

Journal of Petroleum & Chemical Engineering

https://urfpublishers.com/journal/petrochemical-engineering

Vol: 3 & Iss: 3

Analysis and Control of Pneumonia Transmission Dynamic Models

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Citation: Sridhar LN. Analysis and Control of Pneumonia Transmission Dynamic Models. J Petro Chem Eng 2025;3(3):160-166.

Received: 06 September, 2025; Accepted: 08 September, 2025; Published: 10 September, 2025

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ABSTRACT

Pneumonia is an acute respiratory disease that poses a major threat to human health and causes millions of deaths every year. It is a global health challenge and effective strategies must be implemented to minimize the damage. Therefore, the dynamics of pneumonia transmission must be understood and control methods that are beneficial and cost-effective must be implemented.

In this work, bifurcation analysis and multiobjective nonlinear model predictive control is performed on two dynamic models involving pneumonia transmission. Bifurcation analysis is a powerful mathematical tool used to deal with the nonlinear dynamics of any process. Several factors must be considered and multiple objectives must be met simultaneously. The MATLAB program MATCONT was used to perform the bifurcation analysis. The MNLMPC calculations were performed using the optimization language PYOMO in conjunction with the state-of-the-art global optimization solvers IPOPT and BARON. The bifurcation analysis revealed the existence of branch and limit points in the first model and a branch point in the second model. The MNLMPC converged to the utopia solution in both models. The branch and limit points (which cause multiple steady-state solutions from a singular point) are very beneficial because they enable the Multiobjective nonlinear model predictive control calculations to converge to the Utopia point (the best possible solution) in both models.

Keywords: Bifurcation; Optimization; Control; Pneumonia

Background

Pneumonia is a serious respiratory infection that has been recognized as one of the leading causes of morbidity and mortality worldwide, affecting individuals across all age groups and socioeconomic backgrounds. It is defined as an infection that inflames the air sacs of the lungs, known as alveoli, which may fill with fluid or pus and hinder normal breathing. The causes of pneumonia are diverse, including bacteria, viruses, fungi and other microorganisms and the severity of the disease ranges from mild to life-threatening depending on the patient's age, general health and access to medical care. Despite advances in medicine,

pneumonia remains a major global health challenge, with significant impact in both developed and developing nations.

The clinical presentation of pneumonia can vary greatly depending on the pathogen involved, but common symptoms include cough, fever, chills, difficulty breathing, chest pain and fatigue. In bacterial pneumonia, symptoms often progress quickly with high fever and productive cough, while viral pneumonia may develop more gradually and is frequently accompanied by wheezing and muscle aches. In older adults and very young children, symptoms may be nonspecific, such as confusion, loss of appetite or irritability, making the diagnosis more difficult.

This variability complicates the timely recognition of pneumonia and can delay treatment, particularly in settings with limited diagnostic resources. Severe cases can progress to respiratory failure, sepsis or systemic organ dysfunction, underlining the importance of rapid medical attention.

The causative agents of pneumonia are numerous, but the most common bacterial cause is Streptococcus pneumoniae, also called pneumococcus, which remains a leading cause of community-acquired pneumonia. Other bacteria such as Haemophilus influenzae and atypical organisms like Mycoplasma pneumoniae and Chlamydophila pneumoniae are also significant contributors. Viral causes are increasingly recognized, especially influenza virus, respiratory syncytial virus and coronaviruses, which have been highlighted in recent years due to the COVID-19 pandemic. Fungal pneumonia, although less common, can be severe in immunocompromised patients, particularly those with conditions like HIV/AIDS or those undergoing chemotherapy. Because of the diverse spectrum of pathogens, treatment approaches must be tailored to the suspected cause, the patient's medical history and local epidemiological patterns.

Risk factors for pneumonia are multifaceted and involve both host and environmental components. Age is a major determinant, with infants under two years and adults over sixty-five years particularly vulnerable due to weaker immune defenses. Chronic diseases such as asthma, chronic obstructive pulmonary disease, diabetes and heart failure increase susceptibility by impairing lung function or immune responses. Smoking is a well-established risk factor, as it damages the respiratory tract's natural defenses and increases vulnerability to infection. Malnutrition, poor living conditions, overcrowding and lack of access to healthcare amplify the risk in low-income countries, where pneumonia is a leading cause of death in children under five years. On a global scale, pneumonia reflects the deep inequalities in healthcare access, with developing regions bearing the heaviest burden of mortality.

The diagnosis of pneumonia typically involves a combination of clinical assessment, imaging and laboratory tests. Physicians often begin with a physical examination and patient history, noting symptoms such as fever, cough, chest pain and difficulty breathing. A chest X-ray remains the gold standard for confirming pneumonia, as it can reveal areas of lung consolidation where infection is present. Additional tests may include blood tests to assess inflammatory markers, sputum cultures to identify bacterial pathogens or polymerase chain reaction (PCR) tests for viral identification. In severe cases or in hospitalized patients, more advanced imaging and laboratory diagnostics may be used to tailor treatment. However, in many resource-limited settings, pneumonia is diagnosed and treated based primarily on symptoms and physical examination, which can lead to underdiagnosis or misdiagnosis.

Treatment of pneumonia depends on the underlying cause and the patient's condition. Bacterial pneumonia is generally treated with antibiotics and early administration is crucial to prevent complications. The choice of antibiotic depends on the suspected pathogen, local resistance patterns and patient history, with common options including macrolides, beta-lactams and fluoroquinolones. Viral pneumonia often requires supportive care such as oxygen therapy, hydration and rest, though antiviral medications may be used in cases like influenza or severe COVID-19. Fungal pneumonia requires antifungal therapy, which can be

lengthy and complex. Supportive measures such as supplemental oxygen, intravenous fluids and mechanical ventilation may be necessary in severe cases. Despite the availability of effective therapies, challenges such as antibiotic resistance, lack of access to essential medicines and delayed healthcare-seeking behaviors remain significant barriers to improving outcomes.

Prevention of pneumonia has advanced greatly with the development of vaccines and public health interventions. The pneumococcal conjugate vaccine and pneumococcal polysaccharide vaccine have been widely adopted and are highly effective in reducing infections caused by Streptococcus pneumoniae. Vaccines against influenza and pertussis also indirectly reduce pneumonia incidence, particularly in vulnerable groups such as children, older adults and individuals with chronic illnesses. Childhood immunization programs have significantly lowered pneumonia-related mortality in many countries, although disparities in vaccine coverage persist. Beyond vaccination, preventive strategies include smoking cessation, improved nutrition, access to clean water, reduced air pollution and better management of chronic diseases. In hospitals, infection control measures such as hand hygiene and vaccination of healthcare workers help reduce nosocomial pneumonia.

The global burden of pneumonia remains high and it is considered one of the most important infectious diseases from a public health perspective. According to the World Health Organization, pneumonia is responsible for millions of hospitalizations and hundreds of thousands of deaths annually, especially among children under five and elderly populations. In low- and middle-income countries, the burden is exacerbated by limited access to healthcare, insufficient vaccination programs and high prevalence of risk factors such as malnutrition and indoor air pollution. Pneumonia also places a heavy economic burden on health systems and families, due to medical costs, lost productivity and long-term health complications. The COVID-19 pandemic further emphasized the destructive potential of viral pneumonia, highlighting weaknesses in healthcare preparedness and reinforcing the need for robust global health strategies.

Research into pneumonia continues to evolve, with scientists studying novel diagnostic tools, antimicrobial therapies and vaccine technologies. Point-of-care tests that rapidly identify causative pathogens could revolutionize pneumonia management by enabling more precise treatment and reducing unnecessary antibiotic use. Efforts are also being directed toward addressing antimicrobial resistance, which threatens to undermine the effectiveness of standard therapies. New vaccines and improved formulations are being developed to broaden protection, while public health initiatives aim to strengthen surveillance and reduce inequalities in access to preventive care. Pneumonia is also closely studied in relation to climate change, as environmental factors such as air quality and shifting weather patterns influence respiratory health and disease incidence.

Pneumonia remains a major infectious disease with wideranging clinical, social and economic consequences. It represents a complex interaction between host factors, pathogens and environmental conditions and while effective prevention and treatment strategies exist, they are not equitably accessible across the globe. Vaccination, early diagnosis, appropriate treatment and public health interventions have already saved countless lives, but sustained efforts are needed to reduce the global burden further. Addressing pneumonia is not only a matter of medical importance but also one of social justice, as the disease disproportionately affects the most vulnerable populations. Continued advances in research, healthcare access and preventive measures hold the promise of reducing pneumonia-related suffering and mortality worldwide. Through a combination of scientific progress and equitable health strategies, the global community can make significant strides toward controlling one of the world's most persistent respiratory threats.

Melegaro, et al. provided estimations for the transmission parameters of pneumococcal carriage in households. Van der Poll, et al.² discussed the pathogenesis, treatment and prevention of pneumococcal pneumonia. Ongala, et al.³, developed a control strategy for pneumonia and provided a probabilistic estimation of the basic reproduction number Ngari and co-workers^{4,5} developed and improved dynamic models for childhood pneumonia. Cherazard, et al.6 discussed the prevalence, mechanisms and clinical implications of antimicrobial-resistant Streptococcus pneumoniae. Tilaahun, et al.7, modelled and developed optimal control techniques of pneumonia disease with cost-effective strategies. Kizito, et al.8 developed a mathematical model of treatment and vaccination interventions for pneumococcal pneumonia infection dynamics. Mumbu⁹, modelled the dynamics and performed stability analysis of pneumonia disease infection with parameter uncertainties. Almutairi, et al. 10 discussed optimal control strategies for a mathematical model of pneumonia infection. This work aims to perform bifurcation analysis and multiobjective nonlinear control (MNLMPC) studies in two pneumonia transmission models, which are discussed in Tilaahun, et al.⁷ (model 1) and Almutairi, et al.¹⁰ (model 2). The paper is organized as follows. First, the model equations are presented, followed by a discussion of the numerical techniques involving bifurcation analysis and multiobjective nonlinear model predictive control (MNLMPC). The results and discussion are then presented, followed by the conclusions.

Model Equations

In this section the model equations for two dynamic pneumonia models^{7,10} is presented.

Model 1: Tilaahun, et al.⁷

In this model, (sv, vv, cv, iv, rv) represents the susceptible, vaccinated, carrier, infected and recovered population.

The dynamic model equations are

$$\frac{d(sv)}{dt} = ((1-p)\pi) + (\phi vv) + (\delta rv) - (gvsv) - ((\upsilon + \mu)sv)$$

$$\frac{d(vv)}{dt} = (p\pi) + (\upsilon sv) - (gvvv) - ((\phi + \mu)vv)$$

$$\frac{d(cv)}{dt} = (\rho)gv((\varepsilon vv) + sv) + ((1-q)(1-u2)\eta iv) - ((u3 + \chi + \mu + \beta)cv)$$

$$\frac{d(iv)}{dt} = (1-\rho)gv((\varepsilon vv) + sv) + ((u3 + \chi)cv) - ((u2 + \eta + \mu + \alpha)iv)$$

$$\frac{d(rv)}{dt} = (\beta cv) + ((u2 + (q\eta))iv) - ((\mu + \delta)rv)$$

$$nv = sv + vv + cv + iv + rv;$$

$$gv = \left(\frac{(1-u1)\xi((\gamma cv) + iv)}{nv}\right)$$

The base model parameters are

$$\begin{split} \varepsilon &= 0.002; \ \tau = 0.95; \ \phi = 0.0025; \ \chi = 0.005; \ p = 0.2; \\ \upsilon &= 0.008; \mu = 0.01; \alpha = 0.057; \ \rho = 0.05; \ \beta = 0.0115; \ \eta = 0.2; \ q = 0.75; \\ \gamma &= 1.2; \ \delta = 0.1; \xi = 0.475; \ \pi = 1; \end{split}$$

u1, u2 and u3 are the control parameters.

Model 2: Almutairi, et al.10

In this model, (sv, vv, cv, tv, iv, rv) represent the susceptible, vaccinated, carrier, treated, infected and recovered individuals.

The model equations are

$$\frac{d(sv)}{dt} = \Lambda + (\gamma rv) - av - (\mu + \rho)sv$$

$$\frac{d(vv)}{dt} = \rho sv + \sigma rv - \mu vv$$

$$\frac{d(cv)}{dt} = av - (\mu + \delta + \varepsilon + u^2)cv$$

$$\frac{d(tv)}{dt} = (u^2 + \delta)cv + (\tau iv) - (\mu + \theta)tv$$

$$\frac{d(iv)}{dt} = \varepsilon cv - (\mu + \tau + \alpha + k + u^3)iv$$

$$\frac{d(rv)}{dt} = \theta tv + (k + u^3)iv - (\mu + \sigma + \gamma)rv$$

$$av = \beta \frac{(1 - u^1)sviv}{(1 + miv)}$$

The base parameter values are

$$\begin{split} &\Lambda = 10.09; \varepsilon = 0.01096; \delta = 0.04; \beta = 0.0287; \ m = 0.5; \ k = 0.0115; \theta = 0.02; \alpha = 0.36; \\ &\gamma = 0.00095; \ \mu = 0.0002; \rho = 0.0621; \sigma = 0.36; \tau = 0.07. \end{split}$$

u1, u2 and u3 are control parameters

Bifurcation analysis

The MATLAB software MATCONT is used to perform the bifurcation calculations. Bifurcation analysis deals with multiple steady-states and limit cycles. Multiple steady states occur because of the existence of branch and limit points. Hopf bifurcation points cause limit cycles. A commonly used MATLAB program that locates limit points, branch points and Hopf bifurcation points is MATCONT^{11,12}. This program detects Limit points (LP), branch points (BP) and Hopf bifurcation points(H) for an ODE system

$$\frac{dx}{dt} = f(x, \alpha)$$

 $x \in \mathbb{R}^n$ Let the bifurcation parameter be α . Since the gradient is orthogonal to the tangent vector,

The tangent plane at any point $W = [W_1, W_2, W_3, W_4, ..., W_{n+1}]$ must satisfy

$$Aw = 0$$

Where A is

$$A = [\partial f / \partial x | \partial f / \partial \alpha]$$

where $\partial f / \partial x$ is the Jacobian matrix. For both limit and branch points, the Jacobian matrix $J = [\partial f / \partial x]$ must be singular.

For a limit point, there is only one tangent at the point of singularity. At this singular point, there is a single non-zero vector, y, where Jy=0. This vector is of dimension n. Since there is only one tangent the vector

$$y = (y_1, y_2, y_3, y_4, ... y_n)$$
 must align with
$$\hat{w} = (w_1, w_2, w_3, w_4, ... w_n)$$
. Since

$$J\hat{w} = Aw = 0$$

the n+1 th component of the tangent vector $W_{n+1} = 0$ at a limit point (LP).

For a branch point, there must exist two tangents at the singularity. Let the two tangents be z and w. This implies that

$$Az = 0$$

$$Aw = 0$$

Consider a vector v that is orthogonal to one of the tangents (say w). v can be expressed as a linear combination of z and w ($v = \alpha z + \beta w$). Since Az = Aw = 0; Av = 0 and since w and v are orthogonal,

 $w^T v = 0$. Hence $Bv = \begin{bmatrix} A \\ w^T \end{bmatrix} v = 0$ which implies that B is singular.

Hence, for a branch point (BP) the matrix $B = \begin{bmatrix} A \\ w^T \end{bmatrix}$ must be singular.

At a Hopf bifurcation point,

$$\det(2f_{\alpha}(x,\alpha)@I_{\alpha}) = 0$$

@ indicates the bialternate product while I_n is the n-square identity matrix. Hopf bifurcations cause limit cycles and should be eliminated because limit cycles make optimization and control tasks very difficult. More details can be found in Kuznetsov¹³⁻¹⁵.

Multiobjective Nonlinear Model Predictive Control (MNLMPC)

The rigorous multiobjective nonlinear model predictive control (MNLMPC) method developed by Flores Tlacuahuaz, et al. 16 was used.

Consider a problem where the variables $\sum_{t_{i=0}}^{t_i=t_f} q_j(t_i)$ (j=1,

2..n) have to be optimized simultaneously for a dynamic problem

$$\frac{dx}{dt} = F(x, u)$$

 t_f being the final time value and n the total number of objective variables and u the control parameter. The single objective optimal control problem is solved individually

optimizing each of the variables $\sum_{t_i=0}^{t_i=t_f} q_j(t_i)$ The optimization of $\sum_{t_i=0}^{t_i=t_f} q_j(t_i)$ will lead to the values q_j^* . Then, the

multiobjective optimal control (MOOC) problem that will be solved is

$$\min(\sum_{i=1}^{n}(\sum_{t_{i,n}}^{t_{i}=t_{f}}q_{j}(t_{i})-q_{j}^{*}))^{2}$$

subject to
$$\frac{dx}{dt} = F(x, u)$$
;

This will provide the values of u at various times. The first obtained control value of u is implemented and the rest are discarded. This procedure is repeated until the implemented and the first obtained control values are the same or if the Utopia

point where
$$\left(\sum_{t_{i-1}}^{t_i=t_f} q_j(t_i) = q_j^* \text{ for all j}\right)$$
 is obtained.

Pyomo¹⁷ is used for these calculations. Here, the differential equations are converted to a Nonlinear Program (NLP) using the orthogonal collocation method The NLP is solved using IPOPT¹⁸ and confirmed as a global solution with BARON¹⁹.

The steps of the algorithm are as follows

• Optimize
$$\sum_{t_{i=0}}^{t_i=t_f} q_j(t_i)$$
 and obtain q_j^* .

- Minimize $(\sum_{i=1}^{n} (\sum_{j=1}^{n} q_{j}(t_{i}) q_{j}^{*}))^{2}$ and get the control values at various times.
- Implement the first obtained control values
- Repeat steps 1 to 3 until there is an insignificant difference between the implemented and the first obtained value of the control variables or if the Utopia point is achieved. The

Utopia point is when
$$\sum_{t_{i=0}}^{t_i=t_f} q_j(t_i) = q_j^* \text{ for all j.}$$

Sridhar²⁰ demonstrated that when the bifurcation analysis revealed the presence of limit and branch points the MNLMPC calculations to converge to the Utopia solution. For this, the singularity condition, caused by the presence of the limit or branch points was imposed on the co-state equation²¹. If the minimization of q_1 lead to the value q_1^* and the minimization of q_2 lead to the value q_2^* . The MNLPMC calculations will minimize the function $(q_1-q_1^*)^2+(q_2-q_2^*)^2$. The multiobjective optimal control problem is

min
$$(q_1 - q_1^*)^2 + (q_2 - q_2^*)^2$$
 subject to $\frac{dx}{dt} = F(x, u)$

Differentiating the objective function results in

$$\frac{d}{dx_i}((q_1-q_1^*)^2+(q_2-q_2^*)^2)=2(q_1-q_1^*)\frac{d}{dx_i}(q_1-q_1^*)+2(q_2-q_2^*)\frac{d}{dx_i}(q_2-q_2^*)$$

The Utopia point requires that both $(q_1-q_1^*)$ and $(q_2-q_2^*)$ are zero. Hence

$$\frac{d}{dx_i}((q_1-q_1^*)^2+(q_2-q_2^*)^2)=0$$

The optimal control co-state equation is

$$\frac{d}{dt}(\lambda_i) = -\frac{d}{dx_i}((q_1 - q_1^*)^2 + (q_2 - q_2^*)^2) - f_x \lambda_i; \quad \lambda_i(t_f) = 0$$

 λ_i is the Lagrangian multiplier. t_f is the final time. The first term in this equation is 0 and hence

$$\frac{d}{dt}(\lambda_i) = -f_x \lambda_i; \lambda_i(t_f) = 0$$

At a limit or a branch point, for the set of ODE $\frac{dx}{dt} = f(x, u)$

 f_x is singular. Hence there are two different vectors-values for $\left[\lambda_i\right]$ where $\frac{d}{dt}(\lambda_i) > 0$ and $\frac{d}{dt}(\lambda_i) < 0$. In between there is a vector $\left[\lambda_i\right]$ where $\frac{d}{dt}(\lambda_i) = 0$. This coupled with the boundary condition $\lambda_i(t_f) = 0$ will lead to $\left[\lambda_i\right] = 0$. This makes the problem an unconstrained optimization problem and the optimal solution is the Utopia solution.

Results and Discussion

The bifurcation analysis of model 1(u1 is the bifurcation parameter) revealed a limit and a branch point at (sv, vv, cv, iv, rv, u1) values of (37.890795, 21.122761, 3.827876, 1.599404, 2.581193, 0.742103) and (51.219512, 48.780488, 0.0, 0.0, 0.0, 0.715564). This is shown in **(Figure 1a)**.

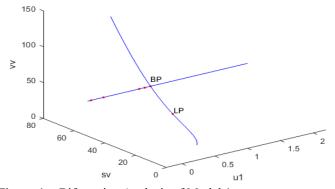


Figure 1a: Bifurcation Analysis of Model 1.

For the MNLMPC calculations in model 1, sv(0) is set to 5000. $\sum_{t_i=t_f}^{t_i=t_f} iv(t_i), \sum_{t_i=0}^{t_i=t_f} cv(t_i) \text{ were minimized individually and each of them led to a value 0. The overall optimal control problem will involve the minimization of <math>(\sum_{t_i=0}^{t_i=t_f} iv(t_i))^2 + (\sum_{t_i=0}^{t_i=t_f} cv(t_i))^2$

was minimized subject to the equations governing the model. This led to a value of zero (the Utopia

The MNLMPC values of the control variables, u1, u2 u3 were 0.2901, 0.2218. 0.1039. The various MNMPC Figures are shown in (Figures 1b-1e). The control profiles u1, u2, u3 (Figure 1d) exhibited noise and this was remedied using the Savitzky-Golay filter (Figure 1e). It is seen that the presence

of the limit and branch points is beneficial because it allows the MNLMPC calculations to attain the Utopia solution, validating the analysis of Sridhar²⁰.

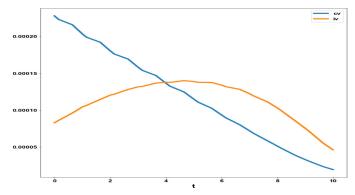


Figure 1b: MNLMPC of Model 1 cv, iv profiles.

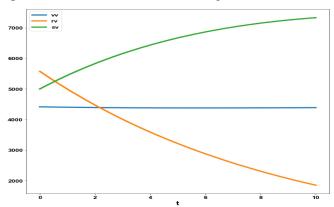


Figure 1c: MNLMPC of Model 1 vv, rv, sv profiles.

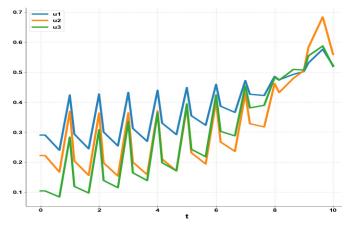


Figure 1d: MNLMPC of Model 1 control profiles (noise exhibited).

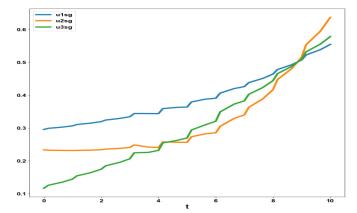


Figure 1e: MNLMPC of Model 1 control profiles (noise eliminated).

The bifurcation analysis of model 2(u2 is the bifurcation parameter) revealed a limit and a branch point at (sv, vv, cv, tv, iv, rv, u2) values of (161.958266, 50288.041734, 0, 0, 0, 0, 0.064177) (**Figure 2a**).

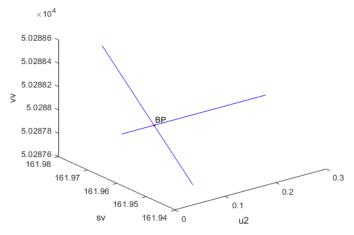


Figure 2a: Bifurcation Analysis of Model 2.

For the MNLMPC calculations in model 2, sv(0) is set to 5000.

$$\sum_{t_{i=0}}^{t_i=t_f} iv(t_i), \sum_{t_{i=0}}^{t_i=t_f} cv(t_i) \text{ were minimized individually and each}$$
 of them led to a values 0. The overall optimal control problem will involve the minimization of
$$(\sum_{t_i=t_f}^{t_i=t_f} iv(t_i))^2 + (\sum_{t_i=t_f}^{t_i=t_f} cv(t_i))^2$$

was minimized subject to the equations governing the model. This led to a value of zero the Utopia.

The MNLMPC values of the control variables, u1, u2 u3 were 0.08173, 0.00968. 0.18883. The various MNMPC figures are shown in (**Figures 2b-2f**). The control profiles u1, u2, u3 (**Figure 2e**) exhibited noise and this was remedied using the Savitzky-Golay filter (**Figure 2f**). It is seen that the presence of the limit and branch points is beneficial because it allows the MNLMPC calculations to attain the Utopia solution, validating the analysis of Sridhar²⁰.

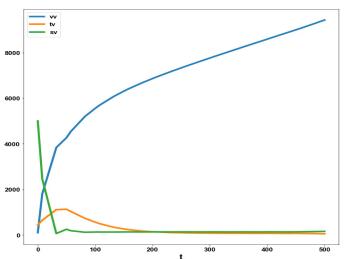


Figure 2b: MNLMPC of Model 2 vv, tv, sv profiles.

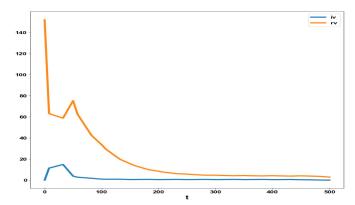


Figure 2c: MNLMPC of Model 2 iv, rv profiles.

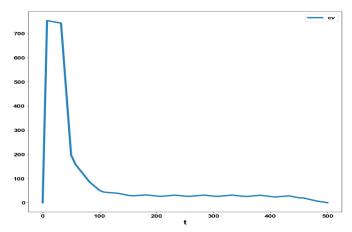


Figure 2d: MNLMPC of Model 2 CV profile.

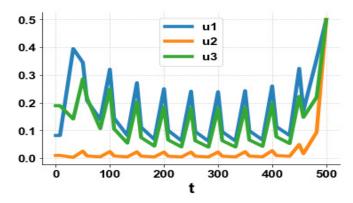


Figure 2e: MNLMPC of Model 2 control profiles (noise exhibited).

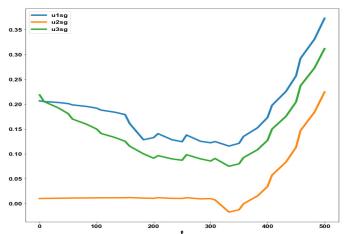


Figure 2f: MNLMPC of Model 2 control profiles (noise eliminated).

Conclusions

Bifurcation analysis and multiobjective nonlinear control (MNLMPC) studies in two dynamic pneumonia transmission models. The bifurcation analysis revealed the existence of a branch and limit point in the birst model and a branch point in the second model. The branch and limit points (which cause multiple steady-state solutions from a singular point) are very beneficial because they enable the Multiobjective nonlinear model predictive control calculations to converge to the Utopia point (the best possible solution) in the models. A combination of bifurcation analysis and Multiobjective Nonlinear Model Predictive Control (MNLMPC) for dynamic pneumonia transmission models is the main contribution of this paper.

Data availability statement

All data used is presented in the paper.

Conflict of interest

The author, Dr. Lakshmi N Sridhar, has no conflict of interest.

Acknowledgement

Dr. Sridhar thanks Dr. Carlos Ramirez and Dr. Suleiman for encouraging him to write single-author papers

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