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STABILITY ANALYSIS AND OPTIMAL CONTROL OF A POPULATION MODEL OF HYPERTENSION

(*Analisis Kestabilan and Kawalan Optimum bagi Model Populasi Hipertensi*)

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ABSTRACT

Hypertension is one of the most common chronic diseases that lead to serious health complications if not treated properly. The purpose of this study is to investigate the population dynamics of hypertension disease with ordinary differential equations and optimal control. A hypertensive population model is developed where the population is categorized into three compartments: non-hypertensive individuals, hypertensive individuals without complications, and hypertensive individuals with complications. The non-negativity and boundedness of the solutions are analyzed. The model has a unique equilibrium point, which is proven to be locally asymptotically stable. After that, an advanced version of the model is proposed where a control variable representing the hypertension intervention strategies is introduced. In the control-based model, we seek to minimize the number of hypertensive individuals and relative costs of implementing the intervention strategies. Numerical simulations are carried out to show the impact of key parameters such as the rate of developing hypertension, recovery rate of complications, and the relative-cost weighting parameter. The results show that implementing intervention strategies can significantly reduce the number of hypertension cases (with and without complications). This study demonstrates how mathematical modeling can help in understanding hypertension progression in the population and planning effective healthcare strategies.

Keywords: hypertension; stability analysis; optimal control

ABSTRAK

Hipertensi merupakan salah satu penyakit kronik yang umum dan boleh membawa kepada komplikasi kesihatan yang serius jika tidak dirawat. Kajian ini bertujuan mengkaji dinamik populasi penyakit hipertensi menggunakan persamaan pembezaan biasa dan kawalan optimum. Satu model populasi pesakit hipertensi dibangunkan di mana sebuah populasi dikelaskan kepada tiga bahagian: individu yang tidak mempunyai hipertensi, individu hipertensi tanpa komplikasi, dan individu hipertensi dengan komplikasi. Sifat tidak negatif dan keterbatasan penyelesaian model dianalisis. Model ini mempunyai satu titik keseimbangan, yang dibuktikan stabil secara asimptotik setempat. Seterusnya, satu versi lanjutan model dicadangkan di mana satu pemboleh ubah kawalan yang mewakili strategi intervensi hipertensi diperkenalkan. Dalam model berasaskan kawalan ini, kita mencari untuk meminimumkan bilangan individu hipertensi dan kos relatif pelaksanaan strategi intervensi. Simulasi berangka dijalankan untuk menunjukkan kesan parameter utama seperti kadar perkembangan hipertensi, kadar pemulihan komplikasi, dan parameter pemberat strategi intervensi dapat mengurangkan dengan ketara bilangan kes hipertensi (dengan dan tanpa komplikasi). Hasil kajian menunjukkan bahawa pelaksanaan strategi intervensi dapat mengurangkan dengan ketara bilangan kes hipertensi (dengan dan tanpa komplikasi). Kajian ini menunjukkan bagaimana pemodelan matematik dapat membantu dalam memahami perkembangan hipertensi dalam populasi dan merancang strategi penjagaan kesihatan dengan lebih berkesan.

Kata kunci: hipertensi; analisis kestabilan; kawalan optimum

1. Introduction

Hypertension, or high blood pressure, is defined as a systolic blood pressure of 130 mmHg or higher and a diastolic blood pressure of 80 mmHg or higher (Essien 2011; Hsu *et al.* 2025; McCarthy *et al.* 2025), frequently written in a fraction form as $\geq 130/80$ mmHg. It represents a chronic condition characterized by persistently elevated blood pressure levels. Hypertension can be classified into two types: primary hypertension and secondary hypertension. More than 95% of hypertensive patients are categorized as having primary hypertension (Essien 2011). The etiology is usually unknown but there are well-known predisposing or risk factors include genetic, prolonged stress, excessive sodium consumption, and obesity (El Meouchy *et al.* 2022). The classification of blood pressure reading can be narrowed into two stages. Stage 1 hypertension is equal to 130-139/80-89 mmHg. Stage 2 is equal to 140/90 mmHg or higher (Essien 2011). These classifications are important for defining treatment targets and appropriateness of intensity of intervention.

Hypertension is somewhat subjectively defined and is often considered the blood pressure level associated with a two-fold increase in long-term health risks. It is a common condition among adults, with a prevalence of around 30% worldwide, and its occurrence rises significantly with age (Burnier & Damianaki 2023). As the country experiences rapid development, many individuals have adopted modern lifestyles and dietary patterns, contributing to the prevalence of hypertension. Table 1 shows the data from Malaysian National Health Morbidity Survey (Institute for Public Health 2024).

Table 1: Malaysian hypertension data

Category/Year	2011	2015	2019	2023
Overall raised blood pressure	32.7%	30.3%	30.0%	29.2%
Known hypertension	12.8%	13.1%	15.9%	17.3%
Not known to have hypertension	19.8%	17.2%	14.1%	11.9%

Many hypertensive individuals remain undiagnosed or fail to receive adequate treatment, resulting in progression to serious complications. Symptoms of hypertension may include persistent headaches, shortness of breath, and chest pain. If ignored, it can cause heart failure, kidney disease, stroke, and even death (Ameer 2022). At an early stage, controlling hypertension is important, as it prevents the patient from the risk of serious complications. Many health authorities, hospitals, and research groups have spent a lot of money and effort to study hypertension, hoping to improve how it is diagnosed and treated to help patients get better results (Javaid *et al.* 2022). Among various approaches, mathematical modeling may provide a framework to study the population dynamics of hypertension and its progression. We hypothesis that using a mathematical model for hypertension, incorporating disease progression and intervention, would provide insights into the underlying dynamics.

Regarding the application of mathematical modeling for population dynamics, many studies have been done related to the non-communicable diseases such as heart failure and heart transplant population dynamics (Tee & Mat Daud 2025), diabetic population dynamics (Yaacob & Nasir 2024), and cardiovascular diseases population dynamics (Nasir 2022). The mathematical modeling of hypertension population dynamics is limited. To the best of our knowledge, one work that is related is done by Mat Daud *et al.* (2019), where the authors constructed a dynamic model using a system of ordinary differential equations to represent the progression and prevalence of hypertensive conditions during pregnancy. The main difference between the present study and Mat Daud *et al.* (2019) is that we consider a more general population, rather than focusing specifically on pregnancy.

This study aims to investigate the population dynamics of hypertension with the implementation of intervention strategies. We first introduce the compartmental modeling in the context

of a hypertension model using ordinary differential equations. The first part involves the analysis of non-negativity and boundedness of the solutions and the stability analysis. The second part involves the development and simulation of a control-based model. Here, we study the effect of varying certain critical parameters on the system, demonstrating some important aspects in managing hypertension. We can investigate how slowing the progression of hypertension, obtained from intervention strategies, can reduce morbidity and mortality through numerical results. The results of this study could provide valuable insights for healthcare providers and policymakers in developing more effective prevention and treatment approaches for hypertension in public health initiatives.

After this introductory section, we discuss the mathematical model of hypertensive population in Section 2. Here, we investigate the non-negativity and boundedness of the solutions and stability of the equilibrium point. In Section 3, we propose a control-based model and solve it numerically using the Forward-Backward Sweep Method. Lastly, the summary of this study is presented in Section 4.

2. The Mathematical Model

Suppose that the total population is assumed divided into three compartments, which are non-hypertensive individuals $M(t)$, hypertensive individuals without complications $H(t)$, and hypertensive individuals with complications $C(t)$. Let $N(t) = M(t)+H(t)+C(t)$ represents the total population. The model parameters to be incorporated are Λ (entry rate of non-hypertensive individuals), α (the rate of developing hypertension), β (the rate of developing complication of hypertension), γ (recovery rate of complications of hypertension), μ_1 (the death rate among non-hypertensive individuals), μ_2 (the death rate among hypertensive individuals without complications), and μ_3 (the death rate among hypertensive individuals with complications).

A compartmental diagram of the model is shown in Figure 1.

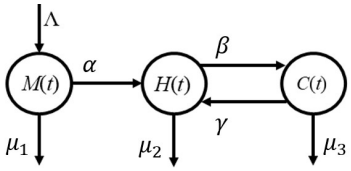

Figure 1: The compartmental diagram for hypertension population dynamics

Figure 1 shows that the individuals enter the system at a constant rate Λ as non-hypertensive individuals $M(t)$. Some of them develop hypertension at a rate of $\alpha M(t)$, transitioning to the compartment of hypertensive individuals without complications $H(t)$. The individual who becomes hypertensive will never return to a non-hypertensive state once they have hypertension. The hypertensive individuals without complications may develop complications at rate $\beta H(t)$ and entering the compartment of hypertensive individuals with complications $C(t)$. Those with complications $(C(t))$ have a chance to recover to hypertensive individuals without complications $(H(t))$ at rate $\gamma C(t)$. Individuals in each compartment experience deaths, where non-hypertensive individuals die at rate $\mu_1 M(t)$, hypertensive individuals without complications die at rate $\mu_2 H(t)$, and hypertensive individuals with complications die at rate $\mu_3 C(t)$.

The rates of change of $M(t)$, $H(t)$, and $C(t)$ form a system of ordinary differential equations as follows

$$\frac{dM(t)}{dt} = \Lambda - (\alpha + \mu_1)M(t), \tag{1a}$$

$$\frac{dH(t)}{dt} = \alpha M(t) - (\beta + \mu_2)H(t) + \gamma C(t), \tag{1b}$$

$$\frac{dC(t)}{dt} = \beta H(t) - (\gamma + \mu_3)C(t), \tag{1c}$$

where $M(t) > 0$, $H(0) > 0$, and $C(0) > 0$.

For simplifying the notations, the functions of time $M(t)$, $H(t)$, $C(t)$, and $N(t)$ are denoted as the variables M, H, C, and N, respectively.

2.1. *Non-negativity and boundedness of the solutions*

Non-negativity refers to the condition in which all variables in the mathematical model have non-negative values (Wu *et al.* 2023). In most studies involving physical or biological systems, such as models of disease spread or population growth, negative values for variables such as the number individuals in a population are illogical and physically unacceptable. Therefore, non-negativity analysis ensures that the solution of the proposed model always remains within a reasonable range. Boundedness refers to the properties of the solutions not increasing infinitely but instead having an acceptable maximum limit. In most situations, resources such as foods, energy, spaces, or chemicals are limited, and therefore, the variables in the mathematical model must have certain maximum values that cannot be exceeded (Cai *et al.* 2022). If a system does not have bounds, it may indicate instability or problems in the model being built.

Theorem 2.1. *The variables M, H, and C of model (1) are non-negative and bounded for $t > 0$.*

Proof. From Eq. (1a), we have

$$\frac{dM}{dt} + (\alpha + \mu_1)M = \Lambda. \tag{2}$$

The integrating factor is $\exp\{(\alpha + \mu_1)t\}$. Multiplying Eq. (2) with the integrating factor yields

$$\frac{d}{dt}\Big[M \exp{(\alpha + \mu_1)t}\Big] = \Lambda \exp\{(\alpha + \mu_1)t\}. \tag{3}$$

Changing t with ξ in Eq. (3) and integrate both sides with respect to ξ from $\xi = 0$ to $\xi = t$ yields

$$M = M(0) \exp\{-(\alpha + \mu_1)t\} + \exp\{-(\alpha + \mu_1)t\} \int_0^t \Lambda \exp\{(\alpha + \mu_1)\xi\} d\xi.$$

Therefore, $M > 0$ for all $t > 0$.

Next, we prove the non-negativity of variables H and C. Variables H and C depend on each other. If we want to prove the non-negativity of H using Eq. (1b), we have a situation where variable C are not yet proven to be always non-negative. We prove by introducing a claim that $H > 0$ for all $t > 0$. If this is not the case, suppose that there exists $\hat{t} > 0$ such that

$$H(t) > 0 \text{ for } t \in [0, \hat{t}),\ H(\hat{t}) = 0, \text{ and } \frac{dH(\hat{t})}{dt} < 0.$$

By rearranging Eq. (1c), we have

$$\frac{dC}{dt} + (\mu_3 + \gamma)C = \beta H. \tag{4}$$

The integrating factor is given as $\exp\{(\mu_3 + \gamma)t\}$. Next, multiplying Eq. (4) with the integrating

factor yields

$$\frac{d}{dt}\Big[C\exp\{(\mu_3+\gamma)t\}\Big] = \beta H\exp\{(\mu_3+\gamma)t\}. \tag{5}$$

Changing t with ξ in Eq. (5) and integrate both sides with respect to ξ from $\xi = 0$ to $\xi = t$ yields

$$C = C(0)\exp\{-(\mu_3+\gamma)t\} + \exp\{-(\mu_3+\gamma)t\}\int_0^t \beta H(\xi)\exp\{(\mu_3+\gamma)\xi\}\,d\xi. \tag{6}$$

This Eq. (6) implies that, for $t \in [0,\hat{t}]$, $C(t) > 0$. Then, from Eq. (1b), we have

$$\frac{dH(\hat{t})}{dt} = \alpha M(\hat{t}) + \gamma C(\hat{t}) > 0,$$

but this lead to a contradiction to the supposition that $\dfrac{dH(\hat{t})}{dt} < 0$. Therefore, $H > 0$ for all $t > 0$. Moreover, from Eq. (6), $C > 0$ for all $t > 0$.

For the boundedness of the solution, adding all Eqs. (1a), (1b), and (1c) gives

$$\frac{dM}{dt} + \frac{dH}{dt} + \frac{dC}{dt} = \Lambda - \mu_1 M - \mu_2 H - \mu_3 C. \tag{7}$$

Let $\mu = \min\{\mu_1,\mu_2,\mu_3\}$ and using $N = M + H + C$, we have

$$\frac{dN}{dt} \le \Lambda - \mu N,$$

which becomes

$$N \le \frac{\Lambda}{\mu} + \Big(N(0) - \frac{\Lambda}{\mu}\Big)\exp\{-\mu t\}.$$

If $N(0) \le \dfrac{\Lambda}{\mu}$, then $N \le \frac{\Lambda}{\mu}$ for all $t > 0$. This implies that the total population is bounded by $\dfrac{\Lambda}{\mu}$. Otherwise, if $N(0) > \dfrac{\Lambda}{\mu}$, then $N(t)$ will decrease to $\dfrac{\Lambda}{\mu}$ because $\lim\limits_{t\to\infty} N(t) = \dfrac{\Lambda}{\mu}$. This completes the proof of Theorem 2.1. □

2.2. *Stability analysis of the equilibrium point*

The equilibrium point (M^*, H^*, C^*) of model (1) is obtained by solving the following equations

$$\begin{aligned} \Lambda - (\alpha+\mu_1)M^* &= 0,\\ \alpha M^* - (\beta+\mu_2)H^* + \gamma C^* &= 0,\\ \beta H^* - (\mu_3+\gamma)C^* &= 0. \end{aligned}$$

Then, we have

$$(M^*, H^*, C^*) = \left(\frac{\Lambda}{\alpha+\mu_1}, \frac{(\mu_3+\gamma)C^*}{\beta}, \frac{\beta\alpha\Lambda}{(\alpha+\mu_1)(\mu_2\gamma+\mu_2\mu_3+\beta\mu_3)}\right).$$

Theorem 2.2. *The equilibrium point (M^*, H^*, C^*) of model (1) is locally asymptotically stable.*

Proof. The Jacobian matrix evaluated at the equilibrium point (M^*, H^*, C^*) is given by

$$\mathbf{J}^* = \begin{bmatrix} -(\alpha+\mu_1) & 0 & 0 \\ \alpha & -(\beta+\mu_2) & \gamma \\ 0 & \beta & -(\mu_3-\gamma) \end{bmatrix}.$$

The characteristic equation, which is obtained by computing $\det(\lambda\mathbf{I} - J^*)$ where λ represents the eigenvalues, is given by

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 = 0, \tag{8}$$

where $a_1 = \alpha+\mu_1+\mu_2+\mu_3+\beta+\gamma$, $a_2 = (\beta+\mu_2)\mu_3 + \mu_2\gamma + (\alpha+\mu_1)(\mu_2+\mu_3+\beta+\gamma)$, and $a_3 = (\alpha+\mu_1)((\beta+\mu_2)\mu_3+\mu_2\gamma)$. According to the Routh-Hurwitz criteria, the real part of all solutions of Eq. (8) is negative if

$$a_1 > 0, \tag{9a}$$
$$a_3 > 0, \tag{9b}$$
$$a_1a_2 - a_3 > 0. \tag{9c}$$

Note that inequalities (9a) and (9b) are satisfied. Inequality (9c) is verified as follows

$$\begin{aligned} a_1a_2 - a_3 &= (\mu_2+\mu_3+\beta+\gamma) \\ &\quad \times \left((\alpha+\mu_1)^2 + (\beta+\mu_2)\mu_3 + \mu_2\gamma + (\alpha+\mu_1)(\mu_2+\mu_3+\beta+\gamma)\right) \\ &\quad > 0. \end{aligned}$$

Therefore, the real part of all roots of Eq. (8) is negative. As a conclusion, the equilibrium point (M^*, H^*, C^*) is locally asymptotically stable. □

2.3. *Numerical simulation of model (1)*

2.3.1. *Stability of the equilibrium point*

The first example is the simulation to validate the stability of the equilibrium point (M^*, H^*, C^*) by examining the time evolution of variables M, H, and C. For this purpose, the following parameter values are hypothetically used:

$$\Lambda = 450,\ \alpha = 0.01,\ \beta = 0.2,\ \gamma = 0.6,\ \mu_1 = 0.01,\ \mu_2 = 0.05,\ \mu_3 = 0.1.$$

The corresponding value of the equilibrium point is $(M^*, H^*, C^*) = (22500, 2863.6, 818.1818)$. Figures 2 and 3 show the simulation results using four different sets of initial conditions. The observed convergence of M, H, and C towards the equilibrium point (M^*, H^*, C^*) suggests that the system exhibits local asymptotic stability. A system is considered locally asymptotically stable if all trajectories that start sufficiently close to the equilibrium point eventually converge to it as time progresses. The graphical results shown in Figures 2 and 3 confirm that the equilibrium point (M^*, H^*, C^*) is locally asymptotically stable.

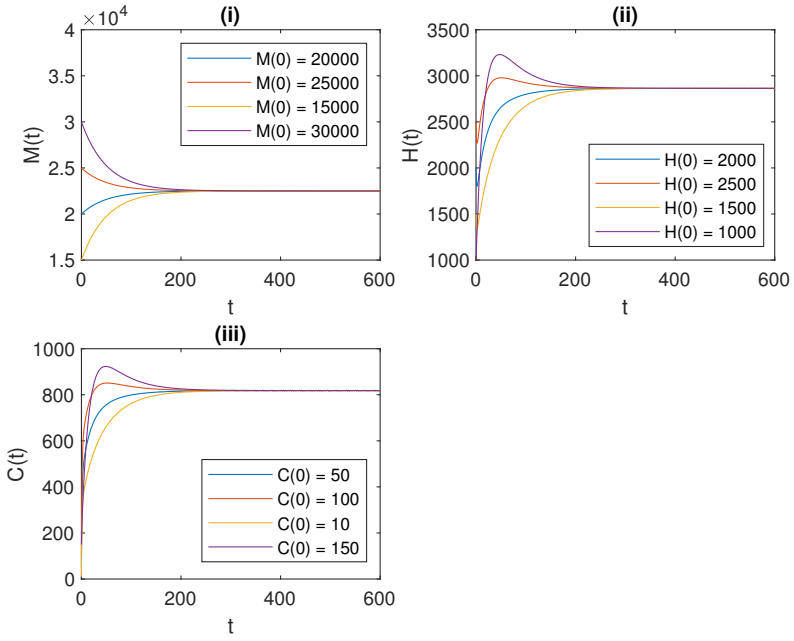

Figure 2: Time series plot simulation for variables M, H, and C with four sets of initial conditions

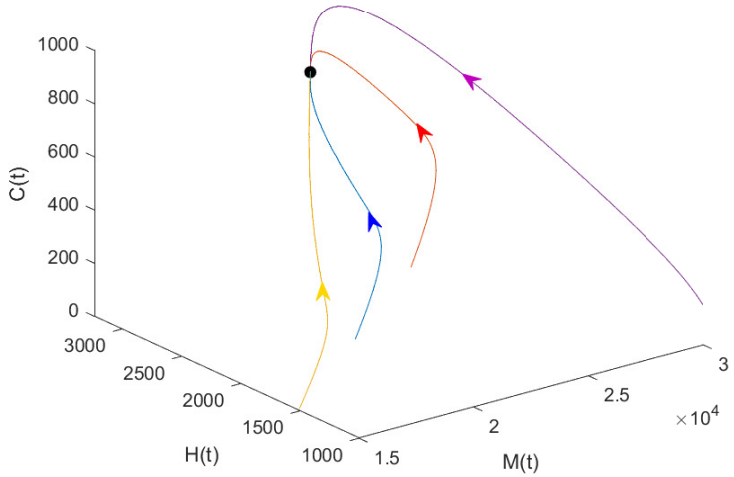

Figure 3: Phase portrait simulation of Figure 2

2.3.2. *Varying the rate of developing hypertension* (α)

The second example is about the simulation of decreasing the rate of developing hypertension (α). The following set of parameter values and initial conditions is hypothetically used:

$$\Lambda = 450,\ \beta = 0.2,\ \gamma = 0.6,\ \mu_1 = 0.01,\ \mu_2 = 0.05,\ \mu_3 = 0.1,$$
$$M(0) = 20000,\ H(0) = 2000,\ C(0) = 50.$$

The simulation presented in Figure 4 examines the effect of varying parameter α. Figure 4(i) illustrates the time evolution of the number of non-hypertensive individuals. As α decreases, the population of M increases. This trend suggests that a lower rate of hypertension development allows more individuals to remain in the non-hypertensive category. Figure 4(ii) displays the

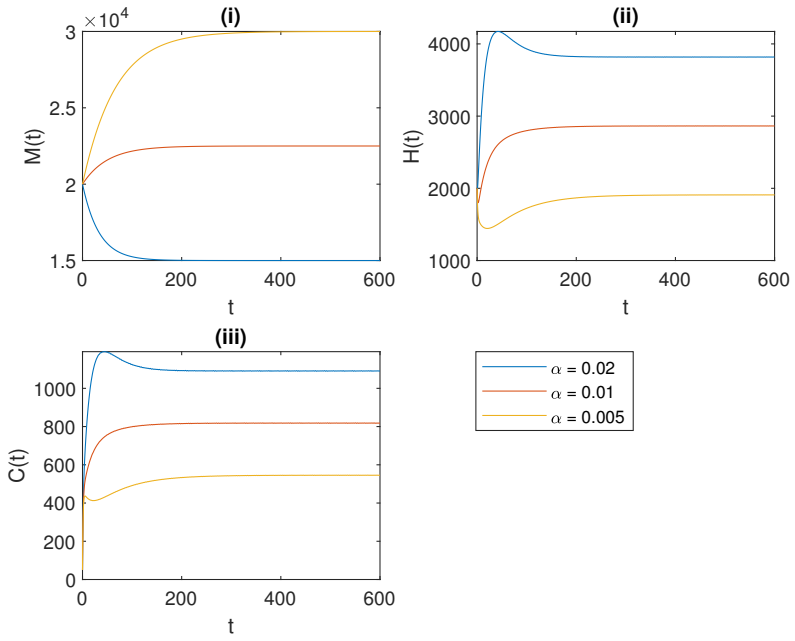

Figure 4: Time series plot simulation with decreasing values of α

population of hypertensive individuals without complications $(H(t))$. A reduction in α leads to a decline in the number of individuals progressing to the hypertensive category. Similarly, the simulation shown in Figure 4(iii) indicates that as α decreases, the population of hypertensive with complications $(C(t))$ also declines. If the parameter α is lower, it results in a reduced of hypertension complications due to the fact that the transition from $H(t)$ to $C(t)$ depends of the number of people with hypertension.

2.3.3. *Varying the recovery rate of complications* (γ)

The third example is about the simulation of increasing the recovery rate of complications (γ). The following set of parameter values and initial conditions is hypothetically used:

$$\Lambda = 450,\ \alpha = 0.02,\ \beta = 0.2,\ \mu_1 = 0.01,\ \mu_2 = 0.05,\ \mu_3 = 0.1,$$
$$M(0) = 20000,\ H(0) = 2000,\ C(0) = 50.$$

The simulation presented in Figure 5 examines the effect of varying parameter γ. Figure 5(i) shows that changes in γ has no impact on the non-hypertensive population $(M(t))$. However, increasing γ from 0.6 to 0.8 reduces the number of individuals with complications $(C(t))$, showing that by increasing the recovery rate of complications, the number of individuals with complications decreases to a much lower level.

These findings reinforce the need to manage the course of hypertension within the public. Understanding the need of high public health awareness through preventive measures like lifestyle modification, early detection and medical interventions is important. A lower rate of development of hypertension results in the overall reduction of the number of hypertensive individuals, and the follow up incidence of hypertension-related complications thus decreases.

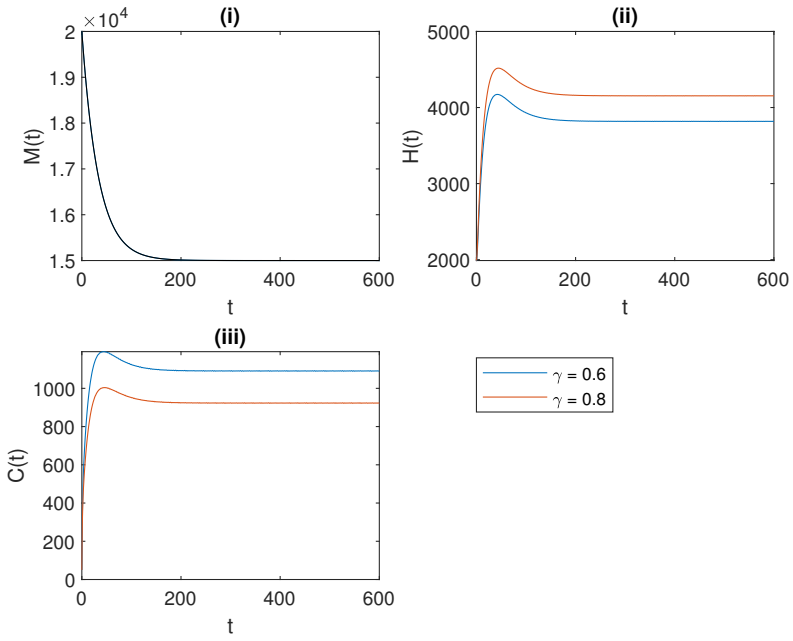

Figure 5: Time series plot simulation with increasing values of parameter γ

3. A Control-Based Mathematical Model

In this section, we formulate a control-based mathematical model for hypertensive population dynamics. Model (1) is extended by including a time-dependent control variable $u(t)$ to reflect the level of intervention efforts at time t. The value of $u(t) = 1$ represents the maximal control efforts and $u(t) = 0$ represents no control efforts. An objective function is defined to evaluate the effectiveness and efforts of the control strategies.

3.1. *Model assumptions*

To formulate the control-based hypertensive population model, several assumptions are introduced as follows.

The control variable $u(t)$ is implemented over a finite time interval $t \in [0, t_f]$ where t_f is the final time. The adherence level of the effectiveness of the intervention efforts is modeled by a parameter $\xi \in [0, 1]$, where $\xi = 0$ implies no adherence while $\xi = 1$ implies that the individuals are adhering the intervention efforts. Hypertension is considered a chronic disease, which it has a long-lasting and persistent effects. Hence, a minimal control level (u_0) is always be implemented in the population even outside of the control period $t \in [0, t_f]$. For example, hypertension awareness through the media.

Thus, the control variable $u(t)$ is bounded as follows:

$$0 \leq u_0 \leq u(t) \leq 1, \text{ for } 0 \leq t \leq t_f.$$

The admissible control set is defined by

$$W = \{u(t) | u_0 \leq u(t) \leq 1\}. \tag{10}$$

Under the influence of $u(t)$, the rate of developing hypertension by non-hypertensive individuals $(M(t))$ is reduced and fewer number of hypertensive individuals without complications $(H(t))$

would develop complications.

Based on model (1) introduced in Section 2, the dynamics of hypertension in the population with a control variable are modeled as follows:

$$\frac{dM(t)}{dt} = \Lambda - \big(1-\xi u(t)\big)\alpha M(t) - \mu_1 M(t), \tag{11a}$$

$$\frac{dH(t)}{dt} = \big(1-\xi u(t)\big)\alpha M(t) - \big(1-\xi u(t)\big)\beta H(t) + \gamma C(t) - \mu_2 H(t), \tag{11b}$$

$$\frac{dC(t)}{dt} = \big(1-\xi u(t)\big)\beta H(t) - (\gamma+\mu_3)C(t), \tag{11c}$$

with the initial conditions $M(0) > 0$, $H(0) > 0$, and $C(0) > 0$. These equations describe the time-dependent evolution of the population across the three compartment under the combined effects of natural progression, recovery and mortality, and intervention efforts modulated the the control variable $u(t)$ and adherence level ξ.

The objective function to be minimized is defined as follows:

$$J\big(u(t)\big) = \int_0^{t_f} H(t) + C(t) + Bu^2(t)\, dt, \tag{12}$$

where $H(t) + C(t)$ is the total burden of hypertension (with and without complications) in the population, $Bu^2(t)$ is a quadratic cost function representing the relative cost of implement control measures (such as human effort, material resources, and infrastructural resources), and B is relative cost-weighting parameter to indicate the relative importance of intervention efforts compared to disease burden. The assumption of this objective functional is the same as in the literature, see Yusuf and Benyah (2012); Olaniyi *et al.* (2020); Jan *et al.* (2020).

The optimal control variable is denoted by $u^*(t)$, where $J(u(t))$ achieves its minimum. The optimal control strategy, therefore, satisfies:

$$J\big(u^*(t)\big) = \min_{u\in W} \int_0^{tf} H(t) + C(t) + Bu^2(t)\, dt.$$

This formulation of control-based hypertension model allows us to find the optimal control $u^*(t)$ such that the accumulated number of total hypertensive individuals during the time period $t \in [0, t_f]$ is minimized, and the relative cost for applying the intervention efforts during the same period is also minimized.

After the formulation of the control-based model, the analysis begins with the derivation of the characterization of the optimal control.

3.2. *Characterization of the optimal control*

The Pontryagin's Maximum Principle (Lenhart & Workman 2007) is applied to obtain the following result.

Theorem 3.1. *If $u^*(t)$ is the optimal control variable to the model proposed in (11) and (12), then there exists adjoint variables $\lambda_1(t)$, $\lambda_2(t)$, and $\lambda_3(t)$ as follows:*

$$\frac{d\lambda_1(t)}{dt} = \alpha\big(1-\xi u(t)\big)(\lambda_1(t) - \lambda_2(t)) + \mu_1\lambda_1(t), \quad \lambda_1(t_f) = 0, \tag{13a}$$

$$\frac{d\lambda_2(t)}{dt} = -1 + \beta\big(1-\xi u(t)\big)(\lambda_2(t) - \lambda_3(t)) + \mu_2\lambda_2(t), \quad \lambda_2(t_f) = 0, \tag{13b}$$

$$\frac{d\lambda_3(t)}{dt} = -1 - \gamma\lambda_2(t) + (\gamma+\mu_3)\lambda_3(t), \quad \lambda_3(t_f) = 0. \tag{13c}$$

Furthermore, the optimal control variable $u^*(t)$ is characterized by

$$u^*(t) = \min\left\{ \max\left\{ u_0, \frac{\beta\xi H(t)(\lambda_3(t) - \lambda_2(t)) + \alpha\xi M(t)(\lambda_2(t) - \lambda_1(t))}{2B} \right\}, 1 \right\}. \tag{14}$$

Proof. The Hamiltonian function $\Psi = \Psi(t)$ is defined as follows:

$$\begin{aligned}\Psi = H + C + Bu^2 &+ \lambda_1\Big(\Lambda - \big(1 - \xi u\big)\alpha M - \mu_1 M\Big) \\ &+ \lambda_2\Big(\big(1 - \xi u\big)\alpha M - \big(1 - \xi u\big)\beta H + \gamma C - \mu_2 H\Big) \\ &+ \lambda_3\Big(\big(1 - \xi u\big)\beta H - (\gamma + \mu_3)C\Big).\end{aligned}$$

Using the Pontryagin's Maximum Principle, Eqs. (13a)-(13c) are derived from

$$\frac{d\lambda_1(t)}{dt} = -\frac{\partial\Psi(t)}{\partial M(t)}, \ \frac{d\lambda_2(t)}{dt} = -\frac{\partial\Psi(t)}{\partial H(t)}, \ \frac{d\lambda_3(t)}{dt} = -\frac{\partial\Psi(t)}{\partial C(t)},$$

with the transversality conditions $\lambda_1(t_f) = 0$, $\lambda_2(t_f) = 0$, and $\lambda_3(t_f) = 0$.

The optimal control $u^*(t)$ is determined from the optimality condition $\dfrac{\partial\Psi(t)}{\partial u(t)} = 0$. Then, solving for $u(t)$ yields

$$u(t) = \frac{\beta\xi H(t)(\lambda_3(t) - \lambda_2(t)) + \alpha\xi M(t)(\lambda_2(t) - \lambda_1(t))}{2B}.$$

By using the bounds on the control variable $u(t)$ (see Eq. (10)), we obtain Eq. (14) as required. □

To numerically apply the optimal control framework, we shall now assemble the complete set of equations to be solved numerically. The system of equations is as follows

$$\frac{dM(t)}{dt} = \Lambda - \big(1 - \xi u(t)\big)\alpha M(t) - \mu_1 M(t), \ M(0) > 0, \tag{15a}$$

$$\frac{dH(t)}{dt} = \big(1 - \xi u(t)\big)\alpha M(t) - \big(1 - \xi u(t)\big)\beta H(t) + \gamma C(t) - \mu_2 H(t), \ H(0) > 0, \tag{15b}$$

$$\frac{dC(t)}{dt} = \big(1 - \xi u(t)\big)\beta H(t) - (\gamma + \mu_3)C(t), \ C(0) > 0, \tag{15c}$$

$$\frac{d\lambda_1(t)}{dt} = \alpha\big(1 - \xi u(t)\big)(\lambda_1(t) - \lambda_2(t)) + \mu_1\lambda_1(t), \ \lambda_1(t_f) = 0, \tag{15d}$$

$$\frac{d\lambda_2(t)}{dt} = -1 + \beta\big(1 - \xi u(t)\big)(\lambda_2(t) - \lambda_3(t)) + \mu_2\lambda_2(t), \ \lambda_2(t_f) = 0, \tag{15e}$$

$$\frac{d\lambda_3(t)}{dt} = -1 - \gamma\lambda_2(t) + (\gamma + \mu_3)\lambda_3(t), \ \lambda_3(t_f) = 0, \tag{15f}$$

$$u^*(t) = \min\left\{ \max\left\{ u_0, \frac{\beta\xi H(t)(\lambda_3(t) - \lambda_2(t)) + \alpha\xi M(t)(\lambda_2(t) - \lambda_1(t))}{2B} \right\}, 1 \right\}. \tag{15g}$$

3.3. *Numerical simulation of system (15)*

In this section, we present the results obtained by solving numerically system (15). The method used is the Forward-Backward Sweep Method with the Runge-Kutta forth order method (Lenhart & Workman 2007). The Forward-Backward Sweep Method is an iterative numerical technique used to solve optimal control problems, particularly those formulated using Pontryagin's Maximum Principle. The method is named for its approach where it solves the state equation forward in time and the adjoint equation backward in time (McAsey *et al.* 2012). This reflects the structure of the resulting system (15), which consists of a forward-in-time initial value problem for the state and a backward-in-time final value problem for the adjoint variables. The Forward-Backward Sweep Method is classified as an indirect method because it solves the boundary value problem resulting from the necessary conditions of optimality.

3.3.1. *Optimal control solution of the model with and without control variable*

Here, the following set of parameter values and initial conditions is used:

$$M(0) = 10000,\ H(0) = 500,\ C(0) = 700,$$
$$\Lambda = 500,\ \alpha = 0.8,\ \beta = 0.1,\ \gamma = 0.7,\ \mu_1 = 0.01,\ \mu_2 = 0.01,\ \mu_3 = 0.02,$$
$$\xi = 0.9,\ B = 8000,\ t_f = 1,\ u_0 = 0.1.$$

These values are assumed hypothetically for illustrative purposes. Figure 6 shows the comparison of the model with and without the control variable. Figure 6 shows that with the inclusion of

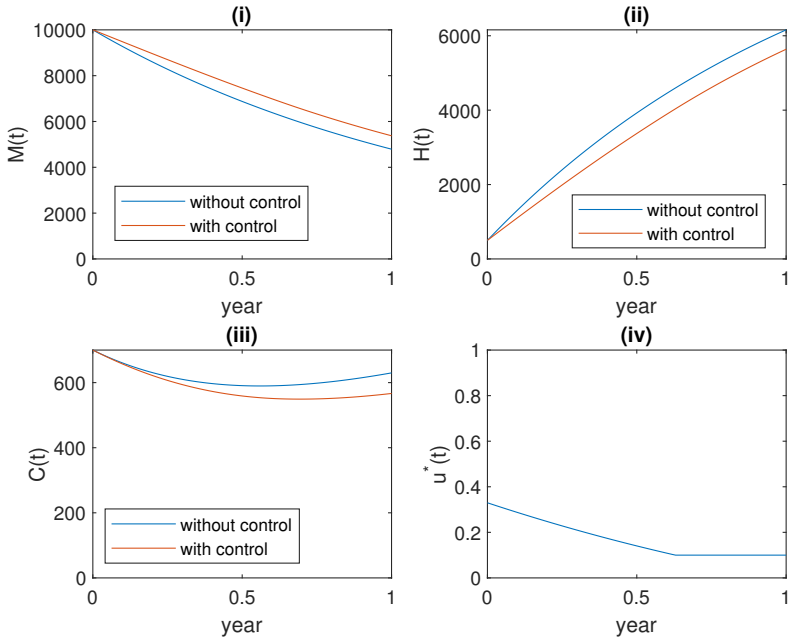

Figure 6: Time series plot simulation of the model with and without control

a control variable, the number of non-hypertensive individuals $(M(t))$ increases, and the number of hypertensive individuals $(H(t)$ and $C(t))$ decreases. Figure 6 shows the optimal control $u^*(t)$ where it starts at the highest value $u^*(0) = 0.3297$ and then declines to the minimum level of control $u_0 = 0.1$.

3.3.2. *Optimal control solution of the model with varying B*

Here, the following set of parameter values and initial conditions is used:

$$M(0) = 10000,\ H(0) = 500,\ C(0) = 700,$$
$$\Lambda = 350,\ \alpha = 0.02,\ \beta = 0.1,\ \gamma = 0.2,\ \mu_1 = 0.01,\ \mu_2 = 0.01,\ \mu_3 = 0.02,$$
$$\xi = 0.9,\ t_f = 1,\ u_0 = 0.1.$$

These values are assumed hypothetically for illustrative purposes. The parameter value for B is varied where B represents the weight on the objective function defined in (12). Figure 7 shows the simulation with the control-based model with decreasing value of B. Decreasing the

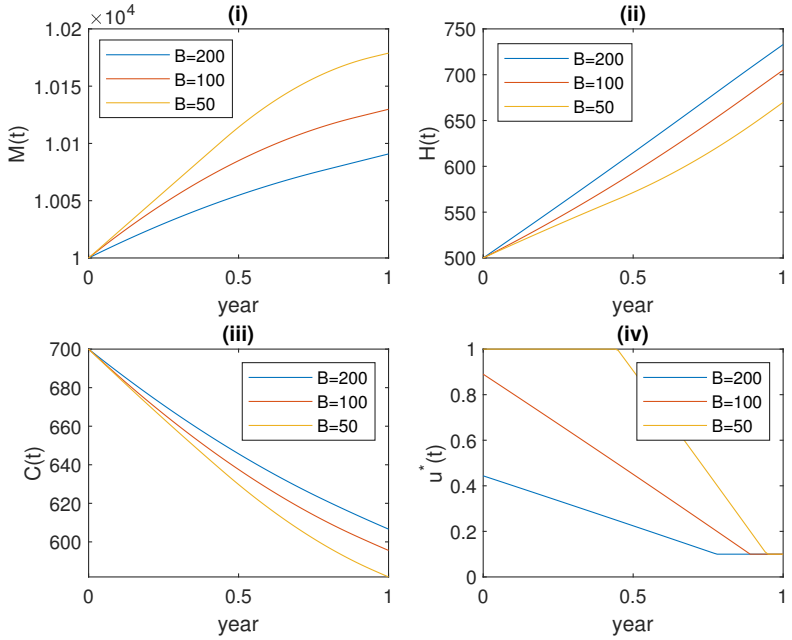

Figure 7: Time series plot simulation of the model with $B = 200$, 100, and 50

weight B indicates that the resources are plenty to implement the intervention efforts. For this reason, when $B = 50$, the control can be implemented at the highest level for a longer duration compared to $B = 100$ and $B = 200$.

In the presence of hypertension intervention, the number of people without hypertension grew over time. The number of hypertensive individuals with complications also decreases over time, indicating that the optimal control has successfully prevented the progression of hypertension to the stage of complications. Consequently, the burden of the healthcare system will be reduced significantly and the efficiency of the treatment rate of hypertension complications will increase.

4. Conclusion

The mathematical model developed in this study effectively portrays the dynamics of hypertension by categorizing the population into non-hypertensive individuals, hypertensive individuals with complications, and hypertensive individuals with complications. Analysis of the model confirms the boundedness and non-negativity of the solutions, ensuring biological feasibility of the results. Stability analysis reveal a unique locally asymptotically stable equilibrium

point, indicating predictable long-term behavior of hypertensive population. Furthermore, a control-based is introduced with the objective function to minimize the number of hypertensive individuals and the relative costs associated with the implementation of the hypertension interventions. The numerical simulations showed that the hypertension intervention efforts prevent people from developing hypertension and lower the number of hypertensive individuals with complications. A 2022 review of non-pharmacological treatments for managing hypertension in primary care confirmed the effectiveness of interventions such as salt reduction, physical activity, and weight loss (Dhungana *et al.* 2022).

The study of hypertension population dynamics with ordinary differential equations and optimal control is relatively new and exciting study area. Future work will concern about the application of data fitting to study the best fit of the proposed model to the real data. This would make the proposed model more useful and reliable. Improving these parts of the model analysis will lead to better tools for healthcare providers and policymakers to plan effective ways to prevent and treat hypertension.

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Disclosure Statement

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