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Fractional-Order Mathematical Modeling of Methicillin-Resistant *Staphylococcus aureus* Transmission in Hospitals

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Abstract: This article investigates the environmental contamination and antibiotic exposure effect on the transmission dynamics of the Methicillin-Resistant *Staphylococcus aureus* (MRSA) model in hospitals using the fractional Adams–Bashforth–Moulton Method (ABMM). This epidemic model simulates the dynamics of patient populations, bacterial contamination, and healthcare worker safety under varying conditions. This model provides critical insights into the interactions between hospital practices, environmental factors, and infection dynamics, demonstrating the importance of symmetry in balancing hospital admission and discharge rates to manage infection spread effectively. The analysis extends to the impact of environmental bacterial density and hospital admission rates on patient colonization. Increasing admission rates introduce more susceptible patients, exacerbating infection spread when bacterial density is high. Conversely, lower admission rates and bacterial density result in a more controlled infection environment. The model further investigates how varying discharge rates influence colonization dynamics, highlighting that effective discharge practices can mitigate infection spread, especially in high-bacterial density scenarios. It must be noted that this model is studied fractionally for the first time. Overall, this model provides critical insights into the interactions between hospital practices, environmental factors, and infection dynamics, offering valuable guidance for infection control strategies and hospital policy formulation. By adjusting fractional order constant (σ) values and analyzing different scenarios, this research aids in understanding and managing bacterial infections in healthcare settings. The proposed method is able to provide the results presented in the figures within this study considering the influence of many factors.

Keywords: Methicillin-resistant *Staphylococcus aureus*; Adams–Bashforth–Moulton method; Antibiotic exposure; Admission rate; Discharge rate

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1. Introduction

Nosocomial infections (NIs) are major contributors to morbidity and mortality. These infections significantly prolong hospital stays and escalate healthcare costs [1]. According to the Centers for Disease Control and Prevention (CDC), "every year, at least 2 million Americans become infected with antibiotic-resistant bacteria, with approximately 23,000 people dying as a direct result of these infections" (CDC, 2018) [2]. In this context, NIs are catheter-associated urinary tract infections (CAUTIs), surgical site infections (SSIs), central line-associated bloodstream infections (CLABSIs), and ventilator-associated pneumonia (VAP). Primary sources of bacterial NIs have been identified, including surgical procedures and invasive devices like catheters and ventilators [3]. These infections are a significant concern for public health due to their prevalence, the potential for severe

outcomes, and the difficulty in treating some cases because of antibiotic resistance. They contribute significantly to healthcare costs and can prolong hospital stays, and reducing their incidence requires strict adherence to infection control measures across all healthcare settings [4].

Several bacterial pathogens have been associated with NIs, with Methicillin-Resistant Staphylococcus Aureus (MRSA) being a major cause in healthcare systems [5]. MRSA is considered an essential reason for hospital-associated infections, particularly among newborns and patients in intensive care units (CDC, 2019) [6]. This pathogen causes a wide range of infections, including skin and soft tissue infections, bloodstream infections, pneumonia, brain abscesses and endophthalmitis (CDC, 2018) [2]. Also, MRSA colonization can occur in different body parts, such as the axillae, groin, perineum, rectum, and anterior nares. These infections are associated with two significant challenges: the growing resistance of bacteria to available antibiotics and the difficulty in controlling their spread. Effective control is hindered by the frequent contamination of surfaces in healthcare facilities and the sharing of medical equipment, which often leads to outbreaks in these environments [7].

Worldwide, the common treatment for staph bacterial infections is the use of antibiotics. However, the overuse of antibiotics has led to MRSA becoming resistant to common antibiotics [8]. Up to 50% of antibiotic use in hospitalized patients is unnecessary or inappropriate (CDC, 2018). MRSA prevalence increased from 4% in the 1980s to 50% in the late 1990s, with MRSA strains accounting for up to 80% of all *S. aureus* strains, as in the NNISS report in [9,10]. In fact, about 64% of MRSA-infected patients are likely to die compared to non-resistant bacterial infection patients (WHO, 2014) [11]. Furthermore, MRSA has emerged as a major pathogen outside of healthcare settings, spreading within local communities and causing infections in healthy individuals [12].

MRSA changes at a constant rate and adapts to newly developed antibiotics, making it challenging for researchers to cope with it. Another perspective for controlling the spread of MRSA is to understand the association between patients' antibiotic exposure and their treatment outcomes. Many studies have observed that patients exposed to antibiotics are more likely to contract MRSA, which results in longer hospital stays, higher chances of treatment failure, increased healthcare costs, greater bacterial shedding into the environment, and higher mortality rates [13,14]. Hence, it is important to consider antibiotic exposure and the uncontrolled use of antibiotics in hospitals as influential factors in the transmission of MRSA. From this perspective, previous works presented a strong correlation between antibiotic use and MRSA infection in healthcare systems [14–17].

Another important factor to consider in studying MRSA is its ability to survive on different environmental surfaces including door handles, healthcare facility equipment, and healthcare workers' gloves [18,19]. This environmental persistence underlines the necessity of also examining environmental contamination as an important factor in the transmission of MRSA in hospital settings and healthcare systems.

Considering these factors, mathematical modeling is commonly employed to enhance our understanding of how nosocomial pathogens spread within hospital settings. Such models can help quantify the infectiousness of MRSA and predict its transmission dynamics within healthcare environments so that new control measurements can be deployed. From this perspective, several models were developed to analyze the effect of different factors contributing to MRSA infection in healthcare systems [19–31]. These models have forecasted the impact of infection control measures on reducing nosocomial cross-transmission and helping control MRSA outbreaks in healthcare facilities. For instance, one major factor identified for the direct transmission of MRSA was the contamination of healthcare worker's hands [32–37]. Other models have analyzed the effects of antibiotic exposure on MRSA transmission within healthcare systems [13]. However, many of these models have focused on the impact of individual factors on MRSA spread and transmission without considering the combined effects of multiple factors. Therefore, this study aims to develop a fractional model to examine the effect of environmental contamination combined with antibiotic exposure on the transmission dynamics of MRSA in hospitals. In recent years,

FDEs have emerged as a significant tool in mathematical modeling [38]. These equations are at least as stable as their integer-order counterparts, namely ordinary differential equations [39]. As a result, fractional-order calculus has garnered substantial attention across various scientific fields [40–44]. Biology, in particular, provides a rich source of mathematical concepts, as many biological systems exhibit memory or after-effects. Modeling such systems with FDEs offers advantages over traditional integer-order models, which often overlook these effects [40], making FDEs especially suitable for studying infectious diseases in hospital environments.

2. Mathematical Models and Preliminaries

Mathematical modeling research improves our knowledge about practical applications [45], disease spread predictions [46], epidemiology [47], and systems biology [48]. Fractional-order models are used to describe the spread of infectious diseases, capturing long-term dependencies and memory effects in disease transmission. For example, they have been applied to model the spread of COVID-19, influenza, and Ebola [49–53]. Also, Zeng et al. [54] demonstrated the use of a fractional system in understanding the dynamics of antibiotic resistance by considering historical antibiotic usage and resistance development over time. Hospitals are critical settings for the transmission of infectious diseases, and fractional-order models can significantly enhance the understanding and management of infection control in these environments. Fractional-order models help in analyzing the spread of HAIs, such as MRSA and C. difficile, considering the complex interactions and memory effects of patient-to-patient transmission and environmental contamination [55,56].

To mathematically describe the transmission of MRSA, a deterministic model is derived based on the following descriptions and assumptions:

$$
\begin{aligned}
{}^{C}D_t^{\sigma} P_u &= \theta_u(\gamma_u P_u + \gamma_c P_c + \gamma_v P_{uA} + \gamma_d P_{cA}) - \alpha_p \beta_p (1-\eta) P_u H_c - \kappa_p P_u B_e - (\gamma_u + \epsilon) P_u,\\
{}^{C}D_t^{\sigma} P_c &= \theta_c(\gamma_u P_u + \gamma_c P_c + \gamma_v P_{uA} + \gamma_d P_{cA}) + \alpha_p \beta_p (1-\eta) P_u H_c + \kappa_p P_u B_e - (\gamma_c + \epsilon) P_c,\\
{}^{C}D_t^{\sigma} P_{uA} &= \theta_v(\gamma_u P_u + \gamma_c P_c + \gamma_v P_{uA} + \gamma_d P_{cA}) - \alpha_p \beta_{pA} (1-\eta) P_{uA} H_c - \kappa_q P_{uA} B_e - \gamma_v P_{uA} + \epsilon P_u,\\
{}^{C}D_t^{\sigma} P_{cA} &= \theta_d(\gamma_u P_u + \gamma_c P_c + \gamma_v P_{uA} + \gamma_d P_{cA}) + \alpha_p \beta_{pA} (1-\eta) P_{uA} H_c + \kappa_q P_{uA} B_e - \gamma_d P_{cA} + \epsilon P_c,\\
{}^{C}D_t^{\sigma} H_u &= -\alpha_p \beta_h (1-\eta) P_c H_u - \alpha_p \beta_{hA} (1-\eta) P_{cA} H_u - \kappa_h H_u B_e + \mu_c H_c,\\
{}^{C}D_t^{\sigma} H_c &= \alpha_p \beta_h (1-\eta) P_c H_u + \alpha_p \beta_{hA} (1-\eta) P_{cA} H_u + \kappa_h H_u B_e - \mu_c H_c,\\
{}^{C}D_t^{\sigma} B_e &= v_p P_c + v_q P_{cA} + v_h H_c - \gamma_b B_e.
\end{aligned}
\tag{1}
$$

We categorize the patients (P), healthcare workers (HCWs), and free-living bacteria in the environment into the following seven sections [43]; $P_u(t)$—number of uncolonized patients without antibiotic exposure at time t; $P_{uA}(t)$—number of uncolonized patients with antibiotic exposure at time t; $P_c(t)$—number of colonized patients without antibiotic exposure at time t; $P_{cA}(t)$—number of colonized patients with antibiotic exposure at time t; $H_u(t)$—number of uncontaminated healthcare workers at time t; $H_c(t)$—number of contaminated healthcare workers at time t; $B_e(t)$—density of the free-living bacteria in the environment at time t, that are demonstrated in Figure 1.

The parameters used in the deterministic model are are demonstrated in Figure 1 as follows. σ typically represents the transition rate of various patient populations and healthcare worker transitions over time, including colonization rate (the rate at which uncolonized patients become colonized), contamination rate (the rate at which healthcare workers become contaminated), and recovery rate (the rate at which patients recover from colonization or contamination). The hospital admission rate θ (the rate at which new patients are admitted to the hospital) and discharge rate λ (the rate at which patients are discharged from the hospital) were studied to develop more effective prevention and control strategies.

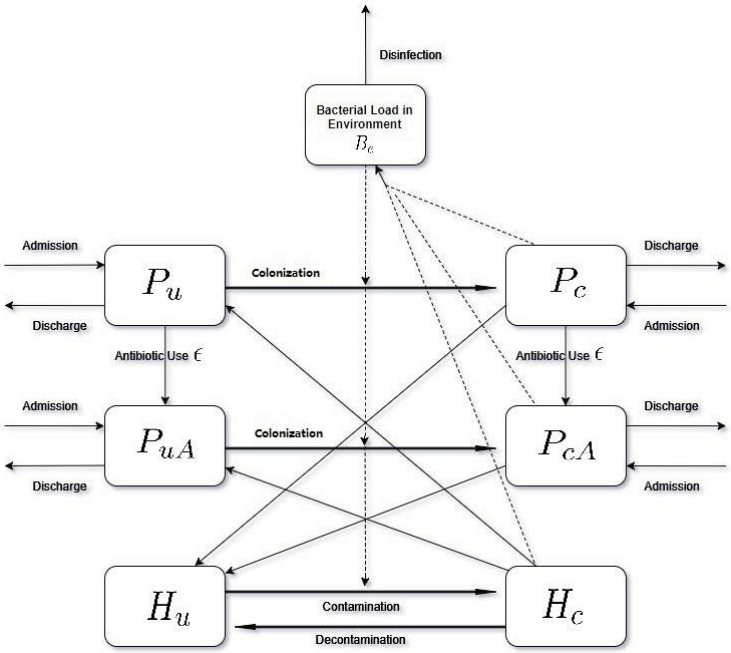

Figure 1. Flowchart of the seven sections in the model: $P_u(t)$, $P_{uA}(t)$, $P_c(t)$, $P_{cA}(t)$, $H_u(t)$, $H_c(t)$ and $B_e(t)$.

In epidemiological models, σ often represents the rate at which patients transition from being susceptible to becoming colonized. Adjusting σ in deterministic epidemic models provides critical insights into the dynamics of patient populations, the spread of contamination among healthcare workers, and the environmental persistence of bacteria. By simulating various scenarios with different σ values, healthcare professionals can better understand the processes at play, evaluate the effectiveness of treatment and infection control strategies, and make informed decisions to manage and mitigate unwanted outcomes in healthcare settings. This approach enhances our knowledge of epidemic dynamics as well as information about the aids in the development of targeted interventions to improve patient safety and infection control [57–59]. The number of colonized patients without antibiotic exposure $P_c(t)$ increases as susceptible individuals become colonized. Antibiotics reduce the number of uncolonized patients $P_u(t)$ transitioning to colonized states. $P_{uA}(t)$ and $P_{cA}(t)$ provide a clearer picture of how antibiotic use impacts MRSA dynamics. Studies such as those by [16,60] support our findings that antibiotic use can increase the prevalence of MRSA colonization. Antibiotics can disrupt normal flora and reduce competition, allowing MRSA to colonize more easily. The peak number of colonized patients (both with and without antibiotic exposure) occurs at different times. The peak for $P_c(t)$ typically occurs earlier and is higher compared to $P_{cA}(t)$ due to the absence of antibiotics slowing down the spread. Over time, the model reaches a steady state where the number of new colonizations balances out with recoveries and isolation. This steady state shows the long-term impact of antibiotics on reducing the prevalence of colonized patients. The rapid spread among patients not exposed to antibiotics leads to an earlier and higher peak in colonization. Without antibiotics, MRSA can spread more quickly among patients because antibiotics would otherwise kill susceptible bacteria, potentially reducing the spread. The density of MRSA bacteria in the environment $B(t)$ increases as the number of colonized patients rises. Rapid initial rise due to high transmission rates from colonized patients. The peak density of bacteria corresponds closely with the peak prevalence of colonized patients, which indicates the importance of controlling patient colonization to manage environmental contamination. The maximum environmental bacterial load is observed when the highest number of patients are colonized, indicating a strong temporal correlation. There is a direct correlation between the number of patients carrying MRSA and the bacterial load in the environment [19]. Colonized patients shed MRSA through skin

cells, nasal secretions, and other bodily fluids, contaminating surfaces and the air around them. Colonized individuals can spread MRSA through direct contact or indirectly via contaminated surfaces and objects, leading to a quick escalation in environmental bacterial density. It is essential to maintain a clean and MRSA-free environment to prevent the further spread of the bacteria, protecting uncolonized patients and staff [18,61].

Deterministic epidemic models are invaluable tools in epidemiology for understanding the complex interactions between environmental factors and patient populations. Specifically, these models can explore how environmental bacterial density influences patient colonization under varying hospital admission and discharge rates. By adjusting parameters related to bacterial density and hospital dynamics, these models provide insights into infection control and patient management in healthcare settings. In deterministic models, environmental bacterial density can be modeled as a time-dependent function or a constant parameter. For example, the model might include a term where the rate of transition from susceptible to colonized is proportional to the bacterial density in the environment. This approach allows researchers to simulate various scenarios, such as increased bacterial load due to insufficient cleaning or environmental contamination [62]. This study emphasizes the impact of environmental bacterial load on infection rates and supports the use of models to understand these dynamics. On the other hand, hospital admission rates affect the influx of new patients who are either susceptible to colonization or already colonized. Deterministic models incorporate admission rates to simulate the impact of varying patient inflow on overall colonization rates. High admission rates can lead to increased patient turnover and a higher risk of introducing and spreading infections within the hospital [63]. This paper discusses how varying hospital admission rates impact infection dynamics and supports the use of modeling to evaluate these effects. Hospital discharge rates influence the turnover of patients who are either colonized or susceptible. High discharge rates can affect the overall number of colonized patients in the hospital and influence the spread of infections. For instance, rapid discharges may lead to an increased risk of patients carrying infections being released into the community or other healthcare settings [64]. This research explores how different discharge practices influence infection spread, highlighting the relevance of discharge rate modeling by integrating varying hospital admission and discharge rates with environmental bacterial density in deterministic models, which allows researchers to gain a comprehensive view of how these factors interact to influence patient colonization. For example, a scenario with high bacterial density and high admission rates might show a rapid increase in colonized patients, while high discharge rates could mitigate this effect by reducing patient numbers. Understanding these interactions helps in formulating effective infection control strategies. For instance, a hospital facing high bacterial density might need to enhance cleaning protocols or adjust admission and discharge practices to manage infection risks more effectively [65]. This review discusses the interplay between environmental factors and hospital practices, emphasizing the importance of such modeling approaches.

There have been several recent proposals for fractional calculus topics, including different definitions [66,67].

Definition 1. *The fractional integral operator of order $\sigma > 0$, of a function $P(t) \in C_\iota, \iota \geq -1$ is denoted by I_a^σ and defined as follows:*

$$\begin{cases} I_a^\sigma P(t) = \frac{1}{\Gamma(\sigma+1)} \int_a^t P(t)(dt)^\sigma = \frac{1}{\Gamma(\sigma)} \int_a^t (t-\xi)^{\sigma-1} P(\xi) d\xi, \quad t, \sigma > 0 \\ I_a^0 P(t) = P(t). \end{cases} \tag{2}$$

Definition 2. *The definition of the Caputo operator may be expressed as follows:*

$${}^C D_t^\sigma P(t) = \frac{\partial^\sigma P(t)}{\partial t^\sigma} = \frac{1}{\Gamma(m-\sigma)} \int_0^t (t-\xi)^{m-\sigma-1} \frac{\partial^m P(t)}{\partial \xi^m} d\xi, (m-1 < \sigma \leq m, \quad m \in \mathbb{N}). \tag{3}$$

3. Mathematical Algorithm

Many practical and physical applications created using fractional PDEs are not exactly solvable. Still, in science and engineering, a numerical estimate of the solution is often enough to solve the issue. This approach may be used to register a solution for the Adams–Bashforth–Moulton method, utilizing the indicator corrector PECE methodology. To show the potentiality, commonality, and supremacy of our method, we investigate the integer order model, defining the transition between compartments under the Caputo fractional derivative in this section. All analytical and numerical calculations were performed during the calculation period using the MATLAB software program.

The Caputo fractional derivative is used to analyze the following generic fractional differential equation with fading memory:

$$\begin{cases} {}^{C}D_{t}^{\sigma}A(t) = \Upsilon(t, A(t)), \\ A(0) = A_0. \end{cases} \tag{4}$$

By stratifying the essential theorem of calculus, we transform Equation (4) into the following:

$$A(t) - A(0) = \frac{1}{\Gamma(\sigma)} \int_0^t \Upsilon(\chi, A(\chi))(t-\chi)^{\sigma-1} d\chi, \tag{5}$$

so that, at $t = t_{n+1}, n = 1, 2, \cdots$, we obtain

$$A(t_{n+1}) - A(0) = \frac{1}{\Gamma(\sigma)} \int_0^{t_{n+1}} \Upsilon(t, A(t))(t_{n+1}-t)^{\sigma-1} dt, \tag{6}$$

and

$$A(t_n) - A(0) = \frac{1}{\Gamma(\sigma)} \int_0^{t_n} \Upsilon(t, A(t))(t_n-t)^{\sigma-1} dt. \tag{7}$$

By subtracting (7) from (6), we obtain the following:

$$A(t_{n+1}) - A(t_n) = \frac{1}{\Gamma(\sigma)} \int_0^{t_{n+1}} \Upsilon(t, A(t))(t_{n+1}-t)^{\sigma-1} dt + \frac{1}{\Gamma(\sigma)} \int_0^{t_n} \Upsilon(t, A(t))(t_n-t)^{\sigma-1} dt \tag{8}$$

This suggests the following:

$$A(t_{n+1}) - A(t_n) = \mathcal{A}_\sigma + \mathcal{B}_\sigma, \tag{9}$$

where

$$\mathcal{A}_\sigma = \frac{1}{\Gamma(\sigma)} \int_0^{t_{n+1}} \Upsilon(t, A(t))(t_{n+1}-t)^{\sigma-1} dt,$$

and

$$\mathcal{B}_\sigma = \frac{1}{\Gamma(\sigma)} \int_0^{t_n} \Upsilon(t, A(t))(t_n-t)^{\sigma-1} dt.$$

By applying the Lagrange interpolation on the function $\Upsilon(t, A(t))$, we obtain

$$\begin{aligned}\mathcal{P}(t) &\cong \frac{t-t_{n-1}}{t_n-t_{n-1}}\Upsilon(t_n,A_n)+\frac{t-t_n}{t_{n-1}-t_n}\Upsilon(t_{n-1},A_{n-1})\\ &= \frac{\Upsilon(t_n,A_n)}{h}(t-t_{n-1})+\frac{\Upsilon(t_{n-1},A_{n-1})}{h}(t-t_n).\end{aligned} \tag{10}$$

Then, we have

$$\begin{aligned}\mathcal{A}_\sigma &= \frac{\Upsilon(t_n,A_n)}{h\Gamma(\sigma)}\int_0^{t_{n+1}}(t-t_{n-1})(t_{n+1}-t)^{\sigma-1}dt+\frac{\Upsilon(t_{n-1},A_{n-1})}{h\Gamma(\sigma)}\int_0^{t_{n+1}}(t-t_n)(t_{n+1}-t)^{\sigma-1}dt\\ &= \frac{\Upsilon(t_n,A_n)}{h\Gamma(\sigma)}\int_0^{t_{n+1}}(t_{n+1}-A-t_{n-1})A^{\sigma-1}dA+\frac{\Upsilon(t_{n-1},A_{n-1})}{h\Gamma(\sigma)}\int_0^{t_{n+1}}(t_{n+1}-A-t_n)A^{\sigma-1}dt.\end{aligned} \tag{11}$$

Subsequently,

$$\mathcal{A}_\sigma = \frac{\Upsilon(t_n,A_n)}{h\Gamma(\sigma)}\left\{\frac{2ht_{n+1}^\sigma}{\sigma}-\frac{t_{n+1}^{\sigma+1}}{\sigma+1}\right\}-\frac{\Upsilon(t_{n-1},A_{n-1})}{h\Gamma(\sigma)}\left\{\frac{ht_{n+1}^\sigma}{\sigma}-\frac{t_{n+1}^{\sigma+1}}{\sigma+1}\right\}. \tag{12}$$

Similarly, we obtain

$$\begin{aligned}\mathcal{B}_\sigma &= \frac{\Upsilon(t_n,A_n)}{h\Gamma(\sigma)}\int_0^{t_n}(t-t_{n-1})(t_n-t)^{\sigma-1}dt+\frac{\Upsilon(t_{n-1},A_{n-1})}{h\Gamma(\sigma)}\int_0^{t_n}(t-t_n)(t_n-t)^{\sigma-1}dt\\ &= \frac{\Upsilon(t_n,A_n)}{h\Gamma(\sigma)}\int_0^{t_n}(t_n-A-t_{n-1})A^{\sigma-1}dA+\frac{\Upsilon(t_{n-1},A_{n-1})}{h\Gamma(\sigma)}\left(\frac{t_n^{\sigma+1}}{\sigma}\right).\end{aligned} \tag{13}$$

Subsequently,

$$\mathcal{B}_\sigma = \frac{\Upsilon(t_n,A_n)}{h\Gamma(\sigma)}\left\{\frac{2ht_n^\sigma}{\sigma}-\frac{t_n^{\sigma+1}}{\sigma+1}\right\}-\frac{\Upsilon(t_{n-1},A_{n-1})}{h\Gamma(\sigma)}\left(\frac{t_n^{\sigma+1}}{\sigma}\right). \tag{14}$$

Thus, the approximate solution is given as

$$A(t_{n+1}) = A(t_n)+\frac{\Upsilon(t_n,A_n)}{h\Gamma(\sigma)}\left\{\frac{2ht_{n+1}^\sigma}{\sigma}-\frac{t_{n+1}^{\sigma+1}}{\sigma+1}+\frac{2ht_n^\sigma}{\sigma}-\frac{t_n^{\sigma+1}}{\sigma+1}\right\}-\frac{\Upsilon(t_{n-1},A_{n-1})}{h\Gamma(\sigma)}\left\{\frac{ht_{n+1}^\sigma}{\sigma}-\frac{t_{n+1}^{\sigma+1}}{\sigma+1}+\frac{t_n^{\sigma+1}}{\sigma}\right\}. \tag{15}$$

Consequently, the solution of the model (1) is

$$\begin{aligned}A_{n+1} &= A_n+\frac{1}{h\Gamma(\sigma)}\{\Theta_n\{\theta_u(\gamma_uA_n+\gamma_cB_n+\gamma_vC_n+\gamma_dD_n)-\Lambda_1A_nF_n-\kappa_pA_nG_n-(\gamma_u+\epsilon)A_n\\ &\quad-\Theta_{n-1}\{\theta_u(\gamma_uA_{n-1}+\gamma_cB_{n-1}+\gamma_vC_{n-1}+\gamma_dD_{n-1})-\Lambda_1A_{n-1}F_{n-1}-\kappa_pA_{n-1}G_{n-1}-(\gamma_u+\epsilon)A_{n-1}\}\end{aligned} \tag{16}$$

$$\begin{aligned}B_{n+1} &= B_n+\frac{1}{h\Gamma(\sigma)}\{\Theta_n\{\theta_c(\gamma_uA_n+\gamma_cB_n+\gamma_vC_n+\gamma_dD_n)+\Lambda_1A_nF_n+\kappa_pA_nG_n-(\gamma_c+\epsilon)B_n\}\\ &\quad-\Theta_{n-1}\{\theta_c(\gamma_uA_{n-1}+\gamma_cB_{n-1}+\gamma_vC_{n-1}+\gamma_dD_{n-1})+\Lambda_1A_{n-1}F_{n-1}+\kappa_pA_{n-1}G_{n-1}-(\gamma_c+\epsilon)B_{n-1}\}\}\end{aligned} \tag{17}$$

$$\begin{aligned}C_{n+1} &= C_n+\frac{1}{h\Gamma(\sigma)}\{\Theta_n\{\theta_v(\gamma_uA_n+\gamma_cB_n+\gamma_vC_n+\gamma_dD_n)-\Lambda_2C_nF_n-\kappa_qC_nG_n-\gamma_vC_n+\epsilon A_n\}\\ &\quad-\Theta_{n-1}\{\theta_v(\gamma_uA_{n-1}+\gamma_cB_{n-1}+\gamma_vC_{n-1}+\gamma_dD_{n-1})-\Lambda_2C_{n-1}F_{n-1}-\kappa_qC_{n-1}G_{n-1}-\gamma_vC_{n-1}+\epsilon A_{n-1}\}\}\end{aligned} \tag{18}$$

$$\begin{aligned} D_{n+1} &= D_n + \frac{1}{h\Gamma(\sigma)}\{\Theta_n\{\theta_d(\gamma_u A_n + \gamma_c B_n + \gamma_v C_n + \gamma_d D_n) + \Lambda_2 C_n F_n + \kappa_q C_n G_n - \gamma_d D_n + \epsilon B_n\} \\ &\quad -\Theta_{n-1}\{\theta_d(\gamma_u A_{n-1} + \gamma_c B_{n-1} + \gamma_v C_{n-1} + \gamma_d D_{n-1}) + \Lambda_2 C_{n-1} F_{n-1} + \kappa_q C_{n-1} G_{n-1} - \gamma_d D_{n-1} + \epsilon B_{n-1}\}\} \end{aligned} \tag{19}$$

$$\begin{aligned} E_{n+1} &= E_n + \frac{1}{h\Gamma(\sigma)}\{\Theta_n\{-\Lambda_3 B_n E_n - \Lambda_4 D_n E_n - \kappa_h E_n G_n + \mu_c F_n\} \\ &\quad -\Theta_{n-1}\{-\Lambda_3 B_{n-1} E_{n-1} - \Lambda_4 D_{n-1} E_{n-1} - \kappa_h E_{n-1} G_{n-1} + \mu_c F_{n-1}\}\} \end{aligned} \tag{20}$$

$$\begin{aligned} F_{n+1} &= F_n + \frac{1}{h\Gamma(\sigma)}\{\Theta_n\{\Lambda_3 B_n E_n + \Lambda_4 D_n E_n + \kappa_h E_n G_n - \mu_c F_n\} \\ &\quad -\Theta_{n-1}\{\Lambda_3 B_{n-1} E_{n-1} + \Lambda_4 D_{n-1} E_{n-1} + \kappa_h E_{n-1} G_{n-1} - \mu_c F_{n-1}\}\} \end{aligned} \tag{21}$$

$$G_{n+1} = G_n + \frac{1}{h\Gamma(\sigma)}\{\Theta_n\{v_p B_n + v_q D_n + v_h F_n - \gamma_b G_n\} - \Theta_{n-1}\{v_p B_{n-1} + v_q D_{n-1} + v_h F_{n-1} - \gamma_b G_{n-1}\}\} \tag{22}$$

where $A_n = A_n(t_n)$, $A_{n-1} = A_{n-1}(t_{n-1})$, $\Theta_n = \frac{2ht_{n+1}^{\sigma}}{\sigma} - \frac{t_{n+1}^{\sigma+1}}{\sigma+1} + \frac{2ht_n^{\sigma}}{\sigma} - \frac{t_n^{\sigma+1}}{\sigma+1}$, $\Theta_{n-1} = \frac{ht_{n+1}^{\sigma}}{\sigma} - \frac{t_{n+1}^{\sigma+1}}{\sigma+1} + \frac{t_n^{\sigma+1}}{\sigma}$, $\Lambda_1 = \alpha_p\beta_p(1-\eta)$, $\Lambda_2 = \alpha_p\beta_{pA}(1-\eta)$, $\Lambda_3 = \alpha_p\beta_h(1-\eta)$, $\Lambda_4 = \alpha_p\beta_{hA}(1-\eta)$, $A = P_u$, $B = P_c$, $C = P_{uA}$, $D = P_{cA}$, $E = H_u$, $F = H_c$, and $G = B_e$.

4. Numerical Manipulations

Here, the numerical simulation is studied over 200 days, and for more detail, a period of 5 days is considered too. The data were collected from Beijing Tongren Hospital, where the emergency ward had 23 initially full beds, and the initial amount of patients and healthcare workers was estimated. We assume the initial density of bacteria is 1000 ACC/cm^2. The data used in this paper were taken from reference [41]. The initialvalues were set as ($P_{u0} = 4$; $P_{uA0} = 6$, $P_{c0} = 7$, $P_{cA0} = 6$, $H_{u0} = 17$, $H_{c0} = 6$, and $B_{e0} = 1000$).

In this context, by understanding patient behavior in a healthcare setting, we can analyze how different populations such as uncolonized patients, colonized patients, or healthcare workers evolve over time under varying conditions. The fractional order σ plays a crucial role in this analysis. To better understand the impact of different σ values on these populations, we create plots that show how each population evolves over time. The following plots will help you see how quickly or slowly the population increases or decreases under different conditions.

The solutions of uncolonized patients without antibiotic exposure $P_u(t)$, with initial values ($P_{u0} = 4$), and with different values of the fractional order σ are given in Figure 2. The parameters have been considered in history [41].

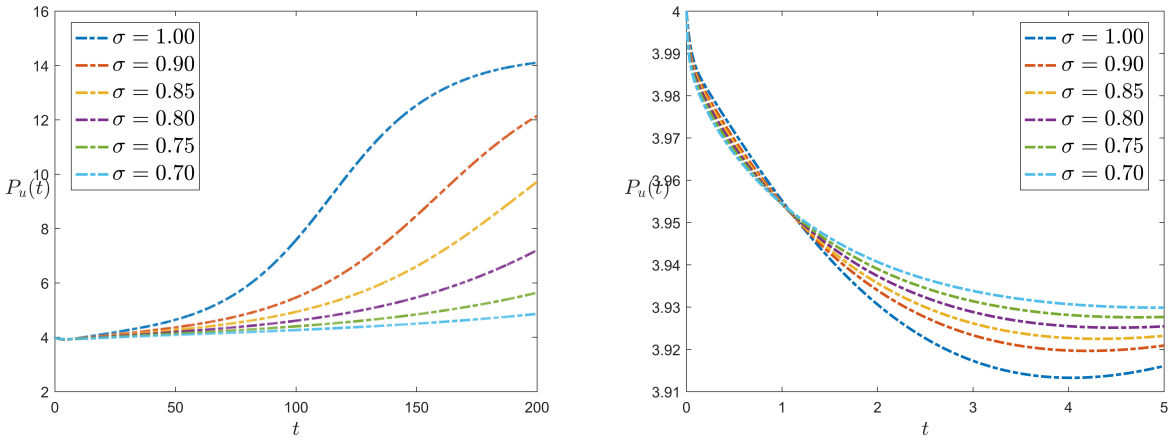

Figure 2. $P_u(t)$ as a function of time for different fractional order σ.

The model predicts an exponential increase in the number of patients, peaking at approximately 40 days with approximately 25% of the population infected when σ equals one, and the number of uncolonized patients decreases rapidly when σ values become smaller as in Figure 2.

The uncolonized patients with antibiotic exposure solutions $P_{uA}(t)$, with initial values ($P_{uA0} = 6$), and with different values of the parameter σ are shown in Figure 3.

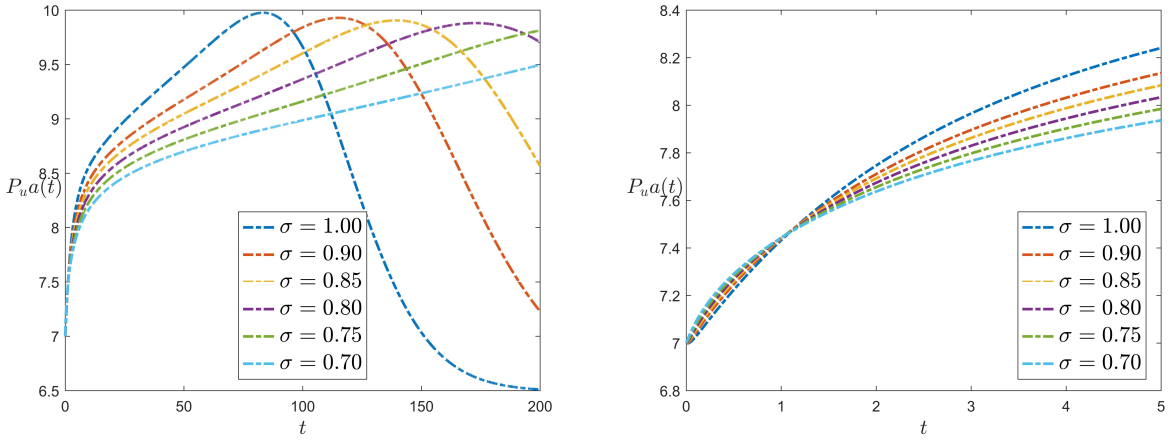

Figure 3. $P_{uA}(t)$ as a function of time with different values of the fractional order σ.

For the uncolonized patients with antibiotic exposure P_{uA}, the higher σ values might lead to faster depletion of the uncolonized population, as shown in Figure 3.

The solutions of colonized patients without antibiotic exposure $P_c(t)$, considering ($P_{c0} = 7$) as the initial value for different values of the parameter σ, are plotted in Figure 4.

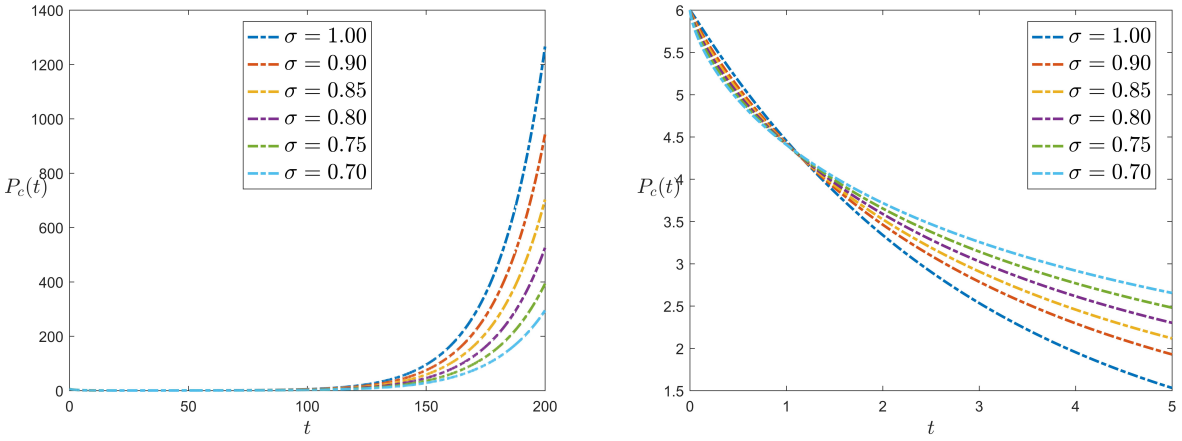

Figure 4. $P_c(t)$ as a function of time for different fractional order σ.

For colonized patients, and if σ is small, the population $P_c(t)$ decreases slowly, meaning patients remain colonized longer, and if σ is large, the colonized population $P_c(t)$ decreases rapidly, leading to a quicker decline in the number of colonized patients as in Figure 4.

The solutions of colonized patients with antibiotic exposure $P_{cA}(t)$, with initial values ($P_{cA0} = 6$), and with different values of the parameter σ are represented in Figure 5.

In Figure 5, if σ is small, the colonized population with antibiotic exposure $P_{cA}(t)$ decreases slowly, indicating that patients remain colonized longer despite antibiotic exposure, and if σ is large, the colonized population with antibiotic exposure $P_{cA}(t)$ decreases rapidly, leading to a quick decline, possibly due to effective antibiotic treatment or other factors.

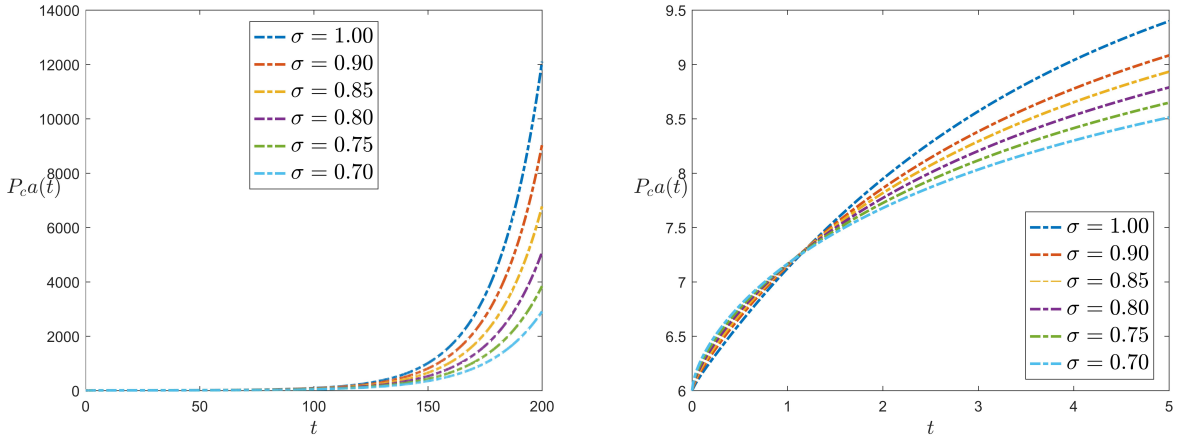

Figure 5. $P_{cA}(t)$ as a function of time for different fractional order σ.

Moreover, the solutions of uncontaminated healthcare workers, with an initial value ($H_0 = 17$) at any time t and with different values of the parameter σ, are considered in Figure 6.

For the number of uncontaminated healthcare workers $H(t)$ in this model with an initial value $H_0 = 17$ and different values of the parameter σ, with an increase in the value of sigma (if σ is large), the number of uncontaminated healthcare workers $H(t)$ decreases rapidly, indicating that contamination occurs quickly as in Figure 6.

Now, the solutions to the contaminated healthcare worker number with initial value ($H_0 = 17$) with different values of the parameter σ are shown in Figure 7.

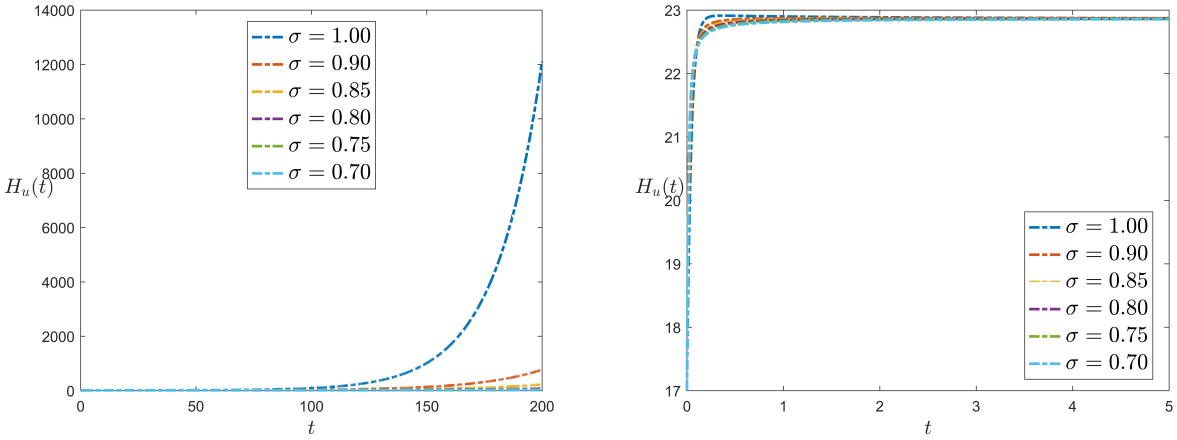

Figure 6. Uncontaminated healthcare workers as a function of time for different fractional order σ.

In Figure 7, the number of contaminated healthcare workers $H_c(t)$ increases slowly with small values of σ, meaning contamination spreads at a slower rate, and the number of contaminated healthcare workers $H_c(t)$ increases rapidly with large values of σ, indicating fast contamination

The solutions of free-living bacteria density in the environment as a function of time (t) with initial value ($B_0 = 1000$) and with different values of the parameter σ are represented in Figure 8.

In Figure 8, the number density of bacteria $B(t)$ grows slowly at small values of σ, and the bacterial density grows rapidly with large values of σ. By adjusting σ values in this model, you can simulate various scenarios, assess the impact of different treatment strategies on the population of colonized patients, understand the spread of contamination among healthcare workers, and predict the environmental persistence of bacteria. This model provides valuable insights into the dynamics of patient populations and other related

factors in healthcare settings. By analyzing how these populations evolve over time with different σ values, you can gain a deeper understanding of the processes at play and make informed decisions to control or mitigate unwanted outcomes.

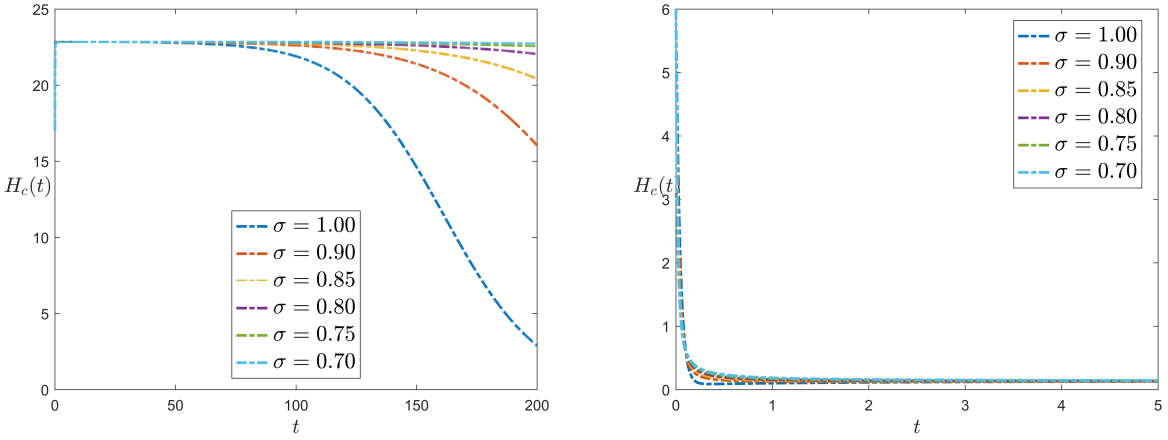

Figure 7. Contaminated healthcare workers as a function of time for different fractional order σ.

The deterministic epidemic model is often used in epidemiology to explore the effect of environmental bacterial density on patient colonization with varying hospital admission and discharge rates and helps to understand how changes in the environment and hospital practices affect the spread of bacterial infections among patients. This can also be used to explore how varying hospital admission rates affect patient colonization, considering the impact of environmental bacterial density.

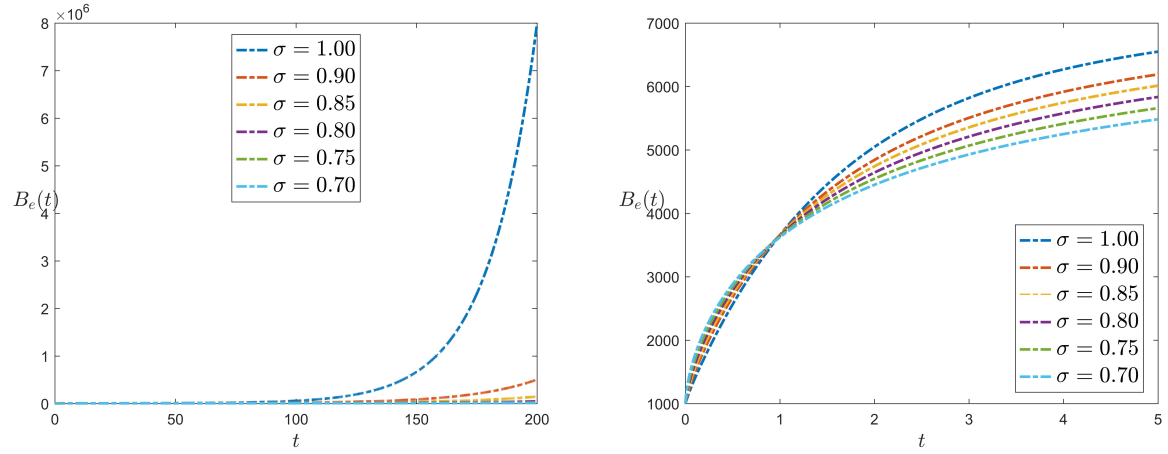

Figure 8. Number of free-living bacteria density in the environment as a function of time for different fractional order σ.

Also, the prevalence of uncolonized patients without antibiotics, uncolonized patients with antibiotic exposure, colonized patients without antibiotics, and colonized patients with antibiotic exposure over time under the effect of the density number of the free-living bacteria in the environment at time t at different hospital admission rates is studied in Figure 9.

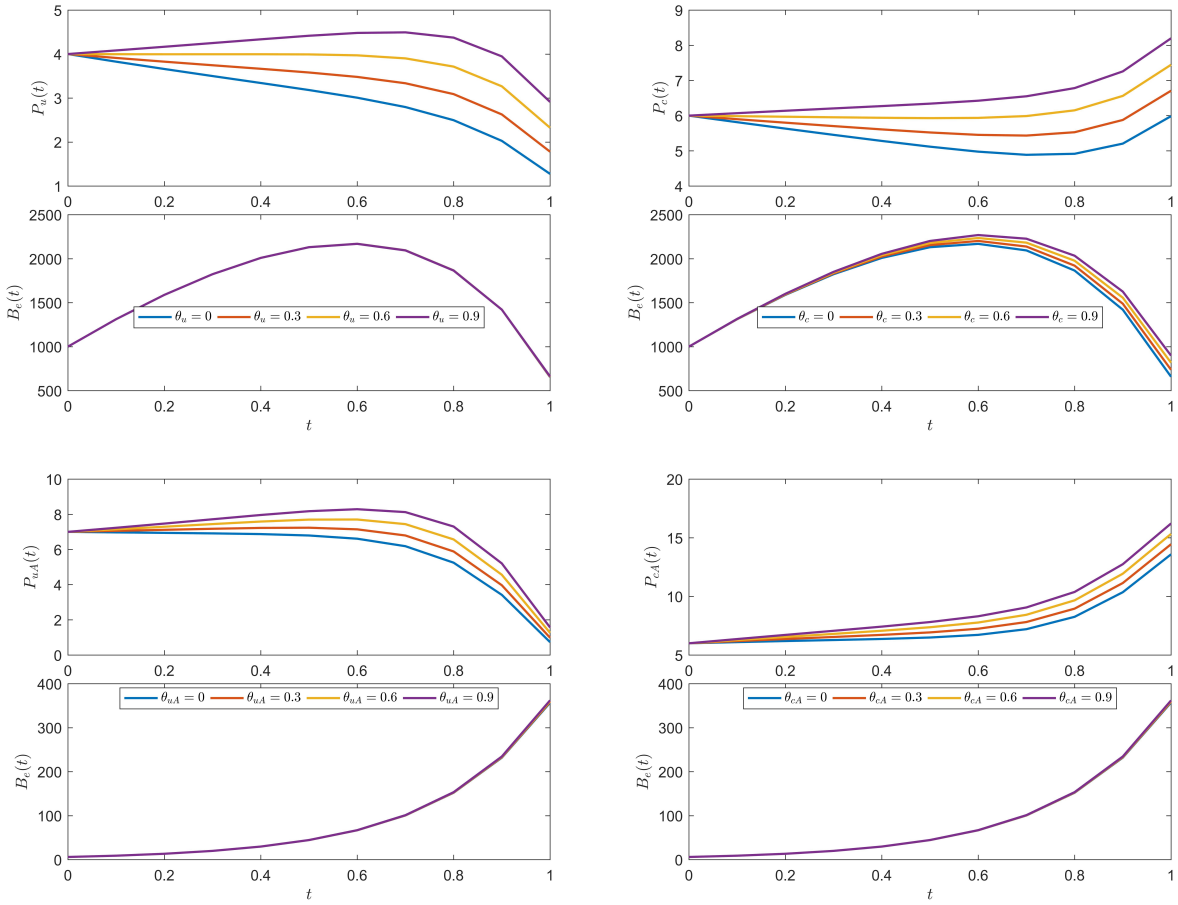

Figure 9. The prevalence of uncolonized patients without antibiotics, uncolonized patients with antibiotic exposure, colonized patients without antibiotics, and colonized patients with antibiotic exposure over time under the effect of the density number of the free-living bacteria in the environment at time t at different hospital admission rates.

In Figure 9, increasing the hospital admission rates introduces more susceptible patients into the system. This can lead to a higher number of infections if the bacterial density is also high. Increasing the environmental bacterial density generally leads to higher rates of patient colonization. This is because the probability of susceptible patients being exposed to and colonized by bacteria is higher when the environmental density is elevated. Also, when both admission rates and bacterial density are high, the system may experience a rapid increase in infections. A large number of susceptible patients combined with high exposure levels can lead to a significant outbreak. Conversely, low bacterial density and low admission rates generally lead to a lower number of infections and a more controlled spread. These results help in understanding how environmental factors and hospital practices interact to influence infection dynamics. They can guide hospital policies and infection control strategies, such as optimizing cleaning schedules and managing patient admissions.

Finally, the prevalence of uncolonized patients without antibiotics, uncolonized patients with antibiotic exposure, colonized patients without antibiotics, and colonized patients with antibiotic exposure over time under the effect of the density number of the free-living bacteria in the environment at time t at different hospital discharge rates is shown in Figure 10.

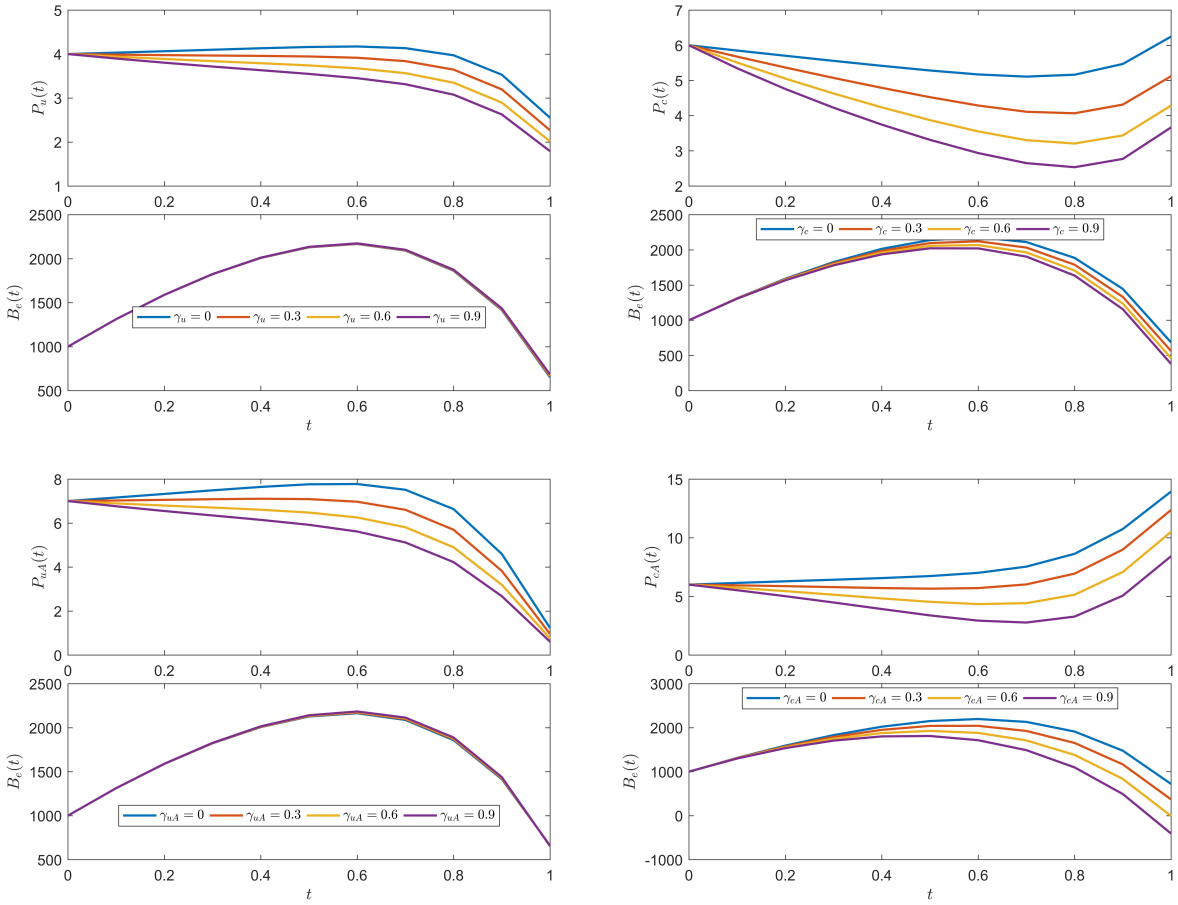

Figure 10. The prevalence of uncolonized patients without antibiotics, uncolonized patients with antibiotic exposure, colonized patients without antibiotics, and colonized patients with antibiotic exposure over time under the effect of the density number of the free-living bacteria in the environment at time t at different hospital discharge rates.

In Figure 10, the deterministic epidemic model can also be used to explore how varying hospital discharge rates affect patient colonization, considering the impact of environmental bacterial density. Increased environmental bacterial density leads to higher rates of colonization among susceptible patients, resulting in more infections. High bacterial density combined with effective discharge practices can help control the spread of infection, as patients who are at risk are promptly removed, and increased discharge rates reduce the number of patients in the hospital, which can decrease the overall number of infectious patients if the discharged patients are effectively removed from the system. On the other hand, high bacterial density with ineffective discharge practices can exacerbate infection rates, leading to outbreaks. These results help in understanding how environmental factors and hospital practices interact to influence infection dynamics. They can guide hospital policies and infection control strategies, such as optimizing cleaning schedules, managing patient admissions, and setting discharge protocols.

5. Conclusions

This study presents a comprehensive deterministic epidemic model that effectively simulates the complex interactions between patient populations, bacterial contamination, and healthcare worker safety. Deterministic epidemic models using the fractional Adams–Bashforth–Moulton method offer a robust framework for understanding hospital MRSA transmission dynamics. This model integrates the effects of antibiotic exposure, environmental contamination, and hospital admission and discharge rates to develop more

effective prevention and control strategies. They enable a detailed analysis of how various patient populations and healthcare workers transition between states over time, influenced by parameters such as the fractional order constant σ and environmental bacterial density. By simulating different scenarios and adjusting parameters, researchers and healthcare professionals can evaluate the impact of treatment and infection control strategies, helping to enhance patient safety and control MRSA outbreaks. The model underscores that higher σ values facilitate quicker depletion of uncolonized patients, while lower σ values prolong the duration of colonization, suggesting that recovery strategies must be tailored to these dynamics. Environmental factors such as bacterial density and hospital admission rates are significant; increased admissions in high-density environments exacerbate infection spread, whereas lower rates contribute to a more controlled situation. The insights gained from this study emphasize the importance of effective discharge practices and their role in mitigating infection spread in high bacterial density scenarios. Overall, this research provides essential guidance for developing targeted infection control strategies and hospital policies, contributing to improved healthcare outcomes and worker safety. By adjusting model parameters fractional order constant σ and exploring various conditions, we can better understand and manage bacterial infections in healthcare settings, ultimately enhancing patient care and safety. Future research should focus on refining parameter estimates and incorporating additional factors, such as patient movement and staff adherence to hygiene protocols, to further improve the effectiveness of these models.

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