) - \rho_R(0). \quad (6.31)$$

We introduce the macroscopic quantities

$$\rho_{\mathcal{J}}^{\tau}(t) = \int_{\Psi \times \Phi} f_{\mathcal{J}}(x, w, t) dx dw, \quad m_{\mathcal{J},w}^{\tau}(t) = \frac{1}{\rho_{\mathcal{J}}^{\tau}(t)} \int_{\Psi \times \Phi} w f_{\mathcal{J}}(x, w, t) dx dw, \quad (6.32)$$

representing the compartment mass fractions and the corresponding mean opinions. The macroscopic system (6.25)–(6.27) is initialized with the mass fractions (6.31) and with initial mean opinions

$$m_{S,w}(0) = m_{E,w}(0) = -0.5, \quad m_{I,w}(0) = m_{R,w}(0) = 0.5.$$

For the opinion dynamics, we assume a consensus-oriented regime by setting $\alpha_{\mathcal{J}} = 1$ and $\sigma_{\mathcal{J}}^2 = 10^{-3}/4$ for all $\mathcal{J} \in \mathcal{C}$, with $m_{\mathcal{J}} = 0.5$. The epidemic parameters are fixed as $\gamma = 1/12$, $\beta = 0.5$, and $\sigma_E = 0.5$. We consider three different settings. In Setting 1 (Fig. 6.12), we adopt a graphon given in functional form as

$$\mathcal{B}(u, v) = \frac{u^2 + v^2}{3} \cos\left(\frac{1}{u^2 + v^2}\right) + 0.15, \quad (6.33)$$

which is non-separable. The first row of Figure 6.12 reports the time evolution of the mass fractions $\rho_{\mathcal{J}}^{\tau}(t)$, while the second row shows the corresponding mean opinions $m_{\mathcal{J},w}^{\tau}(t)$, ordered from left to right by the compartments S, E, I, R .

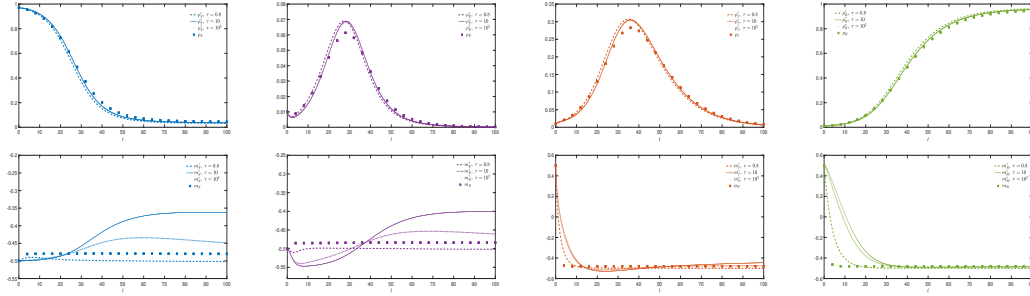


FIGURE 6.12: Epidemic model-Setting 1: Time evolution of the compartment masses (first row) and mean opinions (second row) computed from the SEIR kinetic model (6.2) for $\tau = 0.8, 10, 10^2$, using (6.32). Graphon (6.33) is employed. The domain $[0, 1] \times [-1, 1]$ is discretized with $N_x = 30$, $N_w = 10$, $\Delta t = 0.08$, over $[0, 30]$, with $\sigma^2 = 10^{-3}/4$ and $\beta = 0.5$.

In Setting 2 (Figure 6.13), we replace the functional graphon with an estimated graphon obtained from the ca-AstroPh network. The results are presented in the same format as in Setting 1. Setting 3 (Fig. 6.14) is analogous and uses an estimated

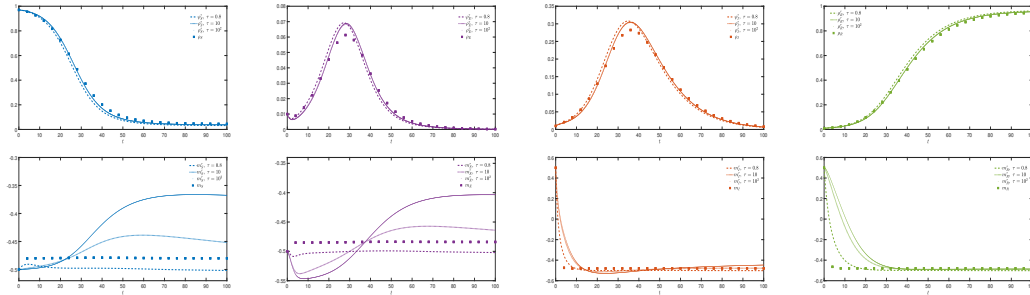


FIGURE 6.13: Epidemic model-Setting 2: Time evolution of the compartment masses (first row) and mean opinions (second row) computed from the SEIR kinetic model (6.2) for $\tau = 0.8, 10, 10^2$, using (6.32), with an estimated graphon from the ca-AstroPh network. The domain $[0, 1] \times [-1, 1]$ is discretized with $N_x = 30$, $N_w = 10$, $\Delta t = 0.08$, over $[0, 30]$, with $\sigma^2 = 10^{-3}/4$, $\beta = 0.5$, and $\eta = 1$.

graphon from the soc-Epinions-1 network. In all cases, decreasing values of τ lead to convergence of the kinetic macroscopic observables ($\rho_{\mathcal{J}}^{\tau}(t), m_{\mathcal{J},w}^{\tau}(t)$) towards the macroscopic quantities predicted by the closure model. Finally, Figure 6.15 illustrates the time evolution of the marginal opinion distributions associated with the Fokker–Planck model (6.24). Rows correspond to Setting 1, Setting 2, and Setting 3,

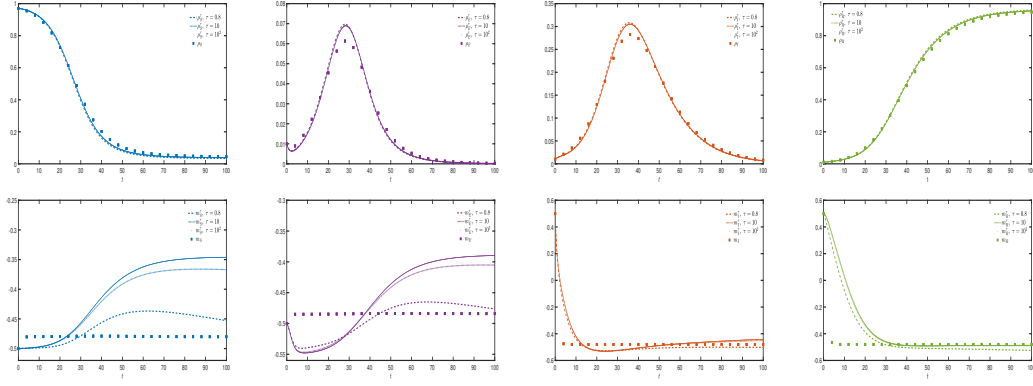


FIGURE 6.14: Epidemic model-Setting 3: Time evolution of the compartment masses (first row) and mean opinions (second row) computed from the SEIR kinetic model (6.2) for $\tau = 0.8, 10, 10^2$, using (6.32), with an estimated graphon from the soc-Epinions-1 network. The domain $[0, 1] \times [-1, 1]$ is discretized with $N_x = 30$, $N_w = 10$, $\Delta t = 0.08$, over $[0, 30]$, with $\sigma^2 = 10^{-3}/4$, $\beta = 0.5$, and $\eta = 1$.

while columns represent the susceptible, exposed, infected, and recovered compartments. These simulations are performed with $\tau = 10$, $\Delta t = 0.2$, and time horizon $[0, 30]$.

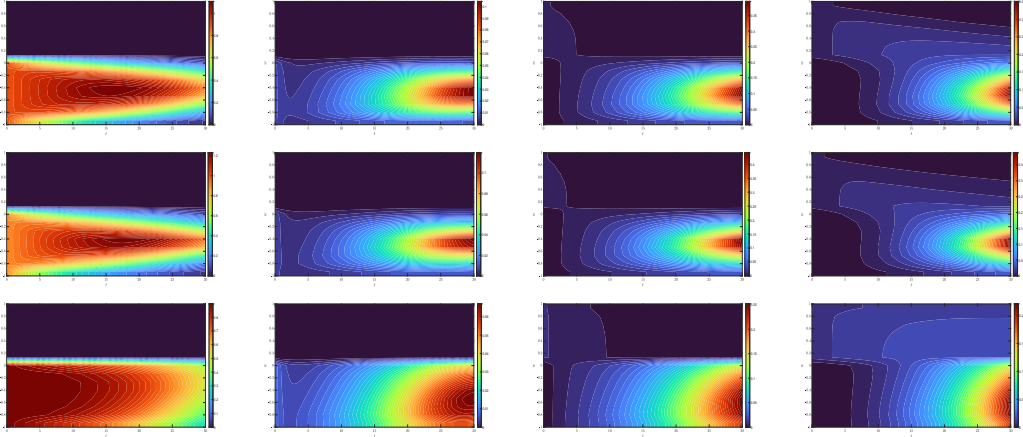


FIGURE 6.15: Time evolution of the marginal opinion distributions for susceptible (first column), exposed (second column), infected (third column), and recovered (fourth column) compartments. Rows correspond to Setting 1, Setting 2 and Setting 3, computed from the Fokker-Planck model (6.24) with $\tau = 10$. The domain $[0, 1] \times [-1, 1]$ is discretized with $N_x = 30$, $N_w = 15$, $\Delta t = 0.2$, over $[0, 30]$, with $\sigma^2 = 10^{-3}/4$ and $\beta = 0.5$.

In conclusion, we consider a bounded confidence model (6.6) in which the interaction function depends explicitly on the fraction of infected individuals $\rho_I(t)$ in the population. The confidence rule is defined as

$$P(w, w_*) = \chi(|w - w_*| \leq \Delta(\rho_I(t))), \quad w, w_* \in [-1, 1], \quad (6.34)$$

where $\chi(\cdot)$ denotes the indicator function and $\Delta(\rho_I(t))$ is a time-dependent confidence threshold driven by the epidemic dynamics. This coupling allows social interactions to adapt to the stage of the outbreak, reflecting behavioral changes such as increased caution and alignment on preventive actions as infection levels rise. We assume that higher values of $\rho_I(t)$ promote consensus formation, mimicking the tendency of individuals to adopt protective attitudes in response to elevated epidemic risk. Specifically, we define

$$\Delta(\rho_I(t)) = \begin{cases} \Delta_1, & \text{if } \rho_I(t) \leq \varepsilon_I, \\ \Delta_2, & \text{if } \rho_I(t) > \varepsilon_I, \end{cases} \quad (6.35)$$

with $0 \leq \Delta_1 < \Delta_2 \leq 2$. For low infection levels ($\rho_I(t) \leq \varepsilon_I$), interactions occur within a narrow confidence bound Δ_1 , favoring opinion clustering. When the infection exceeds the threshold ε_I , the confidence bound expands to Δ_2 , enabling interactions among agents with more heterogeneous opinions and fostering consensus. To evaluate the SEIR Fokker–Planck model (6.24) under this epidemic-driven confidence mechanism and in the presence of graphon interactions, we prescribe the initial conditions

$$f_{\mathcal{J}}(x, w, 0) = \rho_{\mathcal{J}}(0) \chi_{\mathcal{J}}(w) \mathcal{Z}(x),$$

with

$$\chi_{\mathcal{J}}(w) = \begin{cases} 1/2, & w \in [-1, 1], \quad \text{for } \mathcal{J} \in \{S, E\}, \\ 1, & w \in [0, 1], \quad \text{for } \mathcal{J} \in \{I, R\}, \\ 0, & \text{otherwise,} \end{cases}$$

and a uniform connectivity profile $\mathcal{Z}(x)$ for $x \in (0, 1)$. The initial mass fractions are set as

$$\rho_E(0) = \rho_I(0) = \rho_R(0) = 10^{-2}, \quad \rho_S(0) = 1 - \rho_E(0) - \rho_I(0) - \rho_R(0).$$

We investigate the effect of the epidemic-dependent threshold for $\varepsilon_I = 5 \times 10^{-2}$, 3×10^{-2} , 1×10^{-2} , fixing $\Delta_1 = 1/10$ and $\Delta_2 = 1/2$. In all simulations we set $\beta = 0.5$, $\gamma = 1/12$, $\sigma_E = 0.5$, $\eta = 1$, $\tau = 1$, with time step $\Delta t = 0.5$ over $t \in [0, 50]$. Figure 6.16 reports the total marginal opinion density obtained using the non-separable graphon (6.33), Figure 6.17 shows the same analysis using the graphon estimated from the ca-

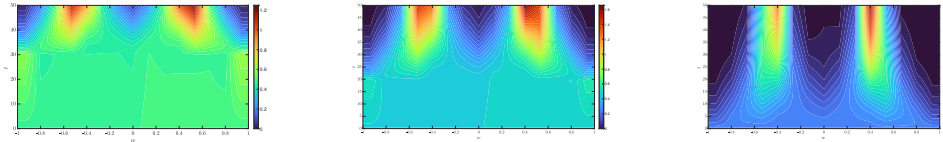


FIGURE 6.16: Graphon 14 case. Total marginal opinion density obtained with the bounded confidence model (6.34) and infection-dependent threshold $\Delta(\rho_I)$. Columns correspond to $\varepsilon_I = 5 \times 10^{-2}$, 3×10^{-2} , and 1×10^{-2} , respectively. The domain $[0, 1] \times [-1, 1]$ is discretized with $N_x = 30$, $N_w = 15$, using $\Delta t = 0.5$ over $t \in [0, 50]$.

AstroPh network. Finally, Fig. 6.18 reports the results obtained using the graphon estimated from the soc-Epinions-1 network.

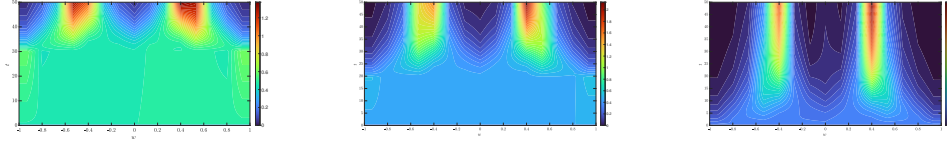


FIGURE 6.17: ca-AstroPh network. Total marginal opinion density obtained with the bounded confidence model (6.34) and infection-dependent threshold $\Delta(\rho_I)$, using the graphon estimated from the ca-AstroPh data. Columns correspond to $\varepsilon_I = 5 \times 10^{-2}$, 3×10^{-2} , and 1×10^{-2} , respectively.

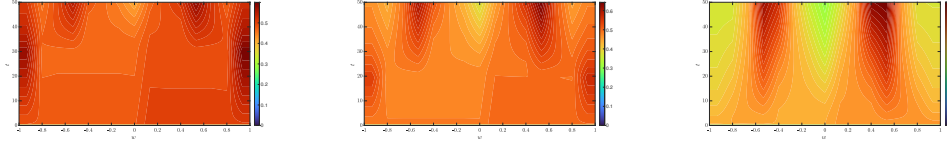


FIGURE 6.18: soc-Epinions-1 network. Total marginal opinion density obtained with the bounded confidence model (6.34) and infection-dependent threshold $\Delta(\rho_I)$, using the graphon estimated from the soc-Epinions-1 data. Columns correspond to $\varepsilon_I = 5 \times 10^{-2}$, 3×10^{-2} , and 1×10^{-2} , respectively.

6.5 Conclusion

In this work, we proposed a novel modeling framework that couples classical epidemiological models with social behavior and opinion formation dynamics over heterogeneous social networks represented through a graphon structure. This integrated perspective provides deeper insight into how infectious diseases propagate in societies, where information spreads rapidly and social influence is shaped by complex and diverse network architectures.

By modeling individual connectivity through a graphon, the proposed approach captures heterogeneous interaction patterns. This allows social effects to emerge naturally from the intrinsic heterogeneity of the network, rather than from ad hoc or time-varying structural assumptions. In particular, the classical SEIR model is extended by incorporating a kinetic description of opinion formation on protective measures, evolving over this graphon-based connectivity structure.

Numerical experiments performed on real network data show that opinion dynamics driven by social interactions can significantly affect epidemic outcomes. In particular, positive influencers promote the adoption of protective behaviors, mitigating disease spread, while negative influencers promote resistance and may trigger recurring outbreaks.

Future work will focus on incorporating additional mechanisms of disease transmission, such as explicit physical contact processes and multilayer social interactions, within the graphon-based framework, thereby further improving the model predictive capability.

Chapter 7

Phase transition in interacting opinion and disease dynamics over multiplex networks

As it was evident during the last COVID-19 pandemic outbreak, disease transmission is heavily influenced by the opinions and beliefs of the population. This is especially important now that the spread of ideas and information is not only confined to physical interactions but instead happens mainly through exchanges on social platforms, leading to a faster evolution of opinions. Therefore, it becomes important to understand how information and opinions on protective measures, such as vaccination or social distancing, evolve during epidemics because, in a population, individual interaction can amplify or mitigate both the spread of information and disease. In this work, we present an SIS opinion model in the presence of social and physical contacts. We start from the microscopic description of the interactions on a multiplex network, then we pass to the kinetic description. We show that, fixing the epidemiological parameters, for different levels of overlap factor between the social and physical network, it is possible to witness a phase transition between the epidemic and the healthy phase. Some numerical simulations for the kinetic model are in process.

7.1 Introduction

Network theory has emerged as a fundamental tool for the study of complex systems across a wide range of disciplines, including biology [174, 202], sociology [121], epidemiology [84, 188], and computer science [194, 244]. Real-world contact networks are inherently heterogeneous, exhibiting features such as variable connection strengths, clustering, and non-trivial degree distributions. These structural properties play a crucial role in shaping the dynamics of biological and social processes, making network-based models particularly suitable for capturing individual-level interactions in a realistic and detailed manner. Beyond biological applications, network theory has proven highly effective in the analysis of social dynamics, especially in the context of opinion formation and diffusion [8, 104, 111, 6, 85, 179], the underlying network topology strongly influencing the speed and outcome of opinion spreading and consensus formation. In parallel, epidemic dynamics on networks have been extensively investigated through compartmental models such as SIS (Susceptible–Infected–Susceptible), SIR (Susceptible–Infected–Recovered), SIAR (Susceptible–Infected–Asymptomatic–Recovered) and many others [131, 253, 26]. In

the these frameworks, individuals transition between health states according to interaction patterns encoded by the network structure. Indeed, the topology of the contact network, and in particular its degree distribution, can dramatically affect disease propagation. For example, highly heterogeneous networks with hub nodes, often display reduced epidemic thresholds, making them more susceptible to outbreaks, whereas clustered networks may slow down disease spread by confining infections within localized groups [216, 234, 253]. On the other hand, the interaction between social behavior and epidemic spreading has recently attracted growing attention [276, 307, 258, 301]. The COVID-19 pandemic clearly demonstrated the pivotal role of public opinion in shaping epidemic outcomes. Beliefs regarding vaccines, social distancing, and perceived disease severity significantly influenced individual behavior and compliance with public health measures, thereby affecting the overall epidemic trajectory. Social influence can thus modify disease transmission, while the epidemic state itself feeds back into opinion formation, generating complex coupled dynamics.

Multiplex networks offer a natural and flexible framework for modeling this interplay [287]. In a multiplex network, multiple layers represent different types of interactions among the same set of individuals. In this paper, in the context of coupled opinion–epidemic dynamics, one layer models social interactions through which opinions are exchanged, while another layer represents physical contacts responsible for disease transmission. These layers are correlated, reflecting the fact that social and physical interactions often overlap, yet they can also exhibit distinct structural properties. The coupling between layers enables a bidirectional feedback mechanism, whereby health states influence opinions and opinions affect individual behavior and infection risk. More in details, we propose a mathematical framework describing the interplay between an opinion dynamics model and a Susceptible–Infected–Susceptible (SIS) epidemic model which takes into account physical connections evolving on distinct but correlated networks. The social layer represents interactions through which individuals exchange information and influence each other’s beliefs, while the physical layer accounts for contacts leading to disease transmission. We explicitly consider correlations between these networks, capturing the realistic scenario in which individuals belonging to the same social group are more likely to interact physically. We assume that both networks are stationary, with fixed topologies and prescribed degree distributions. In particular, following [6], the distribution of social contacts is modeled by a lognormal law, while physical contacts are assumed to follow a Weibull distribution, as suggested in [99]. These assumptions are consistent with empirical observations of real-world social and physical contact networks as discussed later.

The rest of the paper is organized as follows. In Section 7.2, we introduce the multiplex network structure, consisting of two fixed networks defined on the same set of agents, and describe the microscopic interaction rules governing opinion exchange and disease transmission. In Section 7.3, we associate a mesoscopic kinetic description of the same phenomena using Boltzmann-type approaches and obtain a corresponding Fokker-Planck approximation in the quasi-invariant opinion limit. We emphasize that in the proposed model, at the mean-field level, the detailed multiplex structure is replaced by a statistical representation of the dynamics based on the number of connections and not on the specific network structure. Finally, in Section 7.4, we present numerical experiments illustrating the construction of microscopic networks and analyze how different levels of overlap between social and physical layers influence the epidemic evolution.

7.2 Physical and social interactions on multiplex networks: the discrete case.

Starting from the microscopic level, we consider a system composed of $N > 0$ individuals and introduce a two-layer structure consisting of a social contact network and a physical contact network. This setting will be referred to as a multiplex network. For each individual i , $i = 1, \dots, N$, we associate a pair (w_i, d_i) , where $w_i \in [-1, 1] = \Omega$ represents the individual opinion and $d_i \in \{0, 1\}$ denotes the disease status. In particular, $w_i = -1$ corresponds to a strongly negative opinion, while $w_i = 1$ indicates strong agreement with a given idea; moreover, $d_i = 0$ identifies a susceptible individual and $d_i = 1$ an infected one. To formalize the network structure, we introduce a generic unweighted graph $\mathcal{G} = (\mathcal{V}, \mathcal{E})$, where $\mathcal{V}(\mathcal{G})$ denotes the set of vertices (nodes) and $\mathcal{E}(\mathcal{G})$ the set of pairwise distinct edges connecting them. The adjacency matrix associated with $\mathcal{G}$ is defined as

$$\mathcal{A}(\mathcal{G}) \in \{0, 1\}^{N \times N}, \quad \mathcal{A}_{ij}(\mathcal{G}) = \begin{cases} 1, & \text{if } (i, j) \in \mathcal{E}(\mathcal{G}), \\ 0, & \text{otherwise,} \end{cases}$$

where $N = |\mathcal{V}(\mathcal{G})|$ is the number of nodes. Adjacency matrices can be conveniently visualized as pixel images on the unit square $[0, 1] \times [0, 1]$, discretized into an $N \times N$ grid of squares of size $\frac{1}{N} \times \frac{1}{N}$. Each entry $\mathcal{A}_{ij}(\mathcal{G})$ corresponds to a pixel located at $(\frac{i-1}{N}, \frac{j-1}{N})$, which is colored black if $\mathcal{A}_{ij}(\mathcal{G}) = 1$ and white otherwise, namely

$$\text{Pixel}(x, y) = \begin{cases} \text{black,} & \text{if } x \in [\frac{i-1}{N}, \frac{i}{N}), y \in [\frac{j-1}{N}, \frac{j}{N}), \mathcal{A}_{ij}(\mathcal{G}) = 1, \\ \text{white,} & \text{otherwise.} \end{cases}$$

Figure 7.1 illustrates the correspondence between a 6×6 adjacency matrix and its pixel-based representation.

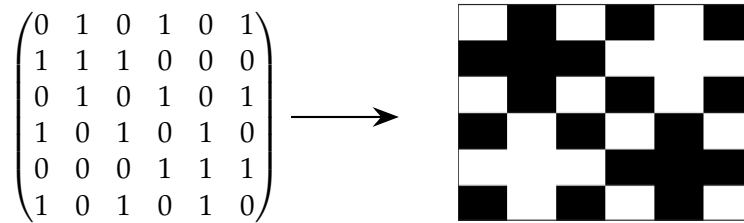


FIGURE 7.1: 6×6 adjacency matrix and corresponding pixel visualization.

The multiplex network is described by two $N \times N$ adjacency matrices as above described, $\mathcal{S} = (s_{ij})$ and $\mathcal{C} = (c_{ij})$, encoding social and physical interactions, respectively. In particular, $s_{ij} = 1$ if individuals i and j are socially connected and $s_{ij} = 0$ otherwise, while $c_{ij} = 1$ if they are physically connected and $c_{ij} = 0$ otherwise. Both networks are assumed to be undirected, so that $s_{ij} = s_{ji}$ and $c_{ij} = c_{ji}$ for all $i, j = 1, \dots, N$. We denote by $\mathcal{E}^{\mathcal{S}}$ and $\mathcal{E}^{\mathcal{C}}$ the corresponding sets of edges. The two layers share the same set of nodes but may differ in their connectivity patterns. The degree of overlap between the social and physical networks is quantified by a parameter $q \in [0, 1]$, representing the fraction of links common to both layers. The

extreme cases $q = 1$ and $q = 0$ correspond to fully coupled and completely uncoupled networks, respectively. Formally, the overlap is measured by the so-called Jaccard index [159]:

$$q(\mathcal{S}, \mathcal{C}) = \frac{|\mathcal{E}^S \cap \mathcal{E}^C|}{|\mathcal{E}^S \cup \mathcal{E}^C|}.$$

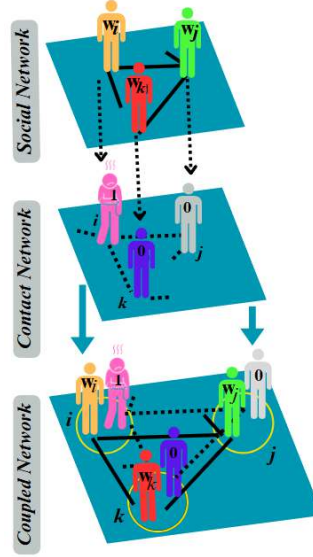


FIGURE 7.2: Schematic representation of a coupled network modeling opinion formation and disease spreading.

Within this two-layer framework, the social network governs the exchange of opinions among individuals, while the physical contact network regulates disease transmission through face-to-face interactions. Figure 7.2 illustrates the coupled structure obtained by superimposing the social layer (upper layer) and the physical contact layer (middle layer), resulting in the combined network shown in the bottom layer. Solid lines denote social connections, whereas dashed lines indicate physical contacts. In the social layer, each node is characterized by an opinion variable $w \in \Omega$, while in the physical contact layer nodes are labeled by their epidemiological state, susceptible (0) or infected (1). As shown in Figure 7.2, nodes i and j are connected by both types of links, indicating simultaneous opinion exchange and potential disease transmission. Nodes j and k interact only socially, exchanging opinions without physical contact, whereas nodes i and k share only a physical link, allowing disease transmission without opinion exchange.

We consider in this setting a scenario in which two individuals interact on a daily basis, for instance by meeting at work or socially, and exchange opinions on various topics. In this case, they are both socially and physically connected. However, when one of the two individuals becomes infected, we assume that their level of social interaction decreases, reflecting common behavioral changes such as staying at home, hospitalization, or the adoption of social distancing measures. As a consequence, face-to-face interactions are reduced or interrupted. This effect is modeled by assigning a probability equal to 1 for social interactions when both individuals are healthy, while reducing it to a value $\mathbb{P}_w \leq 1$ when at least one of the two individuals is infected. Conversely, if two individuals interact only socially and not physically, meaning that they exchange opinions exclusively through remote communication channels, the disease state does not affect their interaction. In this case,

the probability of social interaction and opinion exchange remains equal to 1, independently of the health status of the individuals. Therefore, sickness reduces social interactions only along physical contact links, while remote or electronic interactions in the social network remain unaffected.

We further assume that individuals who are both socially and physically connected are more likely to interact in person when their opinions are similar. In particular, when the distance between their opinions is small, the probability of physical interaction (and hence of disease transmission) is set equal to 1. Conversely, when individuals hold very different opinions, the probability of physical contact is reduced to a value $\mathbb{P}_d \leq 1$. As a result, individuals who meet daily and share similar views transmit the disease with probability β , whereas for individuals with dissimilar opinions the probability of transmission is reduced to $\beta \mathbb{P}_d \leq \beta$. The update rules for opinion dynamics and disease transmission are schematically illustrated in Figure 7.3. As before, social interactions are represented by solid lines, while physical contacts are depicted by dashed lines.

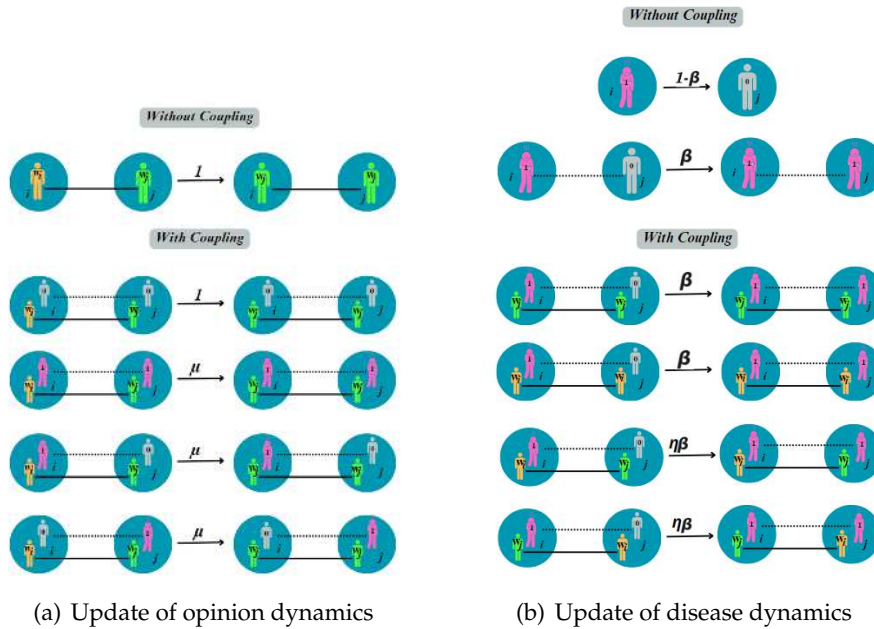


FIGURE 7.3: Schematic representation of the update rules for opinion formation and disease spreading.

7.2.1 The microscopic process of opinion formation

We assume that the two networks are given and fixed in time. Moreover, as $N \rightarrow \infty$, we suppose that the degree distribution of the social network $\mathcal{S}$ converges to a log-normal distribution, as in [6], while following [99] we assume that the degree distribution of the physical contact network $\mathcal{C}$ converges either to a standard Gamma or to a Weibull distribution. This, as already claimed, is consistent with empirical observation about social and contact real-world networks [46]. Throughout the evolution, the total number of agents is assumed to remain constant. Notice that, as discussed in [160], the maximal attainable overlap between two networks depends on their respective degree distributions. As a consequence, for any prescribed choice of distributions there exists an upper bound $d_M \leq 1$ for the overlap factor. We

now formalize the microscopic dynamics described previously. Let $\mathbb{P}_w = \mu \in [0, 1)$ and $\mathbb{P}_d = \eta \in [0, 1)$. Similarly to [277], given two agents $i, j = 1, \dots, N$ with pre-interaction opinions w_i and w_j , their post-interaction opinions are defined by

$$\begin{aligned} w'_i &= w_i + \alpha(w_i, w_j, s_{ij}, c_{ij})(w_j - w_i) + \xi_i D(w_i), \\ w'_j &= w_j + \alpha(w_j, w_i, s_{ji}, c_{ji})(w_i - w_j) + \xi_j D(w_j), \end{aligned} \quad (7.1)$$

where the compromise function $\alpha(w_i, w_j, s_{ij}, c_{ij}) \in [0, 1/2]$ is defined as

$$\alpha(w_i, w_j, s_{ij}, c_{ij}) = \begin{cases} \bar{\alpha} P(w_i, w_j), & s_{ij} = 1, c_{ij} = 0, \\ \bar{\alpha} P(w_i, w_j), & s_{ij} = 1, c_{ij} = 1, d_i = d_j = 0, \\ \mu \bar{\alpha} P(w_i, w_j), & s_{ij} = 1, c_{ij} = 1, (d_i = 1 \text{ or } d_j = 1), \\ 0, & s_{ij} = 0. \end{cases} \quad (7.2)$$

Here $\bar{\alpha} \in [0, 1/2]$ and

$$P(w_i, w_j) = \chi(|w_i - w_j| < \Delta),$$

with $\Delta \in [0, 2]$ modeling bounded confidence interactions. In (7.1), the random variables ξ_i and ξ_j represent self-thinking or external information effects, and satisfy

$$\langle \xi_i \rangle = 0, \quad \langle \xi_i^2 \rangle = \sigma^2 > 0.$$

The function $D(w)$ depends only on the opinion variable and is assumed to vanish at the boundaries, namely $D(\pm 1) = 0$. Since opinions must remain in the bounded interval $[-1, 1]$, we impose the physical admissibility of the interaction rule (7.1). Taking absolute values and applying the triangle inequality yields

$$|w'_i| \leq (1 - \alpha_{ij})|w_i| + \alpha_{ij}|w_j| + |D(w_i)\xi_i|.$$

Using $|w_j| \leq 1$, we obtain

$$|w'_i| \leq (1 - \alpha_{ij})|w_i| + \alpha_{ij} + D(w_i)|\xi_i|.$$

Therefore, a sufficient condition to ensure $|w'_i| \leq 1$ is

$$|\xi_i| \leq \frac{(1 - |w_i|)(1 - \alpha_{ij})}{D(w_i)},$$

for all $w_i \in (-1, 1)$ such that $D(w_i) \neq 0$. This constraint prevents random fluctuations from pushing opinions outside the admissible range. We now define the disease transmission mechanism. The transmission rate $\beta(w_i, w_j, s_{ij}, c_{ij}) \in [0, 1]$ is given by

$$\beta(w_i, w_j, s_{ij}, c_{ij}) = \begin{cases} \beta, & c_{ij} = 1, s_{ij} = 1, |w_i - w_j| < \Delta, \\ \eta\beta, & c_{ij} = 1, s_{ij} = 1, |w_i - w_j| \geq \Delta, \\ \eta\beta, & c_{ij} = 1, s_{ij} = 0, \\ 0, & c_{ij} = 0. \end{cases} \quad (7.3)$$

Thus, physical contact is required for disease transmission, while opinion similarity modulates the effective transmission probability. Finally, each infected agent recovers independently with probability γ , transitioning from $d_i = 1$ to $d_i = 0$, regardless

of network structure. This resulting coupled opinion–epidemic dynamics give rise to behaviors that are not observed in isolated models. In particular, both opinion agreement and disease transmission depend in a nontrivial way on the joint opinion and health states of connected individuals across the two network layers, highlighting the importance of incorporating social effects into epidemic modeling as shown later on.

7.3 A mesoscopic model of opinion formation and disease spread

When passing from the microscopic to the kinetic description, the detailed information on the structure of the coupled networks is necessarily lost, and only statistical information on social and physical contacts can be retained. As a consequence, a kinetic description based on Boltzmann-type equations cannot fully capture key microscopic features, such as the exact coupling between the two network layers or the fine structure of binary interactions induced by the multiplex topology. Nevertheless, it is still possible to introduce a kinetic model that provides an effective, albeit approximate, description of the system in the limit as the number of agents $N \rightarrow \infty$.

We take a different point of view and we assume that the social and physical contact variables take values in the positive real line $\mathbb{R}_+$. We therefore consider the evolution of the density of agents with opinion $w \in \Omega$, number of social connections $s \in \mathbb{R}_+$, and number of physical connections $c \in \mathbb{R}_+$ at time $t \geq 0$, described by

$$f_\ell = f_\ell(w, s, c, t), \quad f_\ell : \Omega \times \mathbb{R}_+ \times \mathbb{R}_+ \times \mathbb{R}_+ \rightarrow \mathbb{R}_+,$$

with $\ell \in \mathcal{C}$. For simplicity, we normalize the total population to one and assume that the number of social and physical contacts remains constant in time, so that

$$\int_{\Omega \times \mathbb{R}_+^2} f(w, s, c, t) \, dw \, ds \, dc = 1, \quad \forall t \geq 0,$$

where $f = f_S + f_I$. The marginal distributions of social and physical contacts are assumed to be fixed and given by

$$h_s(s) = \int_{\Omega \times \mathbb{R}_+} f(w, s, c, t) \, dw \, dc, \quad h_c(c) = \int_{\Omega \times \mathbb{R}_+} f(w, s, c, t) \, dw \, ds,$$

which describe the degree distributions of the networks $\mathcal{S}$ and $\mathcal{C}$, respectively. The joint distribution of social and physical contacts is

$$\int_{\Omega} f(w, s, c, t) \, dw = g(s, c).$$

The degree of correlation between social and physical connectivity is now quantified by the correlation coefficient

$$q = \frac{\text{Cov}_{s,c}(g)}{\sqrt{\text{Var}_s(g) \text{Var}_c(g)}},$$

where

$$\text{Cov}_{s,c}(g) = \int_{\mathbb{R}_+^2} g(s, c) (s - m_s(g)) (c - m_c(g)) \, ds \, dc,$$

with mean values

$$m_s(g) = \int_{\mathbb{R}_+^2} g(s, c) s \, ds \, dc, \quad m_c(g) = \int_{\mathbb{R}_+^2} g(s, c) c \, ds \, dc,$$

and variances

$$\text{Var}_s(g) = \int_{\mathbb{R}_+^2} g(s, c) (s - m_s(g))^2 \, ds \, dc, \quad \text{Var}_c(g) = \int_{\mathbb{R}_+^2} g(s, c) (c - m_c(g))^2 \, ds \, dc.$$

We then suppose that the time evolution of the densities f_S and f_I is governed by the SIS-type kinetic system

$$\begin{cases} \partial_t f_S = -K(f_I)(w, s, c, t) f_S + \gamma f_I + \frac{1}{\tau} \sum_{j \in \mathcal{C}} Q(f_S, f_j)(w, s, c, t), \\ \partial_t f_I = K(f_I)(w, s, c, t) f_S - \gamma f_I + \frac{1}{\tau} \sum_{j \in \mathcal{C}} Q(f_I, f_j)(w, s, c, t), \end{cases} \quad (7.4)$$

where the infection operator is defined as

$$K(f_I)(w, s, c, t) = \int_{\Omega \times \mathbb{R}_+^2} \beta \eta(w, w_*, s, s_*, c, c_*) f_I(w_*, s_*, c_*, t) \, dw_* \, ds_* \, dc_*.$$

Here $\eta \in [0, 1]$ accounts for the modulation induced by opinions and network connectivity, $\gamma > 0$ denotes the recovery rate, and $Q(\cdot, \cdot)$ is a collision operator modeling opinion exchanges.

In weak form, the operator Q reads

$$\begin{aligned} \int_{\Omega \times \mathbb{R}_+^2} Q(f_\ell, f_m)(w, s, c, t) \varphi(w) \, dw \, ds \, dc &= \left\langle \int_{\Omega^2 \times \mathbb{R}_+^4} \theta(s, s_*, c, c_*) \right. \\ &\quad \times (\varphi(w') - \varphi(w)) f_\ell(w, s, c, t) f_m(w_*, s_*, c_*, t) \, dw \, dw_* \, ds \, ds_* \, dc \, dc_* \Big\rangle, \end{aligned} \quad (7.5)$$

for any smooth test function $\varphi : \Omega \rightarrow \mathbb{R}$, where $\theta(s, s_*, c, c_*) > 0$ denotes the interaction frequency and $\langle \cdot \rangle$ denotes expectation with respect to the random variables ξ and ξ_* . The parameter $\tau > 0$ controls the relative time scales of opinion formation and epidemic spreading, with smaller values of τ corresponding to faster opinion dynamics.

Remark 7.1. A possible definition of the interaction frequency $\theta(s, s_*, c, c_*)$ is

$$\theta(s, s_*, c, c_*) = g(s, c)g(s_*, c_*), \quad (7.6)$$

where g is the joint distribution function of social and physical contacts. In the same way, a possible definition of $\alpha(w, w_*, s, s_*, c, c_*)$ that verifies the conditions introduced in Section 7.2 about the opinion formation, reads

$$\alpha(w, w_*, s, s_*, c, c_*) = \bar{\alpha} P(w, w_*) \theta(s, s_*, c, c_*),$$

where $0 < \bar{\alpha} \leq 1$ is a constant, $P(w, w_*)$ is the same interaction kernel used in (7.2) and $\theta(s, s_*, c, c_*)$ is the one defined in (7.6).

7.3.1 A Fokker–Planck limit model for the opinion formation process with physical and social connections

The process of opinion formation involves multiple mechanisms, including the development of personal beliefs shaped by factors such as family, media, and individual experiences. Within this framework, a single interaction contributes only marginally to the overall outcome; rather, the final opinion distribution emerges from the cumulative effect of many small interactions. For this reason, starting from the model derived above, we consider a quasi-invariant opinion variation limit, which leads to a Fokker–Planck–type model obtained from the Boltzmann equations (7.5).

In order to get to this limit, we rescale both the interaction frequency and the magnitude of microscopic opinion exchanges by introducing a small parameter $\epsilon > 0$. In particular, we consider the scaling

$$\alpha_\epsilon = \epsilon \alpha, \quad \kappa_s^\epsilon = \frac{1}{\epsilon} \kappa_s, \quad \langle (\xi^\epsilon)^2 \rangle = \epsilon \sigma^2.$$

This regime corresponds to interactions producing very small opinion changes, but occurring at a very high frequency, so that macroscopic effects are preserved as $\epsilon \rightarrow 0$.

To derive a reduced Fokker–Planck description from the Boltzmann operator (7.5), we define the scaled binary interaction rules as

$$\begin{aligned} w' &= w + \alpha_\epsilon(w, w_*, s, c)(w_* - w) + D(w)\xi^\epsilon, \\ w'_* &= w_* + \alpha_\epsilon(w_*, w, s_*, c_*)(w - w_*) + D(w_*)\xi_*^\epsilon. \end{aligned}$$

Using a Taylor expansion of $\varphi(w')$ around w , we obtain

$$\langle \varphi(w') - \varphi(w) \rangle = \epsilon \alpha(w, w_*, s, c)(w_* - w) \partial_w \varphi(w) + \epsilon \frac{\sigma^2}{2} D^2(w) \partial_w^2 \varphi(w) + R(\epsilon), \quad (7.7)$$

where the remainder term $R(\epsilon)$ is given by

$$\begin{aligned} R(w, w_*, s, c, \epsilon) &= \frac{\epsilon^2}{2} \left(\alpha^2(w, w_*, s, c)(w_* - w)^2 \partial_w^2 \varphi(w) \right. \\ &\quad \left. + \frac{1}{3} \alpha(w, w_*, s, c)(w_* - w) D^2(w) \partial_w^3 \varphi(w) \right) \\ &\quad + \frac{1}{6} \partial_w^3 \varphi(\bar{w}) \left(\epsilon^3 \alpha^3(w, w_*, s, c)(w_* - w)^3 + \langle (\xi^\epsilon)^3 \rangle D^3(w) \right), \end{aligned} \quad (7.8)$$

with $\bar{w} = \zeta w + (1 - \zeta)w'$ for some $\zeta \in [0, 1]$. Notice that $R(\epsilon) = \mathcal{O}(\epsilon^2)$.

Substituting (7.7) into the weak formulation of (7.5), we obtain

$$\begin{aligned} &\int_{\Omega \times \mathbb{R}_+^2} Q(f_l, f_m)(w, s, c, t) \varphi(w) \, dw \, ds \, dc \\ &= \int_{\Omega \times \mathbb{R}_+^2} \left(\mathcal{E}[f_m] \partial_w \varphi + \frac{\sigma^2}{2} \mathcal{D}[f_m] \partial_w^2 \varphi \right) f_l \, dw \, ds \, dc + \frac{\mathcal{R}(\epsilon)}{\epsilon}. \end{aligned}$$

where

$$\mathcal{E}[f_m](w, s, c, t) = \int_{\Omega \times \mathbb{R}_+^2} \theta(s, s_*, c, c_*) \alpha(w, w_*, s, c)(w_* - w) f_m(w_*, s_*, c_*, t) \, dw_* \, ds_* \, dc_*,$$

and

$$\mathcal{D}[f_m](w, s, c, t) = D^2(w) \int_{\Omega \times \mathbb{R}_+^2} \theta(s, s_*, c, c_*) f_m(w_*, s_*, c_*, t) dw_* ds_* dc_*.$$

Here $\mathcal{R}(\epsilon)$ denotes the integrated remainder term, which vanishes as $\epsilon \rightarrow 0$.

Performing integration by parts and letting $\epsilon \rightarrow 0$, we obtain the reduced Fokker–Planck operator $\bar{Q}$ in weak form:

$$\int_{\Omega \times \mathbb{R}_+^2} \bar{Q}(f_I, f_m) \varphi dw ds dc = \int_{\Omega \times \mathbb{R}_+^2} \left[\partial_w (-\mathcal{E}[f_m] f_I) + \frac{\sigma^2}{2} \partial_w^2 (\mathcal{D}[f_m] f_I) \right] \varphi dw ds dc. \quad (7.9)$$

This corresponds to the strong Fokker–Planck equation

$$\partial_t f_I = -\partial_w (\mathcal{E}[f_m] f_I) + \frac{\sigma^2}{2} \partial_w^2 (\mathcal{D}[f_m] f_I), \quad (7.10)$$

supplemented with the boundary conditions

$$\begin{aligned} -\mathcal{E}[f_m] f_I + \frac{\sigma^2}{2} \partial_w (\mathcal{D}[f_m] f_I) &= 0, & w = \pm 1, \\ \mathcal{D}[f_m] f_I &= 0, & w = \pm 1. \end{aligned} \quad (7.11)$$

Finally, the SIS-type kinetic model coupling opinion formation and epidemic spreading in the case of the quasi-invariant opinion limit reads

$$\begin{cases} \partial_t f_S = -K(f_I) f_S + \gamma f_I + \frac{1}{\tau} \sum_{m \in \mathcal{C}} \bar{Q}(f_S, f_m), \\ \partial_t f_I = K(f_I) f_S - \gamma f_I + \frac{1}{\tau} \sum_{m \in \mathcal{C}} \bar{Q}(f_I, f_m), \end{cases} \quad (7.12)$$

where the parameter $\tau > 0$ controls the relative time scale between opinion formation and epidemic dynamics: smaller values of τ correspond to faster opinion evolution.

7.4 Numerical simulations

In this section, we perform several numerical simulations to assess the behaviors of the microscopic model described in Section 7.2 and of the kinetic model discussed in Section 7.3.

7.4.1 Construction of the adjacency matrices

We performed our numerical simulations on coupled $N \times N$ networks. To construct the adjacency matrices $\mathcal{S}$ and $\mathcal{C}$, we employed the configuration model algorithm (see, e.g., [217]), which is summarized in Algorithm 4.

Once the adjacency matrices of the two networks are obtained, we apply a *cross-rewiring procedure* (see [160]) to induce a prescribed level of overlap between the graphs. The procedure is described in Algorithm 5.

Algorithm 4 Configuration model algorithm

Sample a vector $K = \{k_1, \dots, k_N\}$ of integers from the prescribed degree distribution.

Ensure that $\sum_{i=1}^N k_i$ is an even integer.

Construct a *stub list*, i.e., a vector containing k_i copies of node i for each $i = 1, \dots, N$.

while length(stub list) > 0 **do**

Uniformly at random select an element j from the stub list and remove it.

Uniformly at random select an element $k \neq j$ from the stub list and remove it.

Add an undirected edge between nodes j and k .

end while

Algorithm 5 Cross-rewiring algorithm

Let G and G' be two networks, and let $\bar{q}$ denote the desired overlap factor.

Compute the Jaccard index $q(G, G')$ between the two networks.

while $q(G, G') < \bar{q}$ **do**

Randomly select a pair of edges (a, b) and (c, d) in G such that:

1. (a, b) and (c, d) are not edges of G' ;
2. (a, c) and (b, d) are not edges of G ;
3. (a, c) is an edge of G' .

Replace edges (a, b) and (c, d) with (a, c) and (b, d) in G .

Interchange the roles of G and G' .

end while

7.4.2 Numerical simulations for the microscopic model

We consider two different settings. In the first case, both layers are random graphs, while in the second case the social network follows a lognormal degree distribution while the physical contact network follows a Weibull degree distribution. We set $N = 10^3$ and fix the mean degree of the social network to $m_s = 25$, while the mean degree of the physical contact network is set to $m_c = 20$. We emphasize that these two values are not expected to coincide: individuals typically interact with a larger number of people on social media platforms than in face-to-face encounters, which motivates our choice $m_s > m_c$.

Figure 7.4 (left panel) shows a comparison between the normalized degree distributions of $\mathcal{S}$ and $\mathcal{C}$. For the opinion interaction step, in (7.2) we set $\mu = 0$, $\bar{\alpha} = 0.5$, and $\Delta = 2$. The variance σ^2 of the random variable ξ is fixed to 10^{-3} , while we choose $D(w) = 1 - w^2$. For the epidemic dynamics, in (7.3) we set $\eta = 0$ and consider a range of transmission rates $\beta \geq 0.5$, with the recovery rate defined as $\gamma = 1 - \beta$. At the initial time, 50% of the nodes are assigned to the infected compartment. We report the final fraction of infected individuals once a stationary state is reached. The value of ρ_I is obtained by averaging over five independent realizations for each value of β . This setting is used for running simulations for different values of the overlap factor q ranging from 0.1 to 0.6. Figure 7.5 shows that, starting from lower values of $q(\mathcal{S}, \mathcal{C})$, the system undergoes a transition from a healthy phase, where the stationary fraction of infected individuals is zero, to an endemic phase, characterized by a strictly positive stationary fraction of infected individuals. This transition

occurs for transmission rates larger than 0.5, which corresponds to the critical threshold in the classical SIS model. Figure 7.6 displays analogous results obtained with the same set of parameters, except for the bounded confidence interval which is set to $\Delta = 0.5$.

For the second case, we retain the same values of m_s and m_c used in the random graph simulations. This choice yields a lognormal degree distribution for the social network with logarithmic scale parameter equal to 2.8989 and shape parameter 0.8, and a Weibull degree distribution for the physical contact network with scale parameter 17 and shape parameter 1.1765. Figure 7.4 (right panel) shows a comparison between the normalized degree distributions of $\mathcal{S}$ and $\mathcal{C}$. Figure 7.7 presents a comparison of the stationary fraction of infected individuals for different values of the transmission rate β and various overlap factors, in the case of lognormal (social contacts) and Weibull (physical contacts) networks. We finally consider the same setting while modifying the bounded confidence parameter to $\Delta = 0.5$. Figure 7.8 reports the resulting final fraction of infected individuals (left panel) together with the corresponding phase diagram as functions of $q(\mathcal{S}, \mathcal{C})$ and β (right panel). A last Figure 7.9 shows a comparison of the final opinion distributions for $\beta = 0.5$, $\beta = 0.58$, and $\beta = 0.66$, and for different values of the overlapping factor.

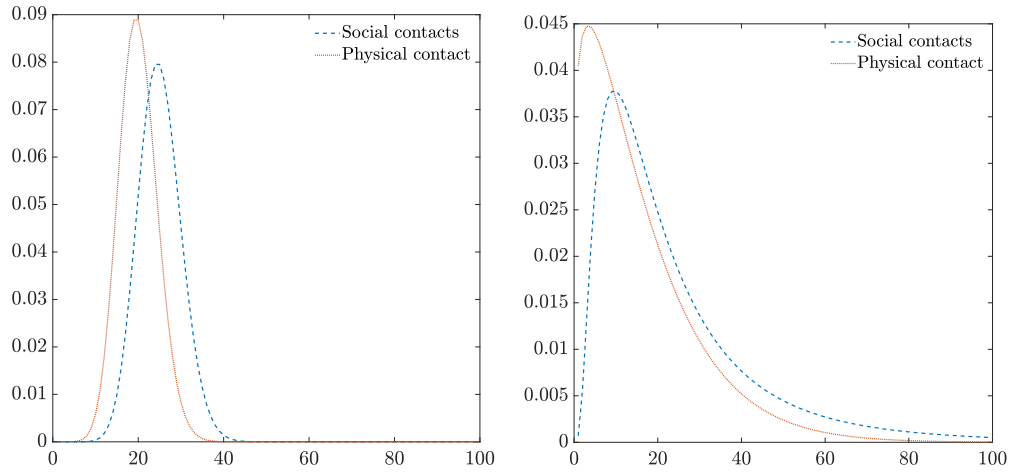


FIGURE 7.4: Comparison between normalized degree distributions of $\mathcal{S}$ and $\mathcal{C}$ in the case of random graphs (left) and in the case of social contacts distributed as a lognormal and physical contacts distributed as a Weibull (right).

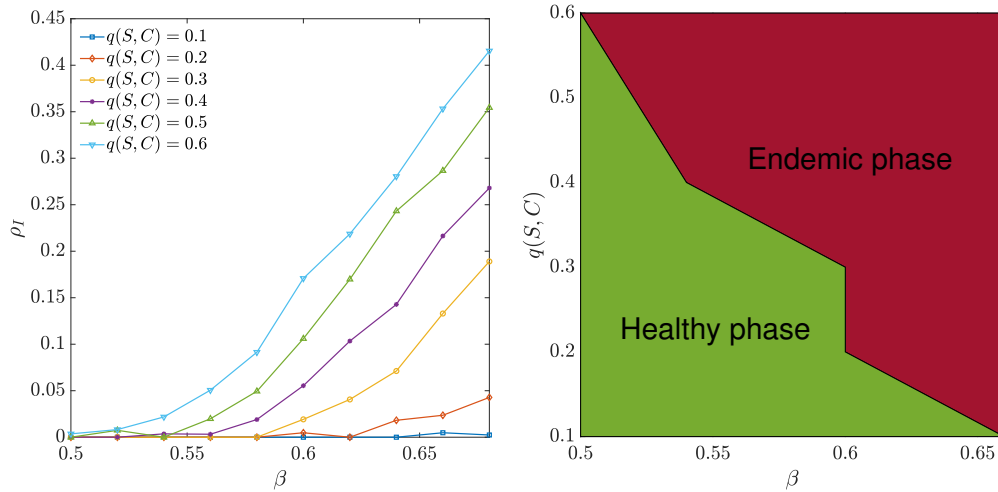


FIGURE 7.5: Bounded confidence parameter $\Delta = 2$. Final normalized number of infected nodes for different values of β and $q(S, C)$ (left) and visual representation of the phase diagram (right). Random graphs case.

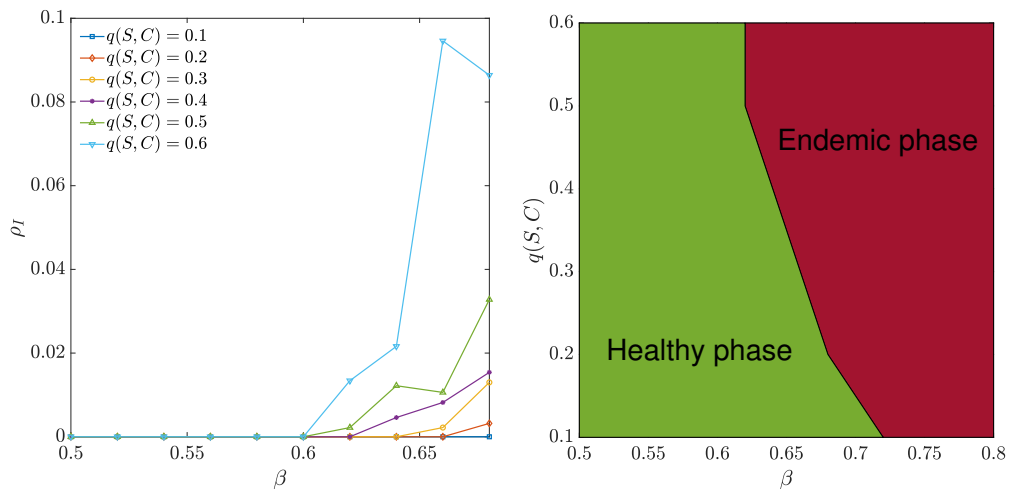


FIGURE 7.6: Bounded confidence parameter $\Delta = 0.5$. Final normalized number of infected nodes for different values of β and $q(S, C)$ (left) and visual representation of the phase diagram (right). Random graphs case.

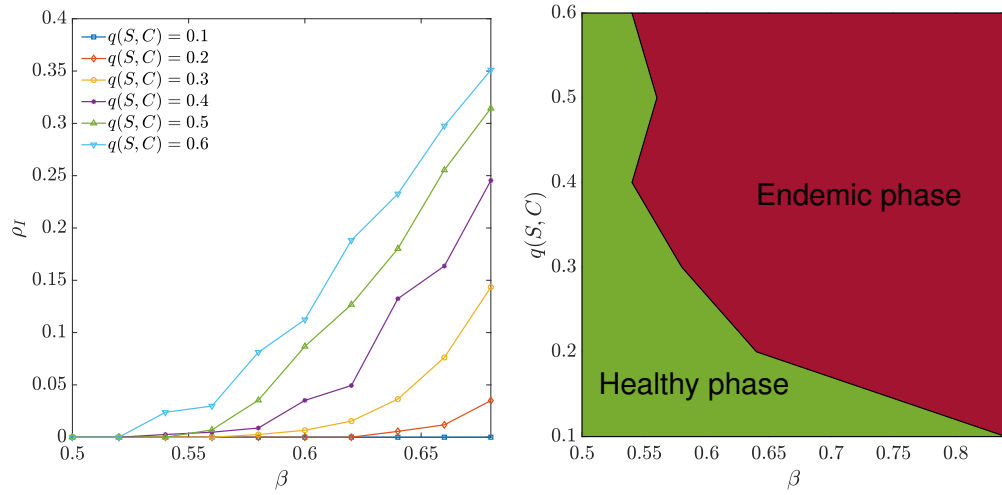


FIGURE 7.7: Bounded confidence parameter $\Delta = 2$. Final normalized number of infected nodes for different values of β and $q(S, C)$ (left) and visual representation of the phase diagram (right). Coupled lognormal-Weibull distributions for social and contacts networks.

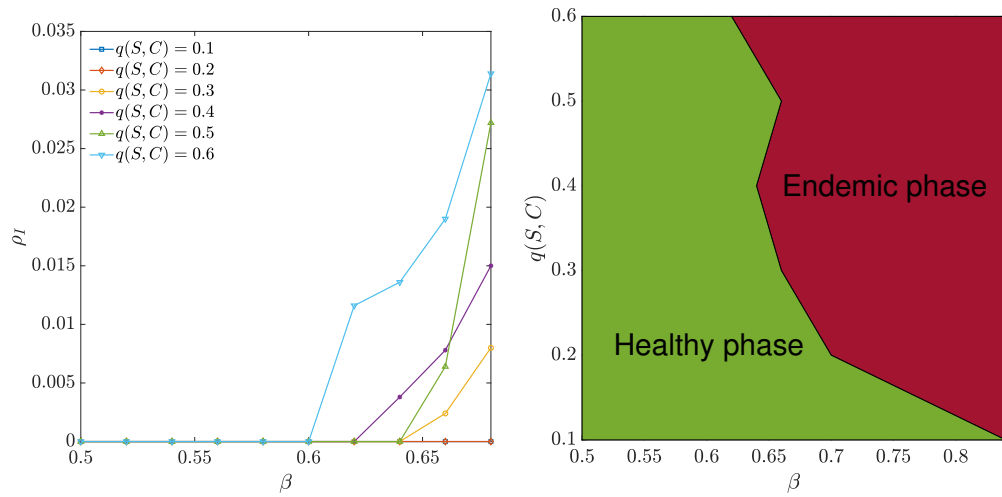


FIGURE 7.8: Bounded confidence parameter $\Delta = 0.5$. Final normalized number of infected nodes for different values of β and $q(S, C)$ (left) and visual representation of the phase diagram (right). Coupled lognormal-Weibull distributions for social and contacts networks.

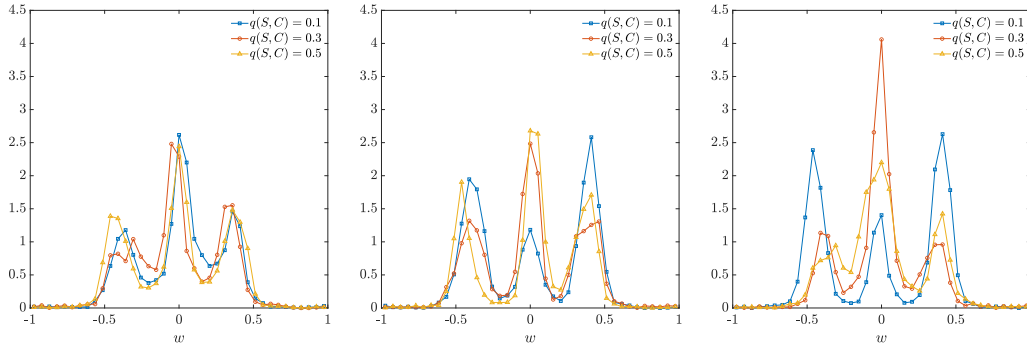


FIGURE 7.9: Bounded confidence parameter $\Delta = 0.5$. Final time opinion distributions for various values of the parameter $q(S, C)$, in the case $\beta = 0.5$ (left), $\beta = 0.58$ (center), $\beta = 0.66$ (right).

7.5 Conclusion

In this work, we have investigated the coupled dynamics of opinion formation and epidemic spreading on multiplex networks, explicitly distinguishing between social interactions and physical contacts. Starting from a microscopic agent-based description, we have proposed a mesoscopic kinetic formulation based on Boltzmann-type equations, and subsequently obtained a Fokker–Planck approximation in the quasi-invariant opinion limit mimicking the multiple network structure of the corresponding microscopic approach. The two proposed models allow for a consistent connection between individual-level interactions and macroscopic observable, providing a flexible framework to analyze the interplay between social behavior and epidemic dynamics.

A key outcome of our analysis is the identification of the overlap between social and physical networks as a crucial control parameter. Numerical simulations show that varying the overlap factor can induce a phase transition between a disease-free (healthy) state and an endemic regime. In particular, lower overlap levels tend to increase the effective epidemic threshold, delaying or suppressing disease spread.

Several extensions of the present work are worth exploring. From a modeling perspective, allowing the network layers to evolve in time or considering more complex epidemic compartments could further increase realism. From an analytical standpoint, an investigation of the passage from the microscopic to the kinetic description would be of great interest.

Appendix A

Derivation of fractional differential system schemes

Here we write the formulation of all schemes used in the chapter 3.

Fractional Forward Euler Scheme (FFE)

The numerical scheme we proposed here is presented in [245]. Suppose we take the initial value problem (IVP)

$$\begin{cases} {}^C D_0^\aleph u(t) = G(t, u(t)), & 0 < \aleph \leq 1 \\ u(0) = u_0 \geq 0, & 0 < t \leq T. \end{cases} \quad (\text{A.1})$$

where the given function $G(t, u(t))$ is solved in the Caputo sense. $u(t)$ is the exact solution, $\aleph$ is the fractional order of derivative, and T is the final time. Given function $G(t, u(t))$ satisfied some smooth characteristics [175]. It's necessary to understand that this procedure will not provide the function $y(t)$, which satisfies the IVP (A.1). Rather, we will calculate an estimate of it at as many points as necessary. The time interval $[0, T]$ is split into sub-intervals $[t_i, t_{i+1}]$ with step size $h=T/N$ using the nodes $t_i = hi$ where $i = 1, 2, 3, \dots, n$.

Apply ${}^C D_0^{-\aleph}$ on IVP (A.1), we get

$$u(t) = u_0 + {}^C D_0^{-\aleph} u(t), \quad (\text{A.2})$$

${}^C D_0^{-\aleph} u(t)$ is evaluated by left fractional integral formula s.t

$$u(t_{n+1}) = u_0 + \frac{h^\aleph}{\Gamma(1+\aleph)} \sum_{i=0}^n v_{i,n+1} G(t_i, u(i)), \quad (\text{A.3})$$

where

$$v_{i,n+1} = \left[(n+1-i)^\aleph - (n-i)^\aleph \right].$$

Now, we implement the application of this numerical scheme for our model (3.22) given below as

$$\begin{cases} {}^C D_t^\aleph S(t) = a_1 + a_2 a_3 R(t) - a_{12} a_7 A(t) S(t) - (a_6 + a_4) S(t), \\ {}^C D_t^\aleph E(t) = a_{12} a_7 A(t) S(t) - (a_9 + a_4) E(t), \\ {}^C D_t^\aleph A(t) = a_{14} a_9 E(t) - (a_4 + a_5 + a_{11}) A(t), \\ {}^C D_t^\aleph D(t) = a_5 A(t) + a_{11} (1 - a_{10}) A(t) - (a_8 + a_{13} + a_4) D(t), \\ {}^C D_t^\aleph R(t) = (1 - a_{14}) a_9 E(t) + a_8 D(t) + a_{11} a_{10} A(t) - (a_4 + a_3) R(t), \\ {}^C D_t^\aleph Q(t) = a_6 S(t) + (1 - a_2) a_3 R(t) - a_4 Q(t). \end{cases}$$

For this purpose, we formulate the model, which is given below

$$\begin{cases} {}^C D_t^\aleph S(t) = J_1(S, E, A, D, R, Q), \\ {}^C D_t^\aleph E(t) = J_2(S, E, A, D, R, Q), \\ {}^C D_t^\aleph A(t) = J_3(S, E, A, D, R, Q), \\ {}^C D_t^\aleph D(t) = J_4(S, E, A, D, R, Q), \\ {}^C D_t^\aleph R(t) = J_5(S, E, A, D, R, Q), \\ {}^C D_t^\aleph Q(t) = J_6(S, E, A, D, R, Q). \end{cases}$$

Implementing the scheme, we get

$$\begin{aligned} S(t_{n+1}) &= S_0 + \frac{h^\aleph}{\Gamma(1+\aleph)} \sum_{i=0}^n \Pi_{i,n+1} J_1(t_i, S, E, A, D, R, Q), \\ E(t_{n+1}) &= E_0 + \frac{h^\aleph}{\Gamma(1+\aleph)} \sum_{i=0}^n \Pi_{i,n+1} J_2(t_i, S, E, A, D, R, Q), \\ A(t_{n+1}) &= A_0 + \frac{h^\aleph}{\Gamma(1+\aleph)} \sum_{i=0}^n \Pi_{i,n+1} J_3(t_i, S, E, A, D, R, Q), \\ D(t_{n+1}) &= D_0 + \frac{h^\aleph}{\Gamma(1+\aleph)} \sum_{i=0}^n \Pi_{i,n+1} J_4(t_i, S, E, A, D, R, Q), \\ R(t_{n+1}) &= R_0 + \frac{h^\aleph}{\Gamma(1+\aleph)} \sum_{i=0}^n \Pi_{i,n+1} J_5(t_i, S, E, A, D, R, Q), \\ Q(t_{n+1}) &= Q_0 + \frac{h^\aleph}{\Gamma(1+\aleph)} \sum_{i=0}^n \Pi_{i,n+1} J_6(t_i, S, E, A, D, R, Q), \end{aligned}$$

where

$$\Pi_{i,n+1} = \left[(n+1-i)^\aleph - (n-i)^\aleph \right].$$

Adams-Bashforth Molten Scheme (ABM)

This section looks at the numerical approach for the suggested model (3.22). To find the numerical solution of the model, we used the generalized Adams-Bashforth-Moulton method [213]. We build a numerical scheme for solving nonlinear fractional-order differential equations using the predictor-corrector technique. To find the approximate solution using this method, consider the following nonlinear fractional differential equation.

$${}_0^C D_t^\aleph \phi(t) = u(t, \phi(t)), \quad 0 \leq t \leq T, \quad (\text{A.4})$$

along with the IC's

$$\phi^{(j)}(0) = \phi_0^j, \quad j = 0, 1, 2, 3, \dots, [\aleph] - 1.$$

The fractional integral operator is applied on the eq.(A.4), then the solution $\phi(t)$ can be determined by the equation given below

$$\phi(t) = \sum_{j=0}^{[\aleph]-1} \frac{\phi_0^j}{j!} t^j + \frac{1}{\Gamma(\aleph)} \int_0^t (t-s)^{\aleph-1} u(s, \phi(s)) ds, \quad (\text{A.5})$$

which is equal to the Volterra integral equation. Diethelm et al. [94] integrated (A.5) using the predictor-corrector method based on the Adams-Bashforth-Moulton algorithm. Letting $h = \frac{T}{N}$, $t_n = nh$, and $n = 0, 1, 2, \dots, N \in \mathbb{Z}^+$, the equation (A.5) can be discretized as follows

$$\phi_h(t_{n+1}) = \sum_{j=0}^{[\aleph]-1} \frac{\phi_0^j}{j!} t_{n+1}^j + \frac{h^\aleph}{\Gamma(\aleph+2)} u(t_{n+1}, \phi_h^z(t_{n+1})) + \frac{h^\aleph}{\Gamma(\aleph+2)} \sum_{m=0}^n c_{m,n+1} u(t_m, \phi_h(t_m)), \quad (\text{A.6})$$

where

$$c_{m,n+1} = \begin{cases} n^{\aleph+1} - (n-\aleph)(n+1)^\aleph, & \text{if } m = 0, \\ (n-m+2)^{\aleph+1} + (n-m)^{\aleph+1} - 2(n-m+1)^{\aleph+1}, & \text{if } 0 < m \leq n, \\ 1, & \text{if } m = n+1, \end{cases} \quad (\text{A.7})$$

and the predicted value $\phi_h^z(t_{n+1})$ determined by

$$\phi_h^z(t_{n+1}) = \sum_{j=0}^{[\aleph]-1} \frac{\phi_0^j}{j!} t_{n+1}^j + \frac{h^\aleph}{\Gamma(\aleph)} \sum_{m=0}^n d_{m,n+1} u(t_m, \phi_h(t_m)), \quad (\text{A.8})$$

with

$$d_{m,n+1} = \frac{h^\aleph}{\aleph} \left((n+1-m)^\aleph \right) \quad (\text{A.9})$$

$$- \left((n-m)^\aleph \right). \quad (\text{A.10})$$

The error estimate is

$$\max_{m=0,1,2,\dots,l} |\phi(t_m) - \phi_h(t_m)| = O(h^Z),$$

in which $Z = \min(2, 1 + \aleph)$. We utilize the application of the Adams-Bashforth-Moulton method to solve numerically the nonlinear Caputo fractional order model (3.22) given as

$$\begin{cases} {}^C D_t^\aleph S(t) = a_1 + a_2 a_3 R(t) - a_{12} a_7 A(t) S(t) - (a_6 + a_4) S(t), \\ {}^C D_t^\aleph E(t) = a_{12} a_7 A(t) S(t) - (a_9 + a_4) E(t), \\ {}^C D_t^\aleph A(t) = a_{14} a_9 E(t) - (a_4 + a_5 + a_{11}) A(t), \\ {}^C D_t^\aleph D(t) = a_5 A(t) + a_{11} (1 - a_{10}) A(t) - (a_8 + a_{13} + a_4) D(t), \\ {}^C D_t^\aleph R(t) = (1 - a_{14}) a_9 E(t) + a_8 D(t) + a_{11} a_{10} A(t) - (a_4 + a_3) R(t), \\ {}^C D_t^\aleph Q(t) = a_6 S(t) + (1 - a_2) a_3 R(t) - a_4 Q(t). \end{cases}$$

Using the ABM approach, the numerical scheme for the suggested model (3.22) is provided as follows:

$$\begin{aligned} S(t) &= S_0 + \frac{h^\aleph}{\Gamma(\aleph + 2)} \Pi_S(t_{p+1}, S_h^k(t_{p+1}), E_h^k(t_{p+1}), A_h^k(t_{p+1}), D_h^k(t_{p+1}), R_h^k(t_{p+1}), Q_h^k(t_{p+1})) + \\ &\quad \frac{h^\aleph}{\Gamma(\aleph + 2)} \sum_{m=0}^p a_{m,p+1} \Pi_S(t_m, S_h(t_m), E_h(t_m), A_h(t_m), D_h(t_m), R_h(t_m), Q_h(t_m)), \\ E(t) &= E_0 + \frac{h^\aleph}{\Gamma(\aleph + 2)} \Pi_E(t_{p+1}, S_h^k(t_{p+1}), E_h^k(t_{p+1}), A_h^k(t_{p+1}), D_h^k(t_{p+1}), R_h^k(t_{p+1}), Q_h^k(t_{p+1})) + \\ &\quad \frac{h^\aleph}{\Gamma(\aleph + 2)} \sum_{m=0}^p a_{m,p+1} \Pi_E(t_m, S_h(t_m), E_h(t_m), A_h(t_m), D_h(t_m), R_h(t_m), Q_h(t_m)), \\ A(t) &= A_0 + \frac{h^\aleph}{\Gamma(\aleph + 2)} \Pi_A(t_{p+1}, S_h^k(t_{p+1}), E_h^k(t_{p+1}), A_h^k(t_{p+1}), D_h^k(t_{p+1}), R_h^k(t_{p+1}), Q_h^k(t_{p+1})) + \\ &\quad \frac{h^\aleph}{\Gamma(\aleph + 2)} \sum_{m=0}^p a_{m,p+1} \Pi_A(t_m, S_h(t_m), E_h(t_m), A_h(t_m), D_h(t_m), R_h(t_m), Q_h(t_m)), \\ D(t) &= D_0 + \frac{h^\aleph}{\Gamma(\aleph + 2)} \Pi_D(t_{p+1}, S_h^k(t_{p+1}), E_h^k(t_{p+1}), A_h^k(t_{p+1}), D_h^k(t_{p+1}), R_h^k(t_{p+1}), Q_h^k(t_{p+1})) + \\ &\quad \frac{h^\aleph}{\Gamma(\aleph + 2)} \sum_{m=0}^p a_{m,p+1} \Pi_D(t_m, S_h(t_m), E_h(t_m), A_h(t_m), D_h(t_m), R_h(t_m), Q_h(t_m)), \\ R(t) &= R_0 + \frac{h^\aleph}{\Gamma(\aleph + 2)} \Pi_R(t_{p+1}, S_h^k(t_{p+1}), E_h^k(t_{p+1}), A_h^k(t_{p+1}), D_h^k(t_{p+1}), R_h^k(t_{p+1}), Q_h^k(t_{p+1})) + \\ &\quad \frac{h^\aleph}{\Gamma(\aleph + 2)} \sum_{m=0}^p a_{m,p+1} \Pi_R(t_m, S_h(t_m), E_h(t_m), A_h(t_m), D_h(t_m), R_h(t_m), Q_h(t_m)), \\ Q(t) &= Q_0 + \frac{h^\aleph}{\Gamma(\aleph + 2)} \Pi_Q(t_{p+1}, S_h^k(t_{p+1}), E_h^k(t_{p+1}), A_h^k(t_{p+1}), D_h^k(t_{p+1}), R_h^k(t_{p+1}), Q_h^k(t_{p+1})) + \\ &\quad \frac{h^\aleph}{\Gamma(\aleph + 2)} \sum_{m=0}^p a_{m,p+1} \Pi_Q(t_m, S_h(t_m), E_h(t_m), A_h(t_m), D_h(t_m), R_h(t_m), Q_h(t_m)), \end{aligned}$$

with

$$a_{m,p+1} = \begin{cases} p^{\aleph+1} - (p - \aleph)(p + 1)^{\aleph}, & \text{if } m = 0, \\ (p - m + 2)^{\aleph+1} + (p - m)^{\aleph+1} - 2(p - m + 1)^{\aleph+1}, & \text{if } 0 < m \leq p, \\ 1, & \text{if } m = p + 1. \end{cases}$$

where

$$\begin{aligned} S_h^k(t_{p+1}) &= S_0 + \frac{1}{\Gamma(\aleph)} \sum_{m,p+1} b_{m,p+1} \Pi_S(t_m, S_h(t_m), E_h(t_m), A_h(t_m), D_h(t_m), R_h(t_m), Q_h(t_m)), \\ E_h^k(t_{p+1}) &= E_0 + \frac{1}{\Gamma(\aleph)} \sum_{m,p+1} b_{m,p+1} \Pi_E(t_m, S_h(t_m), E_h(t_m), A_h(t_m), D_h(t_m), R_h(t_m), Q_h(t_m)), \\ A_h^k(t_{p+1}) &= A_0 + \frac{1}{\Gamma(\aleph)} \sum_{m,p+1} b_{m,p+1} \Pi_A(t_m, S_h(t_m), E_h(t_m), A_h(t_m), D_h(t_m), R_h(t_m), Q_h(t_m)), \\ D_h^k(t_{p+1}) &= D_0 + \frac{1}{\Gamma(\aleph)} \sum_{m,p+1} b_{m,p+1} \Pi_D(t_m, S_h(t_m), E_h(t_m), A_h(t_m), D_h(t_m), R_h(t_m), Q_h(t_m)), \\ R_h^k(t_{p+1}) &= R_0 + \frac{1}{\Gamma(\aleph)} \sum_{m,p+1} b_{m,p+1} \Pi_R(t_m, S_h(t_m), E_h(t_m), A_h(t_m), D_h(t_m), R_h(t_m), Q_h(t_m)), \\ Q_h^k(t_{p+1}) &= Q_0 + \frac{1}{\Gamma(\aleph)} \sum_{m,p+1} b_{m,p+1} \Pi_Q(t_m, S_h(t_m), E_h(t_m), A_h(t_m), D_h(t_m), R_h(t_m), Q_h(t_m)), \end{aligned}$$

with

$$b_{m,p+1} = \frac{h^{\aleph}}{\aleph} \left((p + 1 - m)^{\aleph} - (p - m)^{\aleph} \right).$$

Further, the quantities

$$\begin{aligned} &\Pi_S(t_m, S_h(t_m), E_h(t_m), A_h(t_m), D_h(t_m), R_h(t_m), Q_h(t_m)), \\ &\Pi_E(t_m, S_h(t_m), E_h(t_m), A_h(t_m), D_h(t_m), R_h(t_m), Q_h(t_m)), \\ &\Pi_A(t_m, S_h(t_m), E_h(t_m), A_h(t_m), D_h(t_m), R_h(t_m), Q_h(t_m)), \\ &\Pi_D(t_m, S_h(t_m), E_h(t_m), A_h(t_m), D_h(t_m), R_h(t_m), Q_h(t_m)), \\ &\Pi_R(t_m, S_h(t_m), E_h(t_m), A_h(t_m), D_h(t_m), R_h(t_m), Q_h(t_m)), \end{aligned}$$

and

$$\Pi_Q(t_m, S_h(t_m), E_h(t_m), A_h(t_m), D_h(t_m), R_h(t_m), Q_h(t_m))$$

are computed from the following functions

$$\begin{aligned} \Pi_S &= a_1 + a_2 a_3 R(t) - a_{12} a_7 A(t) S(t) - (a_6 + a_4) S(t), \\ \Pi_E &= a_{12} a_7 A(t) S(t) - (a_9 + a_4) E(t), \\ \Pi_A &= a_{14} a_9 E(t) - (a_4 + a_5 + a_{11}) A(t), \\ \Pi_D &= a_5 A(t) + a_{11} (1 - a_{10}) A(t) - (a_8 + a_{13} + a_4) D(t), \\ \Pi_R &= (1 - a_{14}) a_9 E(t) + a_8 D(t) + a_{11} a_{10} A(t) \\ &\quad - (a_4 + a_3) R(t), \\ \Pi_Q &= a_6 S(t) + (1 - a_2) a_3 R(t) - a_4 Q(t). \end{aligned} \tag{A.11}$$

In addition, the quantities

$$\begin{aligned} & \Pi_S \left(t_{p+1}, S_h^k(t_{p+1}), E_h^k(t_{p+1}), A_h^k(t_{p+1}), D_h^k(t_{p+1}), R_h^k(t_{p+1}), Q_h^k(t_{p+1}) \right), \\ & \Pi_E \left(t_{p+1}, S_h^k(t_{p+1}), E_h^k(t_{p+1}), A_h^k(t_{p+1}), D_h^k(t_{p+1}), R_h^k(t_{p+1}), Q_h^k(t_{p+1}) \right), \\ & \Pi_A \left(t_{p+1}, S_h^k(t_{p+1}), E_h^k(t_{p+1}), A_h^k(t_{p+1}), D_h^k(t_{p+1}), R_h^k(t_{p+1}), Q_h^k(t_{p+1}) \right), \\ & \Pi_D \left(t_{p+1}, S_h^k(t_{p+1}), E_h^k(t_{p+1}), A_h^k(t_{p+1}), D_h^k(t_{p+1}), R_h^k(t_{p+1}), Q_h^k(t_{p+1}) \right), \\ & \Pi_R \left(t_{p+1}, S_h^k(t_{p+1}), E_h^k(t_{p+1}), A_h^k(t_{p+1}), D_h^k(t_{p+1}), R_h^k(t_{p+1}), Q_h^k(t_{p+1}) \right), \\ & \Pi_Q \left(t_{p+1}, S_h^k(t_{p+1}), E_h^k(t_{p+1}), A_h^k(t_{p+1}), D_h^k(t_{p+1}), R_h^k(t_{p+1}), Q_h^k(t_{p+1}) \right), \end{aligned}$$

are computed from equation (A.11) respectively, at the points $t_{p+1}, p = 1, 2, 3, \dots, m$.

Toufik-Atangana Scheme (TAS)

In this subsection, we present a numerical approach that is utilized in [279] to solve the differential equations of type ABC fractional derivatives. Consider the given equation

$${}_0^{ABC}D_t^{\aleph} h(t) = g(t, h(t)), \quad h(0) = 0. \quad (\text{A.12})$$

The fractional integral form of the above equation is obtained using the fundamental theorem of calculus, given below:

$$h(t) - h(0) = \frac{1 - \aleph}{\mathbb{AB}(\aleph)} g(t, h(t)) + \frac{\aleph}{\mathbb{AB}(\aleph)\Gamma(\aleph)} \int_0^t g(u, h(u))(t - u)^{\aleph-1} du. \quad (\text{A.13})$$

At point $t = t_{n+1}, n = 0, 1, 2, \dots$ the above equation becomes

$$h(t_{n+1}) - h(0) = \frac{1 - \aleph}{\mathbb{AB}(\aleph)} g(t_{n+1}, h(t)) + \frac{\aleph}{\mathbb{AB}(\aleph)\Gamma(\aleph)} \int_0^{t_{n+1}} g(u, h(u))(t_{n+1} - u)^{\aleph-1} d\aleph. \quad (\text{A.14})$$

Using a two-step Lagrangian polynomial for $g(u, h(u))$ on the interval $[t_m, t_{m+1}]$, we get the following expression:

$$\begin{aligned} K_m(u) &= g(u, h(u)) \\ &= \frac{u - t_{m-1}}{t_m - t_{m-1}} g(t_m, h(t_m)) - \frac{u - t_m}{t_m - t_{m-1}} g(t_{m-1}, h(t_{m-1})) \\ &= \frac{g(t_m, h_m)}{h} (u - t_{m-1}) - \frac{g(t_{m-1}, h_{m-1})}{h} (u - t_m). \end{aligned} \quad (\text{A.15})$$

So, (A.14) becomes

$$\begin{aligned} h_{n+1} &= h(0) + \frac{1 - \aleph}{\mathbb{AB}(\aleph)} g(t_n, h(t_n)) + \frac{\aleph}{\mathbb{AB}(\aleph)\Gamma(\aleph)} \sum_{m=0}^n \left(\frac{g(t_m, h_m)}{h} \int_{t_m}^{t_{m+1}} (u - t_{m-1}) \right. \\ &\quad \left. (t_{n+1} - u)^{\aleph-1} du - \frac{g(t_{m-1}, h_{m-1})}{h} \int_{t_m}^{t_{m+1}} (u - t_m) (t_{n+1} - u)^{\aleph-1} du \right). \end{aligned} \quad (\text{A.16})$$

After simplification, we get,

$$h_{n+1} = h(0) + \frac{1-\aleph}{\mathbb{AB}(\aleph)} g(t_n, h(t_n)) + \frac{\aleph}{\mathbb{AB}(\aleph)} \sum_{m=0}^n \left(\frac{h^{\aleph} g(t_m, h_m)}{\Gamma(\aleph+2)} ((n+1-m)^{\aleph} \right. \\ \left. (n-m+2+\aleph) - (n-m)^{\aleph} (n-m+2+2\aleph)) - \frac{h^{\aleph} g(t_{m-1}, h_{m-1})}{\Gamma(\aleph+2)} \right. \\ \left. ((n+1-m)^{\aleph} - (n-m)^{\aleph} (n-m+1+\aleph)) \right) + R_n^{\aleph}. \quad (\text{A.17})$$

where $R_n^{\aleph}$ is the remainder term given as

$$R_n^{\aleph} = \frac{\aleph}{\mathbb{AB}(\aleph)\Gamma(\aleph)} \sum_{m=0}^n \int_{t_m}^{t_{m+1}} \frac{(u-t_m)(u-t_{m-1})}{2!} \frac{\partial^2}{\partial u^2} [g(u, h(u))]_{u=\xi_u} (t_{n+1}-u)^{\aleph-1} du. \quad (\text{A.18})$$

The model (3.22) is given as

$$\begin{cases} {}^{\text{ABC}}_t D_0^{\aleph} S(t) = a_1 + a_2 a_3 R(t) - a_{12} a_7 A(t) S(t) - (a_6 + a_4) S(t), \\ {}^{\text{ABC}}_t D_0^{\aleph} E(t) = a_{12} a_7 A(t) S(t) - (a_9 + a_4) E(t), \\ {}^{\text{ABC}}_t D_0^{\aleph} A(t) = a_{14} a_9 E(t) - (a_4 + a_5 + a_{11}) A(t), \\ {}^{\text{ABC}}_t D_0^{\aleph} D(t) = a_5 A(t) + a_{11} (1 - a_{10}) A(t) - (a_8 + a_{13} + a_4) D(t), \\ {}^{\text{ABC}}_t D_0^{\aleph} R(t) = (1 - a_{14}) a_9 E(t) + a_8 D(t) + a_{11} a_{10} A(t) - (a_4 + a_3) R(t), \\ {}^{\text{ABC}}_t D_0^{\aleph} Q(t) = a_6 S(t) + (1 - a_2) a_3 R(t) - a_4 Q(t). \end{cases}$$

For this purpose, we look at the model in the form below

$$\begin{cases} {}^{\text{ABC}}_t D_0^{\aleph} S(t) = F_1(S, E, A, D, R, Q), \\ {}^{\text{ABC}}_t D_0^{\aleph} E(t) = F_2(S, E, A, D, R, Q), \\ {}^{\text{ABC}}_t D_0^{\aleph} A(t) = F_3(S, E, A, D, R, Q), \\ {}^{\text{ABC}}_t D_0^{\aleph} D(t) = F_4(S, E, A, D, R, Q), \\ {}^{\text{ABC}}_t D_0^{\aleph} R(t) = F_5(S, E, A, D, R, Q), \\ {}^{\text{ABC}}_t D_0^{\aleph} Q(t) = F_6(S, E, A, D, R, Q). \end{cases}$$

By implementing the numerical technique at point $t = t_{p+1}$, $p = 1, 2, 3, \dots$ with sub interval $[t_q, t_{q+1}]$, we have model becomes

$$S_{p+1} = S(0) + \frac{1-\aleph}{\mathbb{AB}(\aleph)} F_1(t_p, y(t_p)) + \frac{\aleph}{\mathbb{AB}(\aleph)} \sum_{q=0}^p \left(\frac{h^{\aleph} F_1(t_q, y_q)}{\Gamma(\aleph+2)} ((p+1-q)^{\aleph} (p-q+2+\aleph) \right. \\ \left. - (p-q)^{\aleph} (p-q+2+2\aleph)) - \frac{h^{\aleph} F_1(t_{q-1}, y_{q-1})}{\Gamma(\aleph+2)} ((p+1-q)^{\aleph} \right. \\ \left. - (p-q)^{\aleph} (p-q+1+\aleph)) \right), \\ E_{p+1} = E(0) + \frac{1-\aleph}{\mathbb{AB}(\aleph)} F_2(t_p, y(t_p)) + \frac{\aleph}{\mathbb{AB}(\aleph)} \sum_{q=0}^p \left(\frac{h^{\aleph} F_2(t_q, y_q)}{\Gamma(\aleph+2)} ((p+1-q)^{\aleph} (p-q+2+\aleph) \right. \\ \left. - (p-q)^{\aleph} (p-q+2+2\aleph)) - \frac{h^{\aleph} F_2(t_{q-1}, y_{q-1})}{\Gamma(\aleph+2)} ((p+1-q)^{\aleph} \right. \\ \left. - (p-q)^{\aleph} (p-q+1+\aleph)) \right),$$

$$\begin{aligned}
A_{p+1} &= A(0) + \frac{1-\aleph}{\mathbb{AB}(\aleph)} F_3(t_p, y(t_p)) + \frac{\aleph}{\mathbb{AB}(\aleph)} \sum_{q=0}^p \left(\frac{h^\aleph F_3(t_q, y_q)}{\Gamma(\aleph+2)} ((p+1-q)^\aleph (p-q+2+\aleph) \right. \\
&\quad \left. - (p-q)^\aleph (p-q+2+2\aleph)) - \frac{h^\aleph F_3(t_{q-1}, y_{q-1})}{\Gamma(\aleph+2)} ((p+1-q)^\aleph \right. \\
&\quad \left. - (p-q)^\aleph (p-q+1+\aleph)) \right), \\
D_{p+1} &= D(0) + \frac{1-\aleph}{\mathbb{AB}(\aleph)} F_4(t_p, y(t_p)) + \frac{\aleph}{\mathbb{AB}(\aleph)} \sum_{q=0}^p \left(\frac{h^\aleph F_4(t_q, y_q)}{\Gamma(\aleph+2)} ((p+1-q)^\aleph (p-q+2+\aleph) \right. \\
&\quad \left. - (p-q)^\aleph (p-q+2+2\aleph)) - \frac{h^\aleph F_4(t_{q-1}, y_{q-1})}{\Gamma(\aleph+2)} ((p+1-q)^\aleph \right. \\
&\quad \left. - (p-q)^\aleph (p-q+1+\aleph)) \right), \\
R_{p+1} &= R(0) + \frac{1-\aleph}{\mathbb{AB}(\aleph)} F_5(t_p, y(t_p)) + \frac{\aleph}{\mathbb{AB}(\aleph)} \sum_{q=0}^p \left(\frac{h^\aleph F_5(t_q, y_q)}{\Gamma(\aleph+2)} ((p+1-q)^\aleph (p-q+2+\aleph) \right. \\
&\quad \left. - (p-q)^\aleph (p-q+2+2\aleph)) - \frac{h^\aleph F_5(t_{q-1}, y_{q-1})}{\Gamma(\aleph+2)} ((p+1-q)^\aleph \right. \\
&\quad \left. - (p-q)^\aleph (p-q+1+\aleph)) \right), \\
Q_{p+1} &= Q(0) + \frac{1-\aleph}{\mathbb{AB}(\aleph)} F_6(t_p, y(t_p)) + \frac{\aleph}{\mathbb{AB}(\aleph)} \sum_{q=0}^p \left(\frac{h^\aleph F_6(t_q, y_q)}{\Gamma(\aleph+2)} ((p+1-q)^\aleph (p-q+2+\aleph) \right. \\
&\quad \left. - (p-q)^\aleph (p-q+2+2\aleph)) - \frac{h^\aleph F_6(t_{q-1}, y_{q-1})}{\Gamma(\aleph+2)} ((p+1-q)^\aleph \right. \\
&\quad \left. - (p-q)^\aleph (p-q+1+\aleph)) \right).
\end{aligned}$$

Adams type Predictor-Corrector Scheme (ATPC)

In this section, we evaluate the approximate numerical results for the model. We develop a numerical technique as used in [18]. For this objective, the numerical iterative formula is designed utilizing the Adams-type predictor-corrector rule for Atangana-Baleanu ($\mathbb{AB}$) fractional integral operator. The model is given as

$$\begin{cases}
{}_t^{\mathbb{ABC}} D_0^\aleph S(t) = a_1 + a_2 a_3 R(t) - a_{12} a_7 A(t) S(t) - (a_6 + a_4) S(t), \\
{}_t^{\mathbb{ABC}} D_0^\aleph E(t) = a_{12} a_7 A(t) S(t) - (a_9 + a_4) E(t), \\
{}_t^{\mathbb{ABC}} D_0^\aleph A(t) = a_{14} a_9 E(t) - (a_4 + a_5 + a_{11}) A(t), \\
{}_t^{\mathbb{ABC}} D_0^\aleph D(t) = a_5 A(t) + a_{11} (1 - a_{10}) A(t) - (a_8 + a_{13} + a_4) D(t), \\
{}_t^{\mathbb{ABC}} D_0^\aleph R(t) = (1 - a_{14}) a_9 E(t) + a_8 D(t) + a_{11} a_{10} A(t) - (a_4 + a_3) R(t), \\
{}_t^{\mathbb{ABC}} D_0^\aleph Q(t) = a_6 S(t) + (1 - a_2) a_3 R(t) - a_4 Q(t).
\end{cases}$$

For this purpose, we look at the model in the form below.

$$\begin{cases} {}_t^{\text{ABC}}D_0^\aleph S(t) = H_1(S, E, A, D, R, Q), \\ {}_t^{\text{ABC}}D_0^\aleph E(t) = H_2(S, E, A, D, R, Q), \\ {}_t^{\text{ABC}}D_0^\aleph A(t) = H_3(S, E, A, D, R, Q), \\ {}_t^{\text{ABC}}D_0^\aleph D(t) = H_4(S, E, A, D, R, Q), \\ {}_t^{\text{ABC}}D_0^\aleph R(t) = H_5(S, E, A, D, R, Q), \\ {}_t^{\text{ABC}}D_0^\aleph Q(t) = H_6(S, E, A, D, R, Q). \end{cases}$$

Applying the ABC–fractional integral operator on both sides, we get

$$\begin{aligned} S(t) &= S_0 + \frac{1-\aleph}{\text{AB}(\aleph)} H_1(t, S, E, A, D, R, Q) \\ &\quad + \frac{\aleph}{\text{AB}(\aleph)\Gamma(\aleph)} \int_0^t (t-s)^{\aleph-1} H_1(u, S, E, A, D, R, Q) du, \end{aligned}$$

$$\begin{aligned} E(t) &= E_0 + \frac{1-\aleph}{\text{AB}(\aleph)} H_2(t, S, E, A, D, R, Q) \\ &\quad + \frac{\aleph}{\text{AB}(\aleph)\Gamma(\aleph)} \int_0^t (t-s)^{\aleph-1} H_2(u, S, E, A, D, R, Q) du, \end{aligned}$$

$$\begin{aligned} A(t) &= A_0 + \frac{1-\aleph}{\text{AB}(\aleph)} H_3(t, S, E, A, D, R, Q) \\ &\quad + \frac{\aleph}{\text{AB}(\aleph)\Gamma(\aleph)} \int_0^t (t-s)^{\aleph-1} H_3(u, S, E, A, D, R, Q) du, \end{aligned}$$

$$\begin{aligned} D(t) &= D_0 + \frac{1-\aleph}{\text{AB}(\aleph)} H_4(t, S, E, A, D, R, Q) \\ &\quad + \frac{\aleph}{\text{AB}(\aleph)\Gamma(\aleph)} \int_0^t (t-s)^{\aleph-1} H_4(u, S, E, A, D, R, Q) du, \end{aligned}$$

$$\begin{aligned} R(t) &= R_0 + \frac{1-\aleph}{\text{AB}(\aleph)} H_5(t, S, E, A, D, R, Q) \\ &\quad + \frac{\aleph}{\text{AB}(\aleph)\Gamma(\aleph)} \int_0^t (t-s)^{\aleph-1} H_5(u, S, E, A, D, R, Q) du, \end{aligned}$$

$$\begin{aligned} Q(t) &= Q_0 + \frac{1-\aleph}{\text{AB}(\aleph)} H_6(t, S, E, A, D, R, Q) \\ &\quad + \frac{\aleph}{\text{AB}(\aleph)\Gamma(\aleph)} \int_0^t (t-s)^{\aleph-1} H_6(u, S, E, A, D, R, Q) du. \end{aligned}$$

Using an adaptation of the Adams type predictor-corrector scheme tool, as shown in [18], to get approximate values of the system's right-hand side. In the algorithm's first step, it is assumed that the closed interval $[0, T]$ contains the solution, and this interval setting is $h = T/N$, $t_n = hn$ ($n = 0, 1, 2, \dots, N$). Thus, the variable order

integral form corrector schemes of the $\mathbb{ABC}$ – fractional derivative are provided as follows

$$\begin{aligned}
S_{n+1} &= S_0 + \frac{(1 - \aleph) h^\aleph}{\mathbb{AB}(\aleph)\Gamma(\aleph + 2)} H_1(t_{n+1}, S_{n+1}^p, E_{n+1}^p, A_{n+1}^p, D_{n+1}^p, R_{n+1}^p, Q_{n+1}^p) \\
&\quad + \frac{\aleph h^\aleph}{\mathbb{AB}(\aleph)\Gamma(\aleph + 2)} \sum_{q=0}^n \Xi_{q,n+1} H_1(t_q, S_q, E_q, A_q, D_q, R_q, Q_q), \\
E_{n+1} &= E_0 + \frac{(1 - \aleph) h^\aleph}{\mathbb{AB}(\aleph)\Gamma(\aleph + 2)} H_2(t_{n+1}, S_{n+1}^p, E_{n+1}^p, A_{n+1}^p, D_{n+1}^p, R_{n+1}^p, Q_{n+1}^p) \\
&\quad + \frac{\aleph h^\aleph}{\mathbb{AB}(\aleph)\Gamma(\aleph + 2)} \sum_{q=0}^n \Xi_{q,n+1} H_2(t_q, S_q, E_q, A_q, D_q, R_q, Q_q), \\
A_{n+1} &= A_0 + \frac{(1 - \aleph) h^\aleph}{\mathbb{AB}(\aleph)\Gamma(\aleph + 2)} H_3(t_{n+1}, S_{n+1}^p, E_{n+1}^p, A_{n+1}^p, D_{n+1}^p, R_{n+1}^p, Q_{n+1}^p) \\
&\quad + \frac{\aleph h^\aleph}{\mathbb{AB}(\aleph)\Gamma(\aleph + 2)} \sum_{q=0}^n \Xi_{q,n+1} H_3(t_q, S_q, E_q, A_q, D_q, R_q, Q_q), \\
D_{n+1} &= D_0 + \frac{(1 - \aleph) h^\aleph}{\mathbb{AB}(\aleph)\Gamma(\aleph + 2)} H_4(t_{n+1}, S_{n+1}^p, E_{n+1}^p, A_{n+1}^p, D_{n+1}^p, R_{n+1}^p, Q_{n+1}^p) \\
&\quad + \frac{\aleph h^\aleph}{\mathbb{AB}(\aleph)\Gamma(\aleph + 2)} \sum_{q=0}^n \Xi_{q,n+1} H_4(t_q, S_q, E_q, A_q, D_q, R_q, Q_q), \\
R_{n+1} &= R_0 + \frac{(1 - \aleph) h^\aleph}{\mathbb{AB}(\aleph)\Gamma(\aleph + 2)} H_5(t_{n+1}, S_{n+1}^p, E_{n+1}^p, A_{n+1}^p, D_{n+1}^p, R_{n+1}^p, Q_{n+1}^p) \\
&\quad + \frac{\aleph h^\aleph}{\mathbb{AB}(\aleph)\Gamma(\aleph + 2)} \sum_{q=0}^n \Xi_{q,n+1} H_5(t_q, S_q, E_q, A_q, D_q, R_q, Q_q), \\
Q_{n+1} &= S_0 + \frac{(1 - \aleph) h^\aleph}{\mathbb{AB}(\aleph)\Gamma(\aleph + 2)} H_6(t_{n+1}, S_{n+1}^p, E_{n+1}^p, A_{n+1}^p, D_{n+1}^p, R_{n+1}^p, Q_{n+1}^p) \\
&\quad + \frac{\aleph h^\aleph}{\mathbb{AB}(\aleph)\Gamma(\aleph + 2)} \sum_{q=0}^n \Xi_{q,n+1} H_6(t_q, S_q, E_q, A_q, D_q, R_q, Q_q),
\end{aligned}$$

where

$$\Xi_{q,n+1} = \begin{cases} n^{\aleph+1} - (n - \aleph)(n + 1)^\aleph, & \text{if } q = 0, \\ (n - q + 2)^{\aleph+1} + (n - q)^{\aleph+1} - 2(n - q + 1)^{\aleph+1}, & \text{if } 1 \leq q \leq n. \end{cases}$$

Further, the predictor terms are defined as

$$S_{n+1}^p = S_0 + \frac{(1 - \aleph)}{\mathbb{AB}(\aleph)} H_1(t_n, S_n, E_n, A_n, D_n, R_n, Q_n)$$

$$\begin{aligned}
& + \frac{\aleph}{\mathbb{AB}(\aleph)\Gamma^2(\aleph)} \sum_{q=0}^n \omega_{q,n+1} H_1(t_q, S_q, E_q, A_q, D_q, R_q, Q_q), \\
E_{n+1}^p &= E_0 + \frac{(1-\aleph)}{\mathbb{AB}(\aleph)} H_2(t_n, S_n, E_n, A_n, D_n, R_n, Q_n) \\
& + \frac{\aleph}{\mathbb{AB}(\aleph)\Gamma^2(\aleph)} \sum_{q=0}^n \omega_{q,n+1} H_2(t_q, S_q, E_q, A_q, D_q, R_q, Q_q), \\
A_{n+1}^p &= S_0 + \frac{(1-\aleph)}{\mathbb{AB}(\aleph)} H_3(t_n, S_n, E_n, A_n, D_n, R_n, Q_n) \\
& + \frac{\aleph}{\mathbb{AB}(\aleph)\Gamma^2(\aleph)} \sum_{q=0}^n \omega_{q,n+1} H_3(t_q, S_q, E_q, A_q, D_q, R_q, Q_q), \\
D_{n+1}^p &= S_0 + \frac{(1-\aleph)}{\mathbb{AB}(\aleph)} H_4(t_n, S_n, E_n, A_n, D_n, R_n, Q_n) \\
& + \frac{\aleph}{\mathbb{AB}(\aleph)\Gamma^2(\aleph)} \sum_{q=0}^n \omega_{q,n+1} H_4(t_q, S_q, E_q, A_q, D_q, R_q, Q_q), \\
R_{n+1}^p &= S_0 + \frac{(1-\aleph)}{\mathbb{AB}(\aleph)} H_5(t_n, S_n, E_n, A_n, D_n, R_n, Q_n) \\
& + \frac{\aleph}{\mathbb{AB}(\aleph)\Gamma^2(\aleph)} \sum_{q=0}^n \omega_{q,n+1} H_5(t_q, S_q, E_q, A_q, D_q, R_q, Q_q), \\
Q_{n+1}^p &= S_0 + \frac{(1-\aleph)}{\mathbb{AB}(\aleph)} H_6(t_n, S_n, E_n, A_n, D_n, R_n, Q_n) \\
& + \frac{\aleph}{\mathbb{AB}(\aleph)\Gamma^2(\aleph)} \sum_{q=0}^n \omega_{q,n+1} H_6(t_q, S_q, E_q, A_q, D_q, R_q, Q_q),
\end{aligned}$$

where

$$\omega_{q,n+1} = \frac{h^\aleph}{\aleph} \left((n+1-q)^\aleph - (n-q)^\aleph \right), \quad 0 \leq q \leq n.$$

Appendix B

Steady states and polynomial coefficients

Here we present the relevant coefficients of polynomials described in stability theorems and steady states for chapter 5.

Values of l_i in EEP

$$\begin{aligned}
l_1 &= (\theta ((1 - r_1) \phi_1 + \alpha) \lambda_2 + \lambda_1 ((\phi_2 + \mu) \theta + \mu q)) \nu + \lambda_2 q (\phi_1 + \alpha), \\
l_2 &= (((\nu \pi - \pi + 1) \theta + q + \phi_1 + \phi_2 + \mu + \alpha) \gamma + (\phi_1 + \phi_2 + \mu + \alpha) \theta + (q + \phi_1 + \alpha) \\
&\quad + (\phi_1 + \alpha)(q + \phi_2)), \\
l_3 &= (((-\pi(-\mu + (r_1 - 1)\phi_1 - \phi_2 - \alpha)\nu - (\pi - 1)(\phi_1 + \phi_2 + \mu + \alpha))\theta + (q + \phi_1 \\
&\quad + \alpha)\mu + (\phi_1 + \alpha)(q + \phi_2))\gamma + ((\phi_2 + \mu)\theta + q\mu)(\phi_1 + \alpha)), \\
l_4 &= (\lambda_1 \nu \xi^2 + (\lambda_1 \nu \theta + (\lambda_1 \mu - \lambda_2(r_1 - 1)\phi_1 + (\lambda_1 - \lambda_2)q + \phi_2 \lambda_1 + \alpha \lambda_2)\nu + q \lambda_2) \\
&\quad \xi - \nu(-\lambda_1 \mu + \lambda_2(r_1 - 1)\phi_1 - \phi_2 \lambda_1 - \alpha \lambda_2)\theta + q(\lambda_1 \mu \nu + \lambda_2(\phi_1 + \alpha))), \\
l_5 &= ((-q - \theta - \phi_1 - \phi_2 - \mu - \beta - \alpha)\gamma + (-\phi_1 - \phi_2 - \mu - \beta - \alpha)\theta + (-q - \phi_1 \\
&\quad - \beta - \alpha)\mu + (-q - \phi_2 - \beta)\phi_1 + (-\beta - \alpha)q + (-\phi_2 - \beta)\alpha - \phi_2 \beta), \\
l_6 &= (((-\phi_1 - \phi_2 - \mu - \beta - \alpha)\theta + \lambda_1 \nu \Gamma + (-q - \phi_1 - \beta - \alpha)\mu + (-q - \phi_2 - \beta)\phi_1 \\
&\quad + (-\beta - \alpha)q + (-\phi_2 - \beta)\alpha - \phi_2 \beta)\gamma + ((-\phi_1 - \beta - \alpha)\mu + (-\phi_2 - \beta)\phi_1 + \\
&\quad (-\phi_2 - \beta)\alpha - \phi_2 \beta)\theta + ((-q - \beta)\phi_1 + (-\beta - \alpha)q - \beta \alpha)\mu - \beta(\phi_1 + \alpha)(q + \phi_2)), \\
l_7 &= (((\lambda_1 \nu \Gamma + (-\phi_1 - \beta - \alpha)\mu + (-\phi_2 - \beta)\phi_1 + (-\phi_2 - \beta)\alpha - \phi_2 \beta)\theta + (\lambda_1 \mu - \lambda_2(r_1 - \\
&\quad 1)\phi_1 + (\lambda_1 - \lambda_2)q + \phi_2 \lambda_1 + \alpha \lambda_2)\Gamma \nu + ((-q - \beta)\phi_1 + (-\beta - \alpha)q - \beta \alpha)\mu \\
&\quad - \beta(q + \phi_2)\phi_1 + (\Gamma \lambda_2 - \beta \alpha)q - \phi_2 \beta \alpha)\gamma - ((\phi_2 + \mu)\theta + q\mu)(\phi_1 + \alpha)\beta), \\
l_8 &= (((-\lambda_1 \mu + \lambda_2(r_1 - 1)\phi_1 - \phi_2 \lambda_1 - \alpha \lambda_2)\Gamma \nu - \beta(\phi_2 + \mu)(\phi_1 + \alpha))\theta + q(\lambda_1 \mu \Gamma \nu \\
&\quad + (\Gamma \lambda_2 - \beta \mu)(\phi_1 + \alpha))), \\
l_9 &= (q + \theta + \phi_1 + \phi_2 + \mu + \alpha + \gamma), \\
l_{10} &= (\xi^2 + (q + \theta + \phi_2 + \mu)\xi + (\phi_2 + \mu)\theta + q\mu), \\
l_{11} &= (((-(r_1 - 1)\phi_1 - \alpha)\pi \mu \nu - (\pi - 1)(\phi_2 + \mu)(\phi_1 + \alpha))\theta + q\mu(\phi_1 + \alpha)), \\
l_{12} &= (-(\pi - 1)((r_1 - 1)\phi_1 - \xi - \alpha)I_1^1 - I_2^1(\phi_2 \pi - \phi_2) + \beta S^1).
\end{aligned}$$

Coefficients of polynomials (5.6)-(5.7)

The coefficients of (5.6) is given as

$$\begin{aligned}
 a_3 &= \alpha + \gamma + \mu + \phi_1 + \phi_2 - q + 3\tilde{\zeta} \\
 a_2 &= \alpha\gamma + \alpha\mu + \alpha\phi_2 - \alpha q + 2\alpha\tilde{\zeta} + \gamma\mu + \gamma\nu\pi\theta + \gamma\phi_1 + \gamma\phi_2 - \gamma\pi\theta - \gamma q + 2\gamma\tilde{\zeta} + \mu\phi_1 \\
 &\quad - \mu q + 2\mu\tilde{\zeta} + \phi_1\phi_2 - \phi_1 q + 2\phi_1\tilde{\zeta} - \phi_2 q + 2\phi_2\tilde{\zeta} - 3q\tilde{\zeta} + 3\tilde{\zeta}^2 \\
 a_1 &= \alpha\gamma\mu + \alpha\gamma\nu\pi\theta + \alpha\gamma\phi_2 - \alpha\gamma\pi\theta - \alpha\gamma q + \alpha\gamma\tilde{\zeta} - \alpha\mu q + \alpha\mu\tilde{\zeta} - \alpha\phi_2 q + \alpha\phi_2\tilde{\zeta} - 2\alpha q\tilde{\zeta} \\
 &\quad + \alpha\tilde{\zeta}^2 + \gamma^2\nu\phi_1\pi\theta - \gamma\mu\pi\theta - \gamma\mu q + \gamma\mu\tilde{\zeta} + \gamma\nu\phi_1\pi\theta + \gamma\nu\phi_2\pi\theta + 2\gamma\nu\pi\theta\tilde{\zeta} + \gamma\phi_1\phi_2 \\
 &\quad - \gamma\phi_1\pi\theta - \gamma\phi_1 q + \gamma\phi_1\tilde{\zeta} - \gamma\phi_2\pi\theta - \gamma\phi_2 q + \gamma\phi_2\tilde{\zeta} - 2\gamma\pi\theta\tilde{\zeta} - 2\gamma q\tilde{\zeta} + \gamma\tilde{\zeta}^2 - \\
 &\quad \mu\phi_1 q + \mu\phi_1\tilde{\zeta} - 2\mu q\tilde{\zeta} + \mu\tilde{\zeta}^2 - \phi_1\phi_2 q + \phi_1\phi_2\tilde{\zeta} - 2\phi_1 q\tilde{\zeta} + \phi_1\tilde{\zeta}^2 - 2\phi_2 q\tilde{\zeta} + \\
 &\quad \phi_2\tilde{\zeta}^2 - 3q\tilde{\zeta}^2 + \tilde{\zeta}^3 \\
 a_0 &= \alpha\gamma\mu\nu\pi\theta - \alpha\gamma\mu\pi\theta - \alpha\gamma\mu q + 2\alpha\gamma\nu\phi_2\pi\theta + \alpha\gamma\nu\pi\theta\tilde{\zeta} - \alpha\gamma\phi_2\pi\theta - \alpha\gamma\phi_2 q - \alpha\gamma\pi\theta\tilde{\zeta} \\
 &\quad - \alpha\gamma q\tilde{\zeta} - \alpha\mu q\tilde{\zeta} - \alpha\phi_2 q\tilde{\zeta} - \alpha q\tilde{\zeta}^2 + \gamma^2\nu\phi_1\pi\theta + \gamma^2\nu\phi_1\pi\theta\tilde{\zeta} + \gamma\mu\nu\phi_1\pi\theta + \gamma\mu\nu\pi\theta\tilde{\zeta} \\
 &\quad - \gamma\mu\phi_1\pi\theta - \gamma\mu\phi_1 q - \gamma\mu\pi\theta\tilde{\zeta} - \gamma\mu q\tilde{\zeta} + 2\gamma\nu\phi_1\phi_2\pi\theta + \gamma\nu\phi_1\pi\theta\tilde{\zeta} + \gamma\nu\phi_2\pi\theta\tilde{\zeta} + \\
 &\quad \gamma\nu\pi\theta\tilde{\zeta}^2 - \gamma\phi_1\phi_2\pi\theta - \gamma\phi_1\phi_2 q - \gamma\phi_1\pi\theta\tilde{\zeta} - \gamma\phi_1 q\tilde{\zeta} - \gamma\phi_2\pi\theta\tilde{\zeta} - \gamma\phi_2 q\tilde{\zeta} - \\
 &\quad \gamma\pi\theta\tilde{\zeta}^2 - \gamma q\tilde{\zeta}^2 - \mu\phi_1 q\tilde{\zeta} - \mu q\tilde{\zeta}^2 - \phi_1\phi_2 q\tilde{\zeta} - \phi_1 q\tilde{\zeta}^2 - \phi_2 q\tilde{\zeta}^2 - q\tilde{\zeta}^3
 \end{aligned}$$

The coefficients of (5.7) is given as

$$\begin{aligned}
 b_2 &= \lambda_1 I_1^1 + \lambda_2 I_2^1 + \beta + \gamma + \phi + \sigma, \\
 b_1 &= I_1^1 S^1 \lambda_1^2 + I_2^1 S^1 \lambda_1 \lambda_2 + S^1 \gamma \lambda_1 \nu - I_1^1 \gamma \lambda_1 - I_2^1 \gamma \lambda_2 - \beta \gamma - \beta \phi - \beta \sigma - \gamma \phi - \gamma \sigma \\
 &\quad - 2I_1^1 \lambda_1 \tilde{\zeta} - 2I_2^1 \lambda_2 \tilde{\zeta} - 2\beta \tilde{\zeta} - 2\gamma \tilde{\zeta} - 2\phi \tilde{\zeta} - 2\sigma \tilde{\zeta} - 3\tilde{\zeta}^2, \\
 b_0 &= I_1^1 S^1 \lambda_1^3 + I_2^1 S^1 \lambda_1^2 \lambda_2 + I_1^1 S^1 \gamma \lambda_1^2 (1 + \nu) + I_2^1 S^1 \gamma \lambda_1 \lambda_2 (1 + \nu) + I_1^1 S^1 \lambda_1^2 \tilde{\zeta} + \\
 &\quad I_2^1 S^1 \lambda_1 \lambda_2 \tilde{\zeta} - I_1^1 \lambda_1 (\gamma \phi + \gamma \sigma + \tilde{\zeta} \phi + \tilde{\zeta} \sigma + \gamma \tilde{\zeta} + \lambda_1 \tilde{\zeta}^2) - I_2^1 \lambda_2 (\gamma \phi \\
 &\quad + \gamma \sigma + \tilde{\zeta} \phi + \tilde{\zeta} \sigma + \gamma \tilde{\zeta} + \lambda_2 \tilde{\zeta}^2) + S^1 \beta \gamma \lambda_1 \nu - \beta (\gamma \phi + \gamma \sigma + \tilde{\zeta} \phi + \tilde{\zeta} \sigma + \\
 &\quad \gamma \tilde{\zeta} + \tilde{\zeta}^2) - \gamma (\phi \tilde{\zeta} + \sigma \tilde{\zeta} + \tilde{\zeta}^2) - \phi \tilde{\zeta}^2 - \sigma \tilde{\zeta}^2 - \tilde{\zeta}^3.
 \end{aligned}$$

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