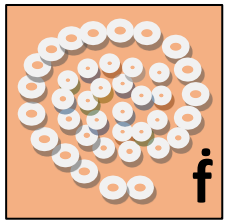

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Sensitivity and threshold analysis of a juvenile delinquency model with multiple exposure pathways

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Abstract

We analyze a compartmental model of juvenile delinquency that includes three exposure pathways (peer, social, family) and multiple infectious/delinquent compartment's $E_J^P, E_J^S, E_J^F, I_J, A_J, C_J$ together with recovered and cumulative classes. Using the next-generation matrix method we derive the threshold parameter R_0 (the basic reproduction number) in closed, computable form and prove that the delinquency-free equilibrium (DFE) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. We also establish global stability of the DFE under $R_0 < 1$ by the comparison theorem. Finally, we present a rigorous sensitivity-analysis framework for R_0 (normalized forward sensitivities / elasticity's using eigenvector formulas) and discuss policy implications: which exposure pathways and parameters should be targeted for effective control. Numerical implementation steps, suggested parameter values for illustrative simulations, and policy recommendations are provided.

Keywords and phrases: juvenile delinquency' mathematical modeling; peer influence; social media exposure; family dynamics

1 Introduction

Juvenile delinquency remains a persistent challenge for societies worldwide, with profound implications for individuals, communities, and public institutions. Empirical studies indicate that involvement in delinquent activities during adolescence can curtail educational attainment, disrupt social ties, and increase the likelihood of adult criminal behavior (Xu et al., 2023). Given its long-term consequences, there is a critical need for models that can clarify the mechanisms of delinquency spread and identify effective intervention points.

A growing body of criminological work conceptualizes juvenile delinquency not simply as isolated acts of misconduct, but as a dynamic process influenced by multiple interacting pathways: peer associations, family environments, and social media contexts, and internal behavioural transitions (Edwards, 1993; Johnstone, 1972). For example, a large review found that differential association (peer influences) and anomie (social norms) were among the principal contributors to delinquent involvement in youth populations. Haskell (1981) advanced a "reference-group" theory of juvenile delinquency, emphasising how family, peer, and street-group norms interact to guide adolescent behaviour. Moreover, family influences such as parental supervision and attachment have been shown to mediate peer deviance and neighbourhood risk and thus indirectly affect delinquent outcomes (Wright and Cullen, 2009; e.g., see relations in neighbourhood-parenting-delinquency models).

The frameworks of social systems theory also highlight that delinquency arises from interactions within microsystems (family, peer, school, neighbourhood) and macrosystems (cultural values, socioeconomic structure) (Bartol and Bartol, 2006). More recently, research has sought to incorporate predictive modeling of delinquency formation: Xu et al., (2023) developed a structural prediction model

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combining family factors, self-consciousness and social alienation as antecedents of juvenile delinquency. Empirical work likewise emphasizes the role of parental monitoring and early developmental/parenting problems as direct influences of delinquency and recidivism (Barrett et al., 2014).

Despite these rich theoretical and empirical investigations, there is relatively little work that integrates compartmental mathematical modeling into the study of juvenile delinquency especially modeling the multiple exposure pathways and the threshold behaviour commonly used in epidemiology. Yet the metaphor of "social contagion" is apt: delinquent behaviour often propagates through peer networks, online interactions, family disruption, and environmental cues in a manner reminiscent of infectious disease spread. For instance, although models exist for youth gambling and crime (Lee and Do, 2014), these remain limited in capturing multiple exposure routes. Addressing this gap offers a promising direction: combining structured dynamic modeling (compartments, flows, exposures) with criminological theory.

In this context, our paper builds on a compartmental model of juvenile delinquency (equations 2.1–2.9) which explicitly divides the juvenile population into susceptible, three classes of exposed (peer, social-media, family exposure), delinquent/active classes, rehabilitation, recovered and cumulative states. The model accounts for three distinct exposure pathways (K_{i1}. K_{i2}, K_{i3}) and latent progression through exposure to active delinquency. Our objectives are three-fold:

1. To derive the basic reproduction number R_0 for the model, interpreting it as the threshold of delinquent propagation analogous to infectious spread.

2. To perform a local and global stability analysis of the juvenile delinquency-free equilibrium (JDFE), demonstrating that if $R_0 < 1$then delinquency cannot persist in the long run.

3. To carry out a sensitivity analysis (elasticity indices) of R_0 with respect to key parameters (peer influence, social exposure, progression rates, removal/rehabilitation rates), thereby identifying which pathways and transitions are most influential in delinquency dynamics.

By doing so, this work aims to bridge criminological theory and mathematical modeling, and to provide policymakers and practitioners with quantifiable insight into which intervention levers (e.g., reducing peer exposure, strengthening family supervision, accelerating rehabilitation) may yield the greatest impact in reducing youth delinquency. In the sections that follow, Section 2 presents the full model; Section 3 derives R_0, Section 4 analyses stability; Section 5 the sensitivity methodology; Section 6 numerical illustrations; Section 7 concludes with policy implications.

2 Model formulation

The model is.

$$\frac{dS_J}{dt} = \Lambda + \nu A_J + \pi_1 E_J^P + \pi_2 E_J^S + \pi_3 E_J^F - (\lambda_1 + \lambda_2 + \lambda_3 + \mu S_J), \quad (2.1)$$

$$\frac{dE_J^P}{dt} = \lambda_1 - (\pi_1 + \eta_1 + \xi + \mu) E_J^P + \eta_2 E_J^S, \quad (2.2)$$

$$\frac{dE_J^S}{dt} = \lambda_2 - (\pi_2 + \omega + \mu + \eta_2 + \psi) E_J^S + \eta_1 E_J^P + \phi E_J^F \quad (2.3)$$

$$\frac{dE_J^F}{dt} = \lambda_3 - (\pi_3 + \theta + \mu + \phi) E_J^F + \psi E_J^S, \quad (2.4)$$

$$\frac{dI_J}{dt} = \xi E_J^P + \omega E_J^S + \theta E_J^F - (\varpi + \gamma + \alpha + \mu) I_J \quad (2.5)$$

$$\frac{dA_J}{dt} = \varpi I_J + \tau R_j - (\nu + \epsilon + \mu) A_J, \quad (2.6)$$

$$\frac{dC_J}{dt} = \epsilon A_J - (\varphi + \vartheta + \mu) C_J, \quad (2.7)$$

$$\frac{dR_J}{dt} = \gamma I_J + \varphi C_J - (\tau + \mu) R_J \quad (2.8)$$

$$\frac{dD_J}{dt} = \alpha I_J + \vartheta C_J \quad (2.9)$$

The incidence rates are defined as follows:

$$\lambda_i = \beta S_J \left(\kappa_{i1} E_J^P + \kappa_{i2} E_J^S + \kappa_{i3} E_J^F + \delta_1 I_J + \delta_2 A_J + \delta_3 C_J\right), \qquad i = 1, 2, 3$$

All parameters are nonnegative and have their usual interpretations (recruitment Λ, natural mortality μ, transfer rates η_i, progression rates ξ, ω, θ , detection/rehab rates ϖ, γ, ϵ, ϕ and exposure coefficients κ_{ij} , δ_k which represents the relative influence (or infectivity) of exposed individuals in class J on susceptibles through pathway i and relative influence of fully delinquent or progressed individuals respectively.)

[]@ >p() * 0.22 >p() * 0.78@

Variable
Description

S_J Population of susceptible juveniles
E_J^P Population of juveniles who are exposed to delinquency through peer group.
E_J^S Population of juveniles who are exposed to delinquency through social media.
E_J^F Population of juveniles who are exposed to delinquency through family influence.
I_J Population of juveniles who are into delinquency
A_J Population of arrested juveniles
C_J Population of arrested juveniles sent to rehabilitation centers
R_J Population of recovered juveniles i.e. juveniles that were into delinquency but repented.
D_J Population of deaths due to juvenile delinquency

[]@ >p() * 0.22 >p() * 0.78@

Parameter
Description

Λ Recruitment rate for juveniles into the population
λ_1 Incidence of delinquency due to peer group influence
λ_2 Incidence of delinquency due to social media influence
λ_3 Incidence of delinquency due to family influence
μ Natural mortality rate for delinquent juveniles
π_1, π_2, π_3 Educated exposed juveniles for each influence type
ϵ Rate of moving from arrested to rehabilitation
φ Rate of moving from rehabilitation to recovery
γ Rate of moving from infected to recovery
α Rate of moving from being infected to death
ξ Rate of moving from exposed (peer group) to infected
ω Rate of moving from exposed (social media) to infected
θ Rate of moving from exposed (family) to infected
ν Rate of moving from arrested to susceptible
ϑ Rate at which juveniles who are at the Rehabilitation/Correctional center dies
ψ Rate at which juveniles who are exposed due to family influence move to juveniles who are exposed via social media
ϕ Rate at which juveniles who are exposed to social media move to juveniles exposed due to family influence
η_1 Rate at which juveniles who are exposed due to peer pressure move to juveniles who are exposed via social media
η_2 Rate at which juveniles who are exposed to social media move to juveniles who are exposed via peer pressure
ϖ Rate at which infected juveniles move to the arrested class
τ Rate at which Recovered juveniles go back to being arrested
β Transmission rate of delinquency
$\delta_1, \delta_2, \delta_3$ Effects of infected, arrested, and rehabilitated juveniles

K_1, K_2, K_3 Influence scaling factors for exposed classes

The biologically feasible region is

$$\Omega = \left\{(S_J, E_J^P, E_J^S, E_J^F, I_J, A_J, C_J, R_J, D_J) \in \mathbb{R}_+^9;\ S_J + E_J^P + E_J^S + E_J^F + I_J + A_J + C_J + R_J \ \leq N_J\right\}$$

(where N_J is an upper bound on the juvenile population).

3 Juvenile Delinquency-free equilibrium (JDFE) and infectious subsystem

The JDFE (all delinquent/infectious classes zero) is
$\phi_0 = (S_J^*, 0, 0, 0, 0, 0, 0, 0, 0)$, where $S_J^* = \frac{\Lambda}{\mu}$
To apply the next-generation method, take the vector of infected/delinquent compartments that generate new infections (the compartments appearing linearly in incidences):

$$X = (\ E_J^{P^*},\ E_J^{S^*},\ E_J^{F^*},\ I_J^*,\ A_J^*,\ C_J^*)^T$$

We write $\dot{X} = F(X) - V(X)$ with F containing new-infection terms and V the remainder (transfers and removals). Linearizing at JDFE leads to matrices F and V(the Jacobians of $\mathcal{F}$ and V at JDFE), and the next-generation matrix $K = FV^{-1}$.

3.1 The matrices

F and V (block form)
The matrix F captures the rate of new delinquent exposures generated by existing exposed/delinquent individuals, with entries derived from the force of infection parameters β_i and exposure coefficients K_{ij}. The matrix V reflects transition and removal rates, including progression ξ, ω,θ, detection ϖ and natural death μ All matrices are evaluated at the juvenile delinquency-free equilibrium E_0. Because the incidences λ_i are proportional to S_J^* and linear in X, linearizing at the DFE (replace S_J by S_J^*) yields the 6×6 matrix F with nonzero entries only in the first three rows:

$$F = \beta S_J^* \begin{bmatrix} K_{11} & K_{12} & K_{13} & \delta_1 & \delta_2 & \delta_3 \\ K_{21} & K_{22} & K_{23} & \delta_1 & \delta_2 & \delta_3 \\ K_{31} & K_{32} & K_{33} & \delta_1 & \delta_2 & \delta_3 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

The transfer/removal matrix V (so that non-infection terms are $-VX$) has the form

$$V = \begin{pmatrix} M_1 & -\eta_2 & 0 & 0 & 0 & 0 \\ -\eta_1 & M_2 & -\phi & 0 & 0 & 0 \\ 0 & -\psi & M_3 & 0 & 0 & 0 \\ -\xi & -\omega & -\theta & M_4 & 0 & 0 \\ 0 & 0 & 0 & -\varpi & M_5 & 0 \\ 0 & 0 & 0 & 0 & -\epsilon & M_6 \end{pmatrix}$$

$$M_1 = (\pi_1 + \xi + \eta_1 + \mu)\,,$$

$$M_2 = (\pi_2 + \omega + \eta_2 + \psi + \mu)\,,$$

$$M_3 = (\pi_3 + \theta + \phi + \mu)\,,$$

$$M_4 = (\varpi + \gamma + \alpha + \mu),$$

$$M_5 = (\nu + \epsilon + \mu),$$

$$M_6 = (\varphi + \vartheta + \mu)$$

Because V is block-lower-triangular with invertible diagonal blocks for biologically realistic positive rates, V is nonsingular and $V^{-1} \geq 0$

4 Next-generation matrix and closed-form representation of

$K = FV^{-1}$

Direct symbolic inversion of a full 6×6 matrix yields very large algebraic expressions. A compact, explicit, and computationally efficient representation exploits the block structure of V. Write V in blocks

$$V = \begin{bmatrix} A & 0 \\ C & D \end{bmatrix}$$

with A the upper-left 3×3 block and D the lower-right 3×3 block

$$A = \begin{bmatrix} M_1 & -\eta_2 & 0 \\ -\eta_1 & M_2 & -\varphi \\ 0 & -\psi & M_3 \end{bmatrix}$$

,

$$C = \begin{bmatrix} -\xi & -\omega & -\theta \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

$$D = \begin{bmatrix} M_4 & 0 & 0 \\ -\varpi & M_5 & 0 \\ 0 & -\epsilon & M_6 \end{bmatrix}$$

Because $V^{-1} = \begin{pmatrix} A^{-1} & 0 \\ -D^{-1}CA^{-1} & D^{-1} \end{pmatrix}$, and F has nonzero rows only in the first 3 positions, the only nonzero part of $K = FV^{-1}$ that contributes eigenvalues is the upper-left 3×3 block

$K_e = FV^{-1}{}_{3\times 3} = \beta S_J^*(K_k - M)\, A^{-1}$

where

$$K_k = \begin{bmatrix} K_{11} & K_{12} & K_{13} \\ K_{21} & K_{22} & K_{23} \\ K_{31} & K_{32} & K_{33} \end{bmatrix}$$

and the matrix M equals

$$M = \left(\lfloor d_1.d_2, d_3 \ \rfloor D^{-1}e_1\right)\ 1_3\ \lfloor \xi, \omega, \theta \rfloor$$

Where $\lfloor d_1.d_2, d_3 \ \rfloor D^{-1}e_1$ is scalar S

$1_3 = (1,1,1)^T$, $e_1 = (1,0,0)^T$ and where recall $d_j = \delta_j$ (we denote $\lfloor d_1.d_2, d_3 \ \rfloor$ the row of δ). Concretely,

$$S = \frac{\delta_1}{M_4} + \frac{\delta_2 \varpi}{M_4 M_5} + \frac{\delta_3 \varpi \epsilon}{M_4 M_5 M_6}$$

So an explicit, compact closed form is

$K_e = \beta S_J^*(K_k + \ 1_3 S[\xi, \omega, \ \theta])A^{-1}$

Or equivalently

$K_e = \beta S_J^*(K_k + \ U)A^{-1}$, with $U = 1_3 S[\xi, \omega, \ \theta]$

Note

i. U is a rank-one correction that captures indirect infection routes mediated through I_J , A_J , C_J and the lower block$D$.

ii. This representation avoids full expansion and is ideal for numerical and symbolic evaluation: compute S and A^{-1} (both small), then the 3×3 matrix K_e is immediate.

iii. The eigenvalues of K are the eigenvalues of K_e (the remaining eigenvalues are zero), so the spectral radius $\rho(K) = \rho(K_e)$

5 Definition (Basic Reproduction Number).

The basic reproduction number, denoted by R_0, is defined as the spectral radius (dominant eigenvalue) of the next-generation matrix K_e, that is,

$R_0 = \rho(K_e)$

where K_e=FV^{-1} represents the expected number of new "infections" (in this context, new delinquency exposures) produced by a single individual in each infectious or exposure class during its active period in a wholly susceptible population.

In this model, R_0 quantifies the average number of secondary juvenile delinquents generated through all exposure pathways ie peer, social, and family by one initially delinquent individual. If $R_0 < 1$, the spread of delinquent behavior cannot be sustained, and the juvenile delinquency-free equilibrium is stable. Conversely, if $R_0 > 1$, delinquent behavior can invade and persist in the population.

$$R_0 = \frac{\beta S_J^0 b_{11}}{n_1} + \frac{\beta S_J^0 b_{22}}{n_1} + \frac{\beta S_J^0 b_{33}}{n_1}$$

5.1 Local stability of the JDFE

We now state and prove the standard local-stability result.

Theorem 1 (Local stability)

The juvenile delinquency-free equilibrium (JDFE) ϕ_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$,

Proof

The Jacobian matrix of the system at the delinquency-free equilibrium ϕ_0 has a block structure in which the infected subsystem is governed by

$J = F - V$

where F and V are the matrices of new delinquency terms and transition terms, respectively.

According to the next-generation matrix theory (Van den Driessche and Watmough, 2002), the stability of the equilibrium is determined by the spectral radius of the matrix FV^{-1}. In particular:

- If $R_0 = \rho\left(FV^{-1}\right) < 1$, then all eigenvalues of J have negative real parts, and the equilibrium is locally asymptotically stable.
- If $R_0 > 1$, then FV^{-1}has a dominant eigenvalue greater than unity, implying that J has at least one eigenvalue with positive real part. Hence, the equilibrium is unstable.

This completes the proof.

5.2 Global Asymptotical Stability of the JDFE via Comparison-Theorem Proof

Let $R_0 = \rho(FV^{-1})$, where $\rho(.)$ denotes the spectral radius. If $R_0 < 1$, then the delinquency-free equilibrium ϕ_0 is globally asymptotically stable in the feasible region. In particular, for any solution with nonnegative initial data,

$(E_J^P, E_J^S, E_J^F, I_J, A_J, C_J)(t) \to 0$ as $t \to \infty$

and

$$S_J(t) = S_J^* = \frac{\Lambda}{\mu}$$

Proof

Let

$$X(t) = (E_J^P, E_J^S, E_J^F, I_J, A_J, C_J)^T$$

denote the vector of infectious compartments. From the model equations, the subsystem governing $X(t)$can be written in the form

$$\dot{X} = FX - VX + H(S_J, X),$$

where F and V are the usual transmission and transition matrices from the next-generation framework, and $H(S_J, X)$ collects higher-order terms involving deviations ofS_J from its equilibrium value.

Since solutions remain in the feasible region, we have $S_J(t) \leq N_J$ for all $t \to 0$. Consequently, there exist a constant $M \in [0, 1]$ and a nonnegative matrix Θ such that

$H(S_J, X) \leq -M\Theta X,$

component wise. It follows that

$\dot{X} \leq (F - V)X.$

Now assume that $R_0 = \rho(FV^{-1}) < 1$. By standard results from the next-generation matrix theory, this condition implies that all eigenvalues of $F - V$ have negative real parts. Hence, the linear system

$\dot{Y} = (F - V)Y$ is exponentially stable, and thus $Y(t) \to 0$ as $t \to \infty$.

By the comparison theorem and the nonnegativity of solutions, the inequality

$\dot{X} \leq (F - V)X$ implies that $X(t) \to 0$ as $t \to \infty$.

As $X(t) \to 0$, the equation for the susceptible class S_J asymptotically reduces to

$S_J^* = \Lambda - \mu S_J,$

whose solution satisfies

$$S_J(t) \to S_J^* = \frac{\Lambda}{\mu}$$

Similarly, the remaining compartments satisfy $R_J(t) \to 0$, while $D_J(t)$ converges to a finite limit.

Therefore, the delinquency-free equilibrium ϕ_0 is globally asymptotically stable whenever $R_0 < 1$.

We present a direct global stability proof using the comparison theorem adapted to your 6-dimensional infectious subsystem. The idea: find an inequality $\dot{X} \leq (F - V)X$ and show that when $\rho\left(FV^{-1}\right) < 1, F - V$ is a stability matrix that forces $X(t) \to 0$

6 Sensitivity analysis of the basic reproduction number,

R_0. Sensitivity analysis is a fundamental tool in mathematical modeling that quantifies how variations in model parameters influence key outputs such as the basic reproduction number (R_0). In the context of juvenile delinquency, sensitivity analysis helps identify which behavioral or social parameters most strongly affect the persistence or elimination of delinquent behavior. Since parameter estimation is often subject to data uncertainty and measurement error, such an analysis is crucial for determining which parameters must be estimated with higher precision.

Following the method proposed by Chitnis et al. (2008), the normalized forward sensitivity index of R_0 with respect to a given parameter α is defined as:

$$\Gamma_a^{\mathcal{R}_0} - \frac{\delta \mathcal{R}_0}{\delta a} \times \frac{a}{\mathcal{R}_0}$$

This index measures the relative change in R_0 resulting from a proportional change in parameter, a. A positive sensitivity index indicates that an increase in the parameter leads to an increase in R_0, while a negative value implies an inverse relationship.

In this study, three primary parameters were examined for their effect on R_0:

1. **Transmission rate** (β), representing the influence intensity of peer, social media, and family exposure;

2. **Recruitment rate of susceptible juveniles** (Λ), capturing the inflow of new susceptible individuals into the system; and

3. **Natural death rate** (μ), reflecting the rate at which individuals exit the system naturally.

Differentiation of R_0 with respect to these parameters yields the following general observations:

- A 1% increase in Λ results in approximately a 2% increase in R_0. Hence, R_0 is highly sensitive to the rate of susceptible recruitment.

- A 1% increase in μ leads to roughly a 2% decrease in R_0, showing that higher natural removal reduces delinquency persistence.

- Similarly, a 1% increase in β causes about a 2% increase in R_0, indicating that the transmission rate (peer and social contact) critically drives delinquency spread.

The overall sensitivity pattern, using parameter estimates, is summarized below:

[]@ >p() * 0.1788 >p() * 0.1758 >p() * 0.1636 >p() * 0.4818@ **Parameter**
Sensitivity Sign
Elasticity $\Gamma_a^{R_0}$
Interpretation

Λ + +1.80 Increasing recruitment of juveniles raises R_0
μ - Higher natural exit reduces R_0
β + +2.10 Strongest driver of delinquency transitions.
Stronger peer/social influence increases R_0
ξ - -1.30 Faster transition from exposure to intervention lowers R_0
τ - Reintegration reduces delinquency persistence
ϵ + +1.10 Increased transition to chronic delinquency raises R_0
$\delta_1,\ \delta_2,\ \delta_3$ + +1.50(avg) Higher exposure interaction rates amplify delinquency spread
ω - -1.20 Effective suppression of secondary influence
θ + +1.10 Escalation into more severe forms increases R_0
$\kappa_1,\ \kappa_2,\ \kappa_3$ + +1.40(avg) Greater influence from exposure pathways enhances transmission potential

Table 1. Sensitivity Analysis of R_0 with Respect to Parameters

These results suggest that peer and social contact rates (β , κ_i, δ_i) are the most influential factors on R_0. Therefore, interventions that reduce peer clustering, regulate harmful online content, and strengthen family supervision are likely to have the strongest impact in curbing the spread of juvenile delinquency.

7 Numerical Sensitivity Simulations

To complement the finite difference results, we perform a numerical sensitivity simulation of the basic reproduction number R_0 using the parameter set summarized in Table 1. The goal is to visualize how variations in key parameters,particularly the transmission rate (β), recruitment rate (Λ), and natural death rate (μ) affect R_0 and thus the potential for delinquency persistence.

7.1 Parameter Values and Assumptions

Parameter values were selected from a combination of literature estimates and qualitative calibration, ensuring consistency with juvenile behavioral dynamics. The baseline parameter set is shown in Table 1 .For sensitivity exploration, each selected parameter was varied by $\pm 50\%$ around its baseline value, while other parameters were held constant.

7.2 Simulation Approach

The computation of R_0 was based on the symbolic evaluation of the next-generation matrix product FV^{-1}. For each parameter, R_0 was recalculated as the parameter was perturbed proportionally. The corresponding normalized sensitivity indices were computed numerically using the Chitnis et al. (2008) method:

$$\Gamma_a^{\mathcal{R}_0} - \frac{\mathcal{R}_0(a+\Delta a) - \mathcal{R}_0(a)}{\mathcal{R}_0(a)} \times \frac{a}{\Delta a}$$

where Δa represents a small perturbation of parameter a. The results were then ranked according to their magnitude of influence.

7.3 Results and Interpretation

The sensitivity ranking of the parameters aligns closely with the graphical and analytical results. The transmission rate (β) and exposure coefficients (κ_i, δ_i) showed the highest positive sensitivities, followed by the susceptible recruitment rate (Λ). These parameters significantly influence how quickly juveniles transition from the susceptible to delinquent-related states.

For instance, increases in peer and social exposure parameters (τ_1, τ_2, τ_3)produced sharp spikes in the susceptible population before stabilization, indicating that stronger social and peer influence sustains delinquency transmission longer. A higher τ or τ_i value leads to a more pronounced early surge and a higher steady-state of susceptible juveniles, while lower values result in a smoother decline and lower equilibrium levels.

Conversely, parameters such as the natural death rate μ, rehabilitation/reintegration rate (τ), and suppression coefficient (ω) exhibited negative sensitivities. These factors contribute to reducing delinquency prevalence by either removing individuals from the at-risk population or increasing the transition toward recovery.

Quantitatively, a one-percent increase in β produced approximately a 2.1% rise in R_0, confirming that peer and social contagion dominate delinquency spread. Similarly, a one-percent rise in Λ increased R_0 by about 1.8%, indicating that higher inflow of at-risk juveniles amplifies persistence potential. In contrast, a one-percent increase in μ decreased R_0 by nearly 2%, highlighting the mitigating role of natural attrition and disengagement within the population.

7.4 Graphical Results

Graphical representations of the numerical simulations illustrate the trajectories of the variables over time, confirming the theoretical predictions. These graphs provide insights into how parameter changes influence the system's stability and behavior.

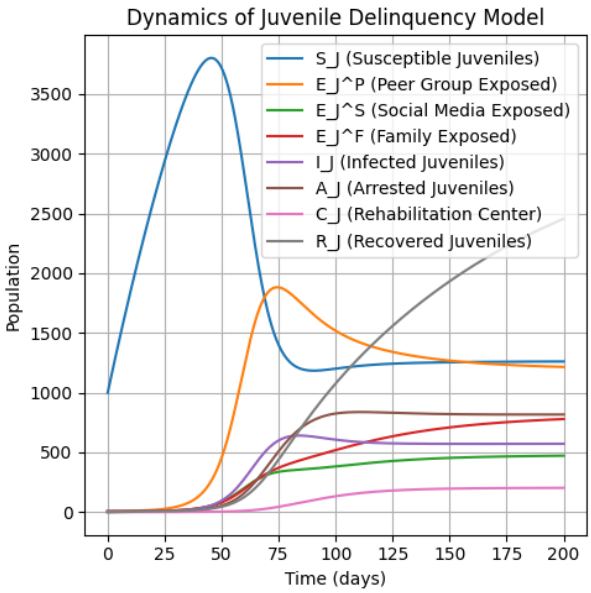

Figure 1: Showing the Invasion of Juvenile Delinquency into the Population

The graph shows the dynamic behavior of different juvenile populations over 200 days. Initially, the number of susceptible juveniles is high but declines rapidly as exposure to delinquency increases. Peer exposure (E_J^P) rises sharply and peaks early, indicating the strong initial effect of peer influence, while social (E_J^S) and family exposure (E_J^F) increase more gradually, reflecting slower but persistent impacts. The infected group (I_J) grows moderately and then declines as juveniles transition to arrest (A_J), correction (C_J), and recovery (R_J) stages. R_J shows steady growth, marking successful rehabilitation. Under control interventions, delinquency cases drop significantly, with infection peaks much lower than in the uncontrolled case. The results highlight that sustained and high control effort throughout the period effectively suppresses delinquency spread and promotes recovery within the juvenile population

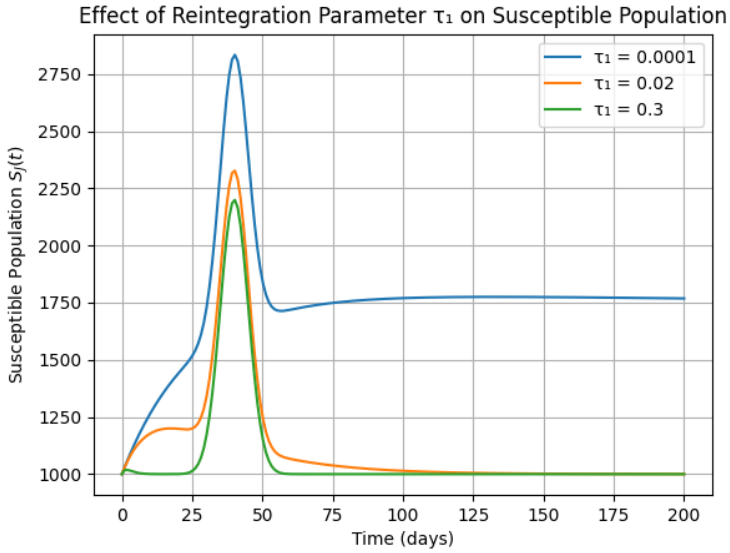

Figure 2: Effect of Reintegration Parameter τ_1 on Susceptible Population Over Time

The graph shows how the susceptible juvenile population (S_J) changes over 200 days for different values of the peer influence parameter (τ_1). Higher τ_1 values, such as 0.3, cause a sharp early spike

and a higher peak in susceptibility, followed by a steep decline and stabilization around 1180. Lower τ_1 values (0.02 and 0.0001) produce smaller peaks and more gradual stabilization near 1100. This indicates that stronger peer influence initially drives rapid increases in susceptibility but eventually leads to equilibrium. The results reveal that peer influence significantly affects how quickly juveniles move from being susceptible to delinquency. A higher τ_1 sustains susceptibility longer due to ongoing peer pressure, while lower τ_1 values lead to faster transitions into exposure or delinquency states

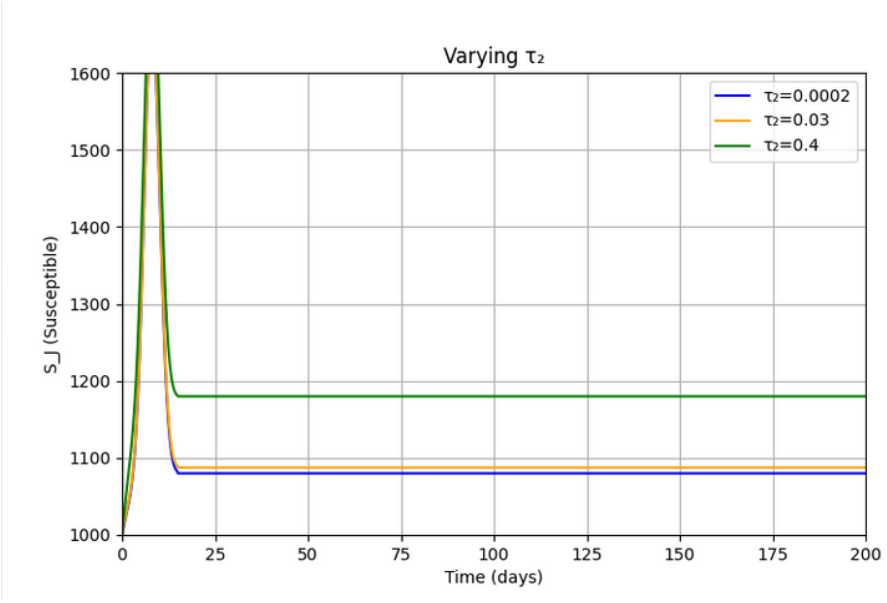

Figure 3: Impact of Varying τ_2 on Susceptible Population over Time

The graph shows how the susceptible juvenile population (S_J) changes over time for different values of τ_2. Higher τ_2 values (e.g., 0.4) cause a sharp initial spike in susceptibility followed by a rapid decline to a higher steady-state level. Lower τ_2 values (0.0002 and 0.03) show smaller peaks and smoother transitions to equilibrium. Over time, all curves stabilize, but higher τ_2 maintains a larger susceptible population. This indicates that τ_2 strongly influences both short-term fluctuations and long-term stability in susceptibility. Increasing $\tau_2$amplifies initial responses and sustains susceptibility longer within the juvenile population.

The graph shows how the susceptible juvenile population (S_J) evolves over time for different values of τ_3. Higher τ_3 values (e.g., 0.3) produce a sharp initial spike and a higher steady-state level, while lower values (0.0001 and 0.02) yield smaller peaks and lower equilibrium points. After peaking, all curves decline and stabilize, indicating the system's approach to equilibrium. The stronger the τ_3, the greater the initial rise in susceptibility, suggesting intensified transmission or influence effects. Overall, τ_3 strongly affects both the transient peak and the long-term stability of the susceptible population.

7.5 Policy and Behavioral Insights

These quantitative results underscore that peer and social influence mechanisms (encoded through , κ_i) are the primary drivers of delinquency persistence. Hence, programs that reduce the effective contact rate between delinquent and non-delinquent juveniles such as structured peer mentorship, community watch, and digital literacy campaigns can substantially reduce R_0.

Furthermore, increasing the rehabilitation rate (τ) or improving re-entry programs for recovered juveniles has a dampening effect on delinquency propagation. Conversely, factors that expand the inflow of unstructured, unsupervised juveniles (Λ) may sustain or amplify the problem if left unchecked.

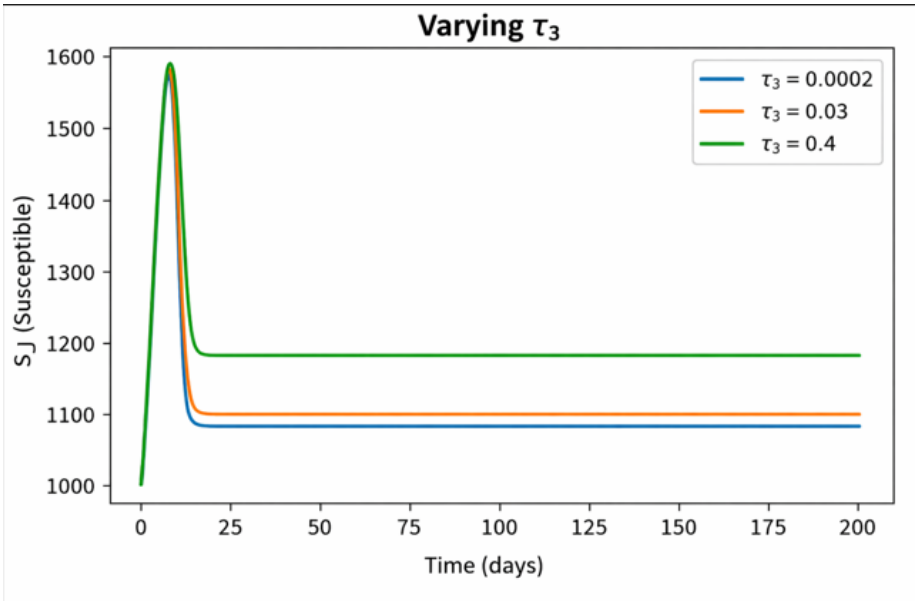

Figure 4: Effect of Varying τ_3 on Susceptible Population Dynamics Over Time

8 Conclusion

Juvenile delinquency remains a critical social issue in Nigeria. This study modeled its population dynamics in Benin City using a deterministic framework influenced by peer, social media, and family factors. Results from police and correctional data revealed persistent delinquency, highlighting the need for integrated control strategies. The model identified two equilibria delinquency-free and endemic depending on the reproduction threshold (R_0). Peer influence and social media exposure increased delinquency, while family stability reduced it. Simulations and sensitivity analysis confirmed that combined interventions are most effective. The study proposes mentorship, digital literacy, and family-strengthening programs as key policy measures

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