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Mathematical Modelling of Family Planning Interventions: A Compartmental Approach to Population Growth Control

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Abstract

This study presents a mathematical modelling approach to analyze the impact of family planning interventions on population growth dynamics. Using a compartmental model, the population is divided into six groups: Susceptible, Informed, Sexually Active Non-Users, Contraceptive Users, Non-Users and General Population. The model incorporates differential equations to describe transitions among these compartments, influenced by factors such as sexual behavior, contraceptive adoption, and public health education. Analytical techniques, including equilibrium analysis and the computation of the basic reproductive number were used to evaluate the model's behavior and stability. Numerical simulations conducted in MATLAB revealed that increased contraceptive usage and awareness significantly reduce the number of high-risk individuals while stabilizing overall population growth. The reproductive number was shown to decrease as contraceptive uptake increased, confirming the effectiveness of intervention strategies. The findings highlight the importance of reproductive health education and contraceptive access in managing population growth, providing valuable insights for policymakers and public health planners. This study demonstrates the potential of mathematical modelling as a predictive and policy-support tool in reproductive health and demographic planning.

Keywords

Family Planning, Population Growth, Mathematical Modelling, Compartmental Model, Reproductive Health, Differential Equations, Basic Reproductive, Numerical Simulation

1. Introduction

Mathematical modelling of family planning intervention through population growth offers a quantitative approach to analyze how reproductive health strategies affect demographic trends [1]. By using differential equations to represent transitions between population groups such as contraceptive users and non-users the model simulates the impact of interventions on fertility rates. This helps policymakers assess the effectiveness of family planning programs and design data-driven strategies to manage population growth.

Population growth poses significant challenges globally, particularly in developing countries where high fertility rates and limited access to reproductive health services exacerbate socio-economic pressures on infrastructure, healthcare, and education [2]. Rapid population expansion in low- and middle-income regions intensifies the demand for resources and highlights the need for urgent and sustainable policy interventions. Family planning has emerged as a crucial strategy for managing population growth and improving public health by enabling individuals and couples to control the number and timing of their children, thereby reducing fertility rates and improving maternal and child health outcomes [3] [4]. However, widespread adoption of family planning services is often hindered by cultural norms, weak healthcare infrastructure, and limited access to education. Nonetheless, research consistently shows that increased access to family planning contributes significantly to lower birth rates and improved health indicators, particularly in developing regions [5] [6].

Family planning plays a critical role in improving public health and socio-economic development by influencing demographic trends and reducing poverty. As population growth continues, especially in developing countries, global efforts have focused on reproductive health education and access to contraception to address related challenges [7]. Effective family planning programs not only support maternal and infant health but also promote gender empowerment and economic advancement [3] [8]. Despite these benefits, access remains uneven due to cultural, infrastructural, and economic barriers [9]. Research highlights the broader impacts of family planning, including improved birth spacing, reduced mortality, and enhanced family welfare, as seen in Pakistan and Indonesia [10] [11]. However, continued resistance and healthcare disparities limit progress. Scholars like [12] emphasize the need for integrated strategies such as digital innovation, policy reform, and community engagement to fully realize family planning's potential. Mathematical models further aid in evaluating intervention effectiveness, offering data-driven insights for shaping sustainable reproductive health policies [1] [13].

[5] utilize the Period Parity Progression Ratio (PPPR) technique to analyze fertility trends in Nepal, revealing that sterilization and injectables are the most effective Family Planning (FP) methods in reducing births. Their model demonstrates a sharp decline in fertility rates from 6.3 children per woman in 1976 to 2.6 in 2011 despite Nepal's limited socio-economic development. By estimating the number of births averted under various contraceptive scenarios, the study pro-

vides valuable insights for designing FP interventions tailored to specific demographic groups, emphasizing the need to address regional and social inequalities in access and usage. Expanding beyond Nepal, global research also affirms the broader benefits of family planning; [2] reports that Indonesia's FP programs have enhanced reproductive autonomy and maternal health, while [14] find that increased post-abortion contraceptive access in Nepal has contributed to reduced maternal mortality.

Rapid population growth presents a major challenge to sustainable development, especially in regions with limited resources and weak health infrastructure. A key contributor to this growth is the high rate of unintended pregnancies resulting from low contraceptive use or method failure. Although family planning interventions aim to reduce fertility and improve reproductive health, many communities still experience rapid population increases, highlighting concerns about the effectiveness and reach of these efforts. A critical gap exists in quantifying the impact of family planning on population dynamics, as traditional demographic methods often lack the precision needed for long-term analysis. To address this, the study employs mathematical modeling to simulate transitions between women who use contraceptives and those who do not, along with broader population changes. This approach provides a detailed understanding of how family planning interventions influence birth rates and supports more informed, data-driven policy development.

2. Methodology

2.1. The Model Formulation

In this section, we will begin by providing a detailed overview of the methodology used in the model, including its key components, underlying assumptions, and computational techniques. The Mathematical modeling and analysis of teenage pregnancies in Kenya, incorporating contraception and education by [1]. Some assumptions were incorporated, and parameters and variables were defined to formulate the model. The model equation was then used in the study.

The model was obtained by incorporating three compartments, such as the Class of women who use contraceptive pills as family planning, Individual women who don't engage in family planning, and Individual women who don't engage in family planning to the model due to [1].

2.2. Assumption of the Model

The following assumptions were made based on the mathematical modelling of family planning intervention through population growth;

1) There is a possibility for the susceptible population to be informed about sex and can be moved to T class;

2) Death rate μ applies to all the compartments;

3) There is a possibility for the susceptible population to be corrupted sexually and hence move to I compartment;

4) A sexually corrupted individual can be informed about sex and moved to T class;

5) Those who were informed about sex can use or not use contraceptive pills;

6) There is a possibility for Sexually Active Non-Users not to engage in family planning and;

7) A class of population can also be susceptible (Table 1 and Table 2).

Table 1. Variables and their meaning in the model.

Variables	Meaning
$S(t)$	Susceptible population at a given time t
$T(t)$	Class of those who were informed about sex at a given time t
$I(t)$	Sexually Active Non-Users class at a given time t
$W_w(t)$	Class of women who use contraceptive pills as family planning at time t
$W_A(t)$	Individual women who don't engage in family planning at time t
$P(t)$	Population at time t

Table 2. Parameters and their meaning in the model.

Parameter	Meaning
Λ	Rate of recruitment into the susceptible population
γ	Rate of movement from the susceptible class to the Informed class T
π	Death due to the use of contraceptive pills
σ	Movement rate from the Sexually Active Non-Users class to the informed class
μ	Natural death rate
β	Contact rate between the Susceptible class and the Corrupted Class
α	Rate of movement from the class of women who do family planning to those who don't
τ	Movement rate from those who don't use contraceptive pills to those who use it
λ	Movement rate from the Sexually informed class to those who use contraceptive pills.
$(1-\lambda)$	Proportion of sexually informed class who are not willing to engage in family planning
ψ	Movement rate from women who do family planning to population class
η	Transfer rate from women who are not using pills to the population class
φ	Movement rate from Population class to Susceptible compartment
ω	Movement rate from Sexually corrupted class to the class that doesn't use pills

2.3. Description of Model

The mathematical modelling of family planning intervention through population growth consists of six compartments. The susceptible compartment denotes $S(t)$ increases by $\Lambda, \varphi P$ and decreases by $\beta SI, \mu S, \gamma S$. Based on the increase and decrease, the differential equation is given by

$$\frac{\mathrm{d}S}{\mathrm{d}t} = \Lambda + \varphi P - \beta SI - \gamma S - \mu S \tag{1}$$

The Sexually Active Non-Users class denoted by $I(t)$ which increase by βSI and decrease by ωI and $(\mu + \pi)I$. The differential equation is

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \beta SI - \omega I - \pi I - \mu I \tag{2}$$

The class $T(t)$ signifies the class of those who were informed about sex. The class is increase by γS and decreases by $\lambda T, (1-\lambda)T, \mu T$ gives the differential equation as

$$\frac{\mathrm{d}T}{\mathrm{d}t} = \gamma S - T - \mu T \tag{3}$$

The class of women who use contraceptive pill as family planning measure, denoted by $W_w(t)$ is increase by $\lambda T, \tau W_A$ and decreases by $\alpha W_A, \psi W_w, (\mu + \pi)W_w$ to give the equation as

$$\frac{\mathrm{d}W_w}{\mathrm{d}t} = \lambda T + \tau W_A - \alpha W_w - \psi W_w - \pi W_w - \mu W_w \tag{4}$$

The class of individual women who don't engage in family planning is denoted by $W_A(t)$, which increases by $(1-\lambda)T, \alpha W_w, \omega I$ and decreases by $\tau W_A, \eta W_A$, μW_A to yield the following differential equation

$$\frac{\mathrm{d}W_A}{\mathrm{d}t} = \alpha W_w + \omega I + (1-\lambda)T - \eta W_A - \tau W_A - \mu W_A \tag{5}$$

The class of population individual signify $P(t)$ is increases by $\eta W_A, \psi W_w$ and decrease by $\mu R, \varphi P$. The equation is given by

$$\frac{\mathrm{d}P}{\mathrm{d}t} = \psi W_w + \eta W_A - \varphi P - \mu P \tag{6}$$

Based on the assumptions and description of the model, the schematic diagram of the family planning intervention model is given as

Based on the model formulation and diagram in **Figure 1**, the model equations were derived as

$$\frac{\mathrm{d}S}{\mathrm{d}t} = \Lambda + \varphi P - \beta SI - (\gamma + \mu)S \tag{7}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \beta SI - (\omega + \mu + \pi) \tag{8}$$

$$\frac{\mathrm{d}T}{\mathrm{d}t} = \gamma S - T - \mu T \tag{9}$$

$$\frac{\mathrm{d}W_w}{\mathrm{d}t} = \lambda T + \tau W_A - (\alpha + \psi + \pi + \mu)W_w \tag{10}$$

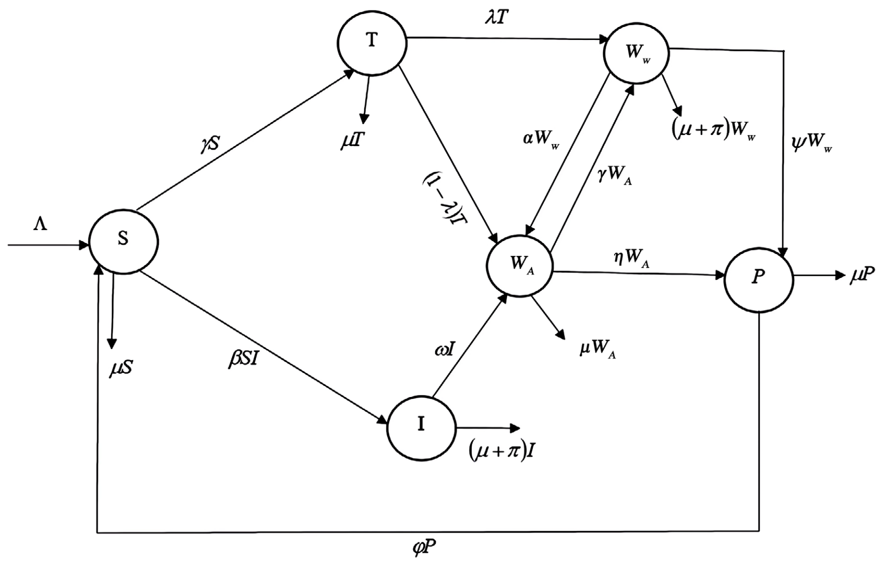

Figure 1. Schematic diagram of the model.

$$\frac{\mathrm{d}W_A}{\mathrm{d}t} = \alpha W_w + \omega I + (1-\lambda)T - (\eta + \tau + \mu)W_A \tag{11}$$

$$\frac{\mathrm{d}P}{\mathrm{d}t} = \psi W_w + \eta W_A - (\varphi + \mu)P \tag{12}$$

3. Model Analysis

The mathematical modelling of family planning intervention through population growth was analyzed in this section. Model analysis involves various techniques and approaches used to examine and interpret the behavior, characteristics, and implications of the mathematical model Equations (7) to (12). This analytical process includes examining the existence and uniqueness of the solution, the positivity and boundedness of the solution, the disease-free and endemic equilibrium points, and the basic reproductive number of the model.

3.1. Positivity of Solution

The positivity solution of the model Equations (7) to (12) involves examining how the variables and parameters interact to maintain positivity, ensuring that the solutions of the model remain physically meaningful.

Theorem 1

Let the initial data be $(S, I, T, W_w, W_A, P)(0) \geq 0 \in Z$. Then, the solution set S, I, T, W_w, W_A, P of Equations (7) to (12), is positive for all $t > 0$.

Proof

To confirm the positivity of the model's solution, it is important to demonstrate that all population compartments remain non-negative over time, as negative values lack physical meaning in this context. This property will be established by proving Theorem 3.1, which states as follows:

Let the initial solution set be

$\{S_0 \ge 0, I_0 \ge 0, T_0 \ge 0, W_{w0} \ge 0, W_{A0} \ge 0, P_0 \ge 0\} \in R_+^6$. Then the solution set $\{S(t), I(t), T(t), W_w(t), W_A(t), P(t)\}$ is positive for all $t > 0$.

$$\frac{dS}{dt} = \Lambda + \varphi P - \beta SI - (\gamma + \mu)S$$

i.e.

$$\frac{dS}{dt} = \Lambda + \varphi P - (\beta I + \gamma + \mu)S \tag{13}$$

Since we are considering only the negative terms susceptible population S, then

$$\frac{dS}{dt} \ge -[\beta I + \gamma + \mu]S \tag{14}$$

This results to

$$\frac{dS}{dt} \ge -(\Upsilon + \mu)S \tag{15}$$

where

$$\Upsilon = \beta I + \gamma \tag{16}$$

Solving for (14) by separating the variables, we have

$$\frac{dS}{S} \ge -(\Upsilon + \mu)dt \tag{17}$$

Integrating (16) we have

$$\ln(S) \ge -\int(\Upsilon + \mu)dt \tag{18}$$

i.e.,

$$\ln(S) \ge -(\Upsilon + \mu)t + C \tag{19}$$

Taking the exponential of (19)

$$S(t) \ge e^{-(\Upsilon+\mu)t+C} \tag{20}$$

i.e.,

$$S(t) \ge e^{-(\Upsilon+\mu)t} + e^{C} \tag{21}$$

$$S(t) \ge Me^{-(\Upsilon+\mu)t} \tag{22}$$

where

i.e.,

$$M = e^{C} \tag{23}$$

Applying the initial conditions at, *i.e.*, $t = 0$, Equation (22) becomes

$$S(t) \ge Me^{-(\Upsilon+\mu)0} \tag{24}$$

$$S(0) \ge K \tag{25}$$

Substituting (25) into (22), we have

$$S(t) \ge S(0)e^{-(\Upsilon+\mu)t} > 0 \tag{26}$$

For all $t \ge 0$.

In a similar way, we can text the positivity of the remaining variables in Equations (7) to (12).

3.2. Equilibrium Point of the Model

The Disease-Free Equilibrium (DFE) represents a condition where the population has no infected individuals, implying that all compartments associated with infection are zero. To determine the DFE, the derivatives of the infected compartments are set to zero, and the resulting system of equations is solved under the assumption that there is no active infection in the population.

Theorem 2

A disease-free equilibrium state of the model (7) to (12) exists where $S = I = T = W_w = W_A = P = 0$ point and is locally asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$. Therefore, at the disease-free equilibrium points, we consider Equation (7).

Proof

To find the equilibrium points of the model Equations (7) to (12), we need to equate the differential Equations (7) to (12) representing the model to zero and solve for the steady-state values of S, I, T, W_w, W_A, P. That is $\frac{\mathrm{d}S}{\mathrm{d}t} = \frac{\mathrm{d}I}{\mathrm{d}t} = \frac{\mathrm{d}T}{\mathrm{d}t} = \frac{\mathrm{d}W_w}{\mathrm{d}t} = \frac{\mathrm{d}W_A}{\mathrm{d}t} = \frac{\mathrm{d}P}{\mathrm{d}t} = 0$.

In particular the disease-free equilibrium point (DFE), E_0, which is obtained by adding the condition $I = T = W_w = W_A = P = 0$. With these conditions, the resolution of the system (7) to (12) gives $E_0 = \left(\frac{\Lambda}{\gamma + \mu}, 0, 0, 0, 0, 0\right)$.

To find the endemic equilibrium, we solve these equations simultaneously to obtain specific values for S, I, T, W_w, W_A, P.

$$\frac{\mathrm{d}S}{\mathrm{d}t} = \Lambda + \varphi P - \beta SI - \gamma S - \mu S$$
$$0 = \Lambda + \varphi P - \beta SI - \gamma S - \mu S$$
$$\Rightarrow \Lambda + \varphi P - \beta SI - \gamma S - \mu S = 0$$
$$\Rightarrow \Lambda - \mu S = 0$$
$$\Rightarrow \Lambda = \mu S$$
$$\Rightarrow S = \frac{\Lambda}{\mu}$$

$$E_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0\right) \tag{27}$$

3.3. Basic Reproduction Number (R_0)

The Basic Reproduction Number, commonly symbolized as R_0, is a fundamental concept in epidemiology that reflects the average number of new infections generated by a single infectious individual in a wholly susceptible population. It is influenced by parameters such as the contact rate, transmission likelihood, and duration of infectiousness. To determine R_0 for the model described by Equations (7) to (12), the Next Generation Matrix (NGM) approach is applied. This method involves linearizing the model around the Disease-Free Equilibrium (DFE), allowing the NGM to represent both the rate at which new infections occur and the progression of infection across compartments.

To find the basic reproduction number R_0, for the model Equations (7) to (12), we use the next-generation matrix approach, which involves linearizing the model around the Disease-Free Equilibrium (DFE). The infected compartments are $E(t), I(t), W_w(t), P(t)$ with the relevant equations

$$\left.\begin{aligned} \frac{\mathrm{d}I}{\mathrm{d}t} &= \beta SI - (\omega+\mu+\pi)I \\ \frac{\mathrm{d}T}{\mathrm{d}t} &= \gamma S - (1+\mu)T \\ \frac{\mathrm{d}W_w}{\mathrm{d}t} &= \lambda T + \tau W_A - (\alpha+\psi+\pi+\mu)W_w \\ \frac{\mathrm{d}P}{\mathrm{d}t} &= \psi W_w + \eta W_A - (\varphi+\mu)P \end{aligned}\right\} \tag{28}$$

The infected Matrix (F)

$$F = \begin{pmatrix} \beta SI \\ \gamma S \\ \lambda T + \tau W_A \\ \psi W_w + \eta W_A \end{pmatrix} = \begin{bmatrix} \beta S & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & \lambda & \tau & 0 \\ 0 & 0 & \eta & 0 \end{bmatrix}$$

and Transition Matrix (V)

$$V = \begin{pmatrix} (\omega+\mu+\pi)I \\ (1+\mu)T \\ (\alpha+\psi+\pi+\mu)W_w \\ (\varphi+\mu)P \end{pmatrix} = \begin{bmatrix} \omega+\mu+\pi & 0 & 0 & 0 \\ 0 & 1+\mu & 0 & 0 \\ 0 & 0 & \alpha+\psi+\pi+\mu & 0 \\ 0 & 0 & 0 & \varphi+\mu \end{bmatrix}$$

The next-generation matrix G of the F (new infection terms) and V (transition terms) is given by:

$$G = F \cdot V^{-1}$$

To calculate G we first need to find the inverse of V as

$$V^{-1} = \begin{bmatrix} \frac{1}{\omega+\mu+\pi} & 0 & 0 & 0 \\ 0 & \frac{1}{1+\mu} & 0 & 0 \\ 0 & 0 & \frac{1}{\alpha+\psi+\pi+\mu} & 0 \\ 0 & 0 & -\frac{\eta}{(\alpha+\psi+\pi+\mu)(\varphi+\mu)} & \frac{1}{\varphi+\mu} \end{bmatrix}$$

The basic reproduction number R_0 is the spectral radius (dominant eigenvalue) of the next-generation matrix G.

$G = F \cdot V^{-1}$ as

$$G = \begin{bmatrix} \beta S & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & \lambda & \tau & 0 \\ 0 & 0 & \eta & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{\omega+\mu+\pi} & 0 & 0 & 0 \\ 0 & \frac{1}{1+\mu} & 0 & 0 \\ 0 & 0 & \frac{1}{\alpha+\psi+\pi+\mu} & 0 \\ 0 & 0 & -\frac{\eta}{(\alpha+\psi+\pi+\mu)(\varphi+\mu)} & \frac{1}{\varphi+\mu} \end{bmatrix}$$

$$= \begin{bmatrix} \frac{\beta S}{\omega+\mu+\pi} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & \frac{\lambda}{1+\mu} & \frac{\tau}{\alpha+\psi+\pi+\mu} & 0 \\ 0 & 0 & \frac{\eta}{\alpha+\psi+\pi+\mu} & 0 \end{bmatrix}$$

$\psi = G - zI = 0$ as

$$\psi = \begin{bmatrix} \frac{\beta S}{\omega+\mu+\pi} - z & 0 & 0 & 0 \\ 0 & -z & 0 & 0 \\ 0 & \frac{\lambda}{1+\mu} & \frac{\tau}{\alpha+\psi+\pi+\mu} - z & 0 \\ 0 & 0 & \frac{\eta}{\alpha+\psi+\pi+\mu} & -z \end{bmatrix}$$

The determinant is given by

$$-\frac{\left(\frac{\beta S}{\omega+\mu+\pi} - z\right) z^2 \left(-\tau + z\alpha + z\psi + z\pi + z\mu\right)}{\alpha+\psi+\pi+\mu}$$

From the structure, the eigenvalues are the entries:

$$\frac{\beta S}{\omega+\mu+\pi}$$

$$z_1 = \frac{\beta S}{\omega+\mu+\pi}, z_2 = 0, z_3 = 0, z_4 = 0$$

Hence, the basic reproductive number is obtained as

$$R_0 = \frac{\beta S}{\omega+\mu+\pi} \tag{29}$$

3.4. Numerical Simulation

This study uses MATLAB software to simulate a compartmental model representing population dynamics influenced by sexual awareness and family planning. By applying assumed parameter values and initial conditions, the system of differential Equations (7) to (12) was solved to observe how interventions such as education and contraceptive use affect population behavior over time. The simulation produced two key outputs: a time-series plot showing the dynamics of each compartment, Susceptible (S), Informed (T), Corrupted (I), Contraceptive users (F), Non-users (N) and General Population (P) and a plot of the basic reproductive number as a function of the contact rate β. (**Table 3**)

Table 3. Variables/Parameters and their assumed values.

Symbol	Values	Reference
$S(t)$	500	[1]
$T(t)$	50	[1]
$I(t)$	20	[1]
$W_w(t)$	30	[5]
$W_A(t)$	40	[5]
$P(t)$	640	[1]
Λ	100	[2]
γ	0.02	[7]
π	0.005	[1]
σ	0.03	[3]
μ	0.01	[1]
β	0.05	[5]
α	0.02	[1]
τ	0.04	[12]
λ	0.06	[5]
$(1-\lambda)$	0.03	[2]
ψ	0.02	[1]
η	0.02	[1]
φ	0.015	[5]
ω	0.025	[1]

Figure 2 below shows how the basic reproductive number (R_0) varies with the contact rate (β), assuming other parameters are held constant.

Figure 3 illustrates the simulation of each compartment (Susceptible, Informed, Corrupted, Family Planning users, Non-users, and Population) over 200 days.

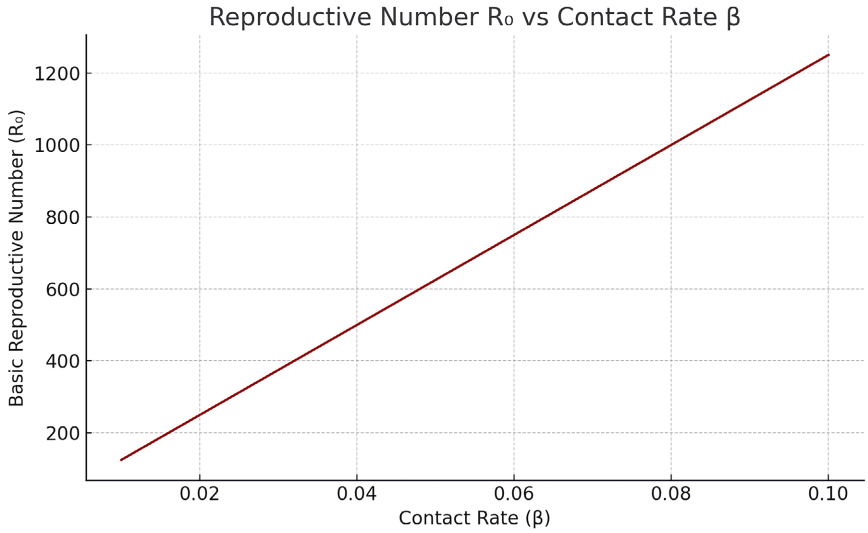

Figure 2. Reproductive number curve.

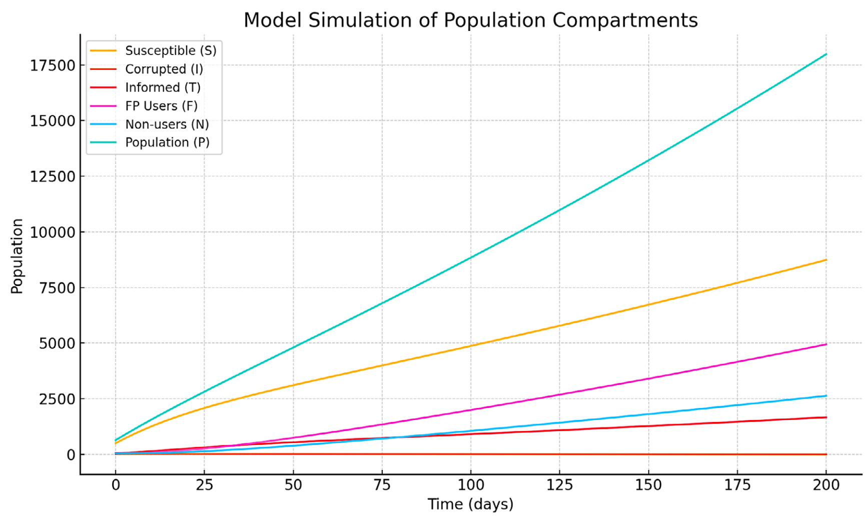

Figure 3. Model compartment simulation.

4. Discussion of Results

The model analysis began with examining key mathematical properties such as the positivity, boundedness and equilibrium states of the system of differential equations representing the population compartments. The analysis confirmed that all solutions remain positive over time, ensuring the model's biological feasibility. The Disease-Free Equilibrium (DFE) was established by setting all infection-related compartments to zero and the stability condition was examined using standard linearization techniques. The basic reproductive number was derived using the Next Generation Matrix (NGM) approach, which quantified the average number of new sexually corrupted individuals generated by a single corrupted individual in a fully susceptible population. A critical result of the model was that the system is locally asymptotically stable at the DFE if $R_0 < 1$, indicating that effective family planning and education interventions can suppress uncontrolled population growth.

The numerical simulation, implemented using MATLAB, utilized assumed parameter values to evaluate the dynamics of each compartment over a 200-day period. The time-series plots revealed that the population of susceptible and sexually corrupted individuals gradually declined, while the number of informed individuals and contraceptive users increased. This shift highlights the impact of sustained awareness and accessibility to contraceptive methods, showing that individuals transition from uninformed or high-risk states to actively managing their reproductive choices. Furthermore, the general population growth rate showed signs of stabilization as a result of the balance achieved between recruitment, education, and contraceptive adoption. These compartmental changes suggest that interventions targeting informed behavior and family planning uptake can reshape the population structure over time.

The reproductive number curve, which was plotted against varying contact rates (β), confirmed that higher rates of sexual contact significantly increase the potential for population expansion if left unregulated. However, as contraceptive use increases and the informed population grows, the effect of contact rate on R_0 is diminished, bringing the value below the critical threshold of one. This affirms the theoretical and empirical understanding that effective family planning reduces fertility and stabilizes population growth. The consistency between these simulation outcomes and existing literature, such as [1] and [5], demonstrates the practical value of mathematical modelling in designing and evaluating reproductive health policies. Ultimately, the results emphasize the need for data-driven, targeted interventions that focus on education, access to contraception, and behavior change to manage population dynamics sustainably.

The parameters of the model can be directly linked to real-world policy interventions, making the results highly relevant for reproductive health planning. For instance, the rate of becoming informed (γ) reflects the effectiveness of public health education campaigns. By expanding sexual education in schools, running community outreach, and increasing media-based awareness, policymakers can raise γ, thereby ensuring that more individuals transition into the informed group. Similarly, the rate of contraceptive adoption (λ) can be improved through policies that guarantee affordable or free contraceptives, strengthen supply chains, and integrate family planning into primary healthcare services. A higher λ means greater uptake of contraceptives, which reduces the pool of high-risk individuals and stabilizes population dynamics.

Other parameters also align with specific policy levers. The rate of transition from informed individuals to contraceptive users (α_1) can be enhanced through counseling, peer education, and support programs that motivate adoption. Improvements in healthcare infrastructure and quality control can reduce risks associated with contraceptive use (μ_1), while broader socio-economic policies that empower women may influence fertility norms and indirectly moderate the recruitment rate (Λ). By linking parameters to policy, the model highlights how targeted interventions in education, contraceptive access, and health systems can reshape demographic outcomes and strengthen family welfare.

5. Discussion of Results

This study developed and analyzed a compartmental mathematical model to examine the impact of family planning interventions on population growth. The model incorporated six compartments: Susceptible, Informed, Sexually Corrupted, Contraceptive Users, Non-Users, and the General Population to simulate transitions influenced by education, sexual activity, and contraceptive behavior. Analytical techniques such as positivity, equilibrium analysis, and the computation of the basic reproductive number ensured the model's biological and mathematical soundness. The simulation, conducted using MATLAB, demonstrated how effective awareness and increased contraceptive uptake could significantly reduce the number of sexually corrupted and uninformed individuals, leading to a gradual shift in population behavior toward more stable reproductive patterns. Additionally, the R_0 curve highlighted the role of contact rate in influencing population dynamics, reinforcing the critical need for controlling risky sexual behavior through education and reproductive health services.

The findings from this model and simulation underscore the pivotal role of family planning in managing population growth, especially in regions with high fertility rates and limited health infrastructure. By providing a data-driven framework, the study offers valuable insights into how targeted interventions such as improving sex education, expanding access to contraceptives, and addressing socio-cultural barriers can reshape population trends and promote long-term socio-economic stability. The model's alignment with empirical studies validates its relevance and provides a tool for policymakers to evaluate the effectiveness of reproductive health programs. In conclusion, mathematical modelling proves to be an essential approach in understanding and guiding public health strategies, particularly in designing sustainable solutions for population control and reproductive well-being.

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Conflicts of Interest

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

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