Examining the Relationship Between Infection Power Rate and the Critical Threshold in Stochastic SIS Epidemic Modeling

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Abstract: The stochastic SIS epidemic model is well-known for its critical threshold $\mathcal{R}_s$, indicating the transition between disease eradication ($\mathcal{R}_s < 1$) and epidemic outbreaks ($\mathcal{R}_s > 1$). However, the scenario where $\mathcal{R}_s = 1$ has been uncertain. We present a definitive resolution to this pivotal issue. Additionally, we introduce advancements in analyzing the disease-free state of equilibrium when $\mathcal{R}_s < 1$ to deepen our understanding of the system dynamics. To validate our theoretical developments and provide visual evidence, extensive computer simulations are conducted, enhancing the comprehensiveness and applicability of our findings to the broader field of epidemiology and infectious disease modeling. The implications of our results extend to public health policies and interventions aimed at effectively managing and controlling infectious diseases in different communities where $\mathcal{R}_s$ hovers around the critical value.

Key-Words: SIS model, Stochastic epidemic models, Extinction of disease, Stability of disease, Lyapunov function, Threshold.

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1 Introduction

In the early 20th century, a transformative shift occurred in epidemiology with the pioneering work of renowned scientists, including Anderson Gray McKendrick and Janet-Leigh Claypon, who introduced mathematical modeling. Since then, mathematical modeling has become an indispensable tool, profoundly impacting outbreak and epidemic management and playing a crucial role in guiding evidence-based public health interventions. Epidemiology has evolved through the contributions of notable physicians like Quinto Tiberio Angelerio, who skillfully handled the plague outbreak in Alghero, Sardinia, for 1582. However, it was during the 19th century that modern epidemiology as a scientific discipline began to take shape. Often regarded as the father of modern epidemiology, John Snow made a pivotal discovery when he traced a devastating cholera outbreak in London to the contaminated water from the Broad Street pump. This groundbreaking investigation can be considered the seminal event that laid the foundation for the science of epidemiology as we know it today. Epidemiology is a scientific discipline that delves into the study of epidemics, diseases in general, and even health-related conditions unrelated to diseases. The origins of this field can be traced back to ancient Greece, where the renowned physician Hippocrates of Kos made significant contributions by distinguishing between epidemic and endemic diseases. Epidemiology extends beyond human health, examining diseases affecting plants and domestic and livestock animals. An epidemic is an abnormal and substantial upsurge of a particular disease within a population that occurs relatively quickly. The intricate process of disease transmission involves many influential factors, encompassing both the characteristics of the infectious agent and the complexities of the host population. Regarding the infectious agent, its inherent properties, such as the mode of transmission (e.g., respiratory droplets, direct contact), the duration of infectivity, and its response to medical interventions like treatments and vaccines, are critical determinants of its spread among individuals. Equally significant are the host population factors that contribute to the dynamics of an epidemic. Social interactions, demographics (e.g., age, gender), cultural practices, geographic distribution, and economic conditions all play pivotal roles in shaping the vulnerability and resilience of a population to the disease. Throughout the annals of recorded history, human civilization has grappled with recurrent epidemics and pandemics. These infectious disease outbreaks have inflicted profound human suffering, societal turbulence, and economic disruptions. In light of such formidable challenges, accurately predicting outbreak progression is paramount to effectively mitigating their adverse impacts. Central to this endeavor lies the field of epidemiologic modeling, which serves as a fundamental

tool in understanding the dynamics of disease transmission and devising informed strategies for containment and prevention. The impact of infectious diseases on human life is enormous. Each year, millions of people suffer or die from different diseases. There are many questions about the propagation of diseases. For instance, how many people will be touched together and thus need treatment? How long will the disease outbreak last? What is the potential for a vaccination strategy to reduce the seriousness of the epidemic? In epidemiology, the importance of mathematical modeling is significant because it can provide information about the mechanisms underlying the propagation of diseases and propose strategies for their control. The first mathematical model of epidemiology known was developed and solved by Daniel Bernoulli in 1760. Using compartmentalized models, the basics of modern mathematical epidemiology were established in the early 20th century, [1]. Mathematical epidemiology expanded exponentially. Various mathematical models were articulated and discussed, [2], [3], [4], [5], [6]. The classical SIS model for a constant population is

$$\begin{cases} dS = [\mu(1-S) + \gamma I - \beta SI]\, dt, \\ dI = [\beta SI - (\mu+\gamma)I]\, dt. \end{cases} \tag{1}$$

Nonetheless, real-world scenarios are often characterized by abundant stochastic elements and precarious occurrences. This underscores the significance of employing stochastic calculus as a robust methodology for elucidating these genuine random phenomena manifesting in the natural world. The integration of stochasticity into epidemic models and population dynamics has yielded compelling findings of notable consequence. The technique of parameter perturbation has been widely adopted by various researchers, [7], [8], [9], [10], [11], [12], to enhance their investigations. The studies, [6], [13], [14], specifically delved into color noise effects. Their studies meticulously scrutinized the intricate dynamics of an epidemic model under finite regime-switching conditions. Motivated by the insights gained from these preceding endeavors, our current research focuses on the influence of environmental fluctuations, [4], [6], [15], [16], [17], [18], [19]. These fluctuations are hypothesized to manifest as variations in the parameter β within the deterministic model (1) discussed earlier. As such, this study aims to unravel the implications of integrating these fluctuations. So that

$$\beta \longrightarrow \beta + \sigma \frac{dB}{dt}.$$

We establish the ensuing stochastic model by assimilating the described perturbations into the deterministic framework presented as equation (1).

$$\begin{cases} dS = [\mu(1-S) + \gamma I - \beta SI]\, dt - \sigma SI dB, \\ dI = [\beta SI - (\mu+\gamma)I]\, dt + \sigma SI dB. \end{cases} \tag{2}$$

In this work, we study the stochastic SIS epidemic model with the non-linearity power function, that is

$$\begin{cases} dS(t) = [\mu(1-S) + \gamma I - \beta S^p I]\, dt \\ \qquad\qquad -\sigma S^p I dB(t), \\ dI(t) = [\beta S^p I - (\mu+\gamma)I]\, dt \\ \qquad\qquad +\sigma S^p I dB(t), \end{cases} \tag{3}$$

where $p \geq 1$ is the non-linearity power constant. Therefore, it is enough to study the SDE for $I(t)$

$$\begin{aligned} dI(t) &= [\beta(1-I(t))^p I(t) - (\mu+\gamma)I(t)]\, dt \\ &\quad +\sigma(1-I(t))^p I(t) dB(t), \end{aligned} \tag{4}$$

where S and I are the numbers of susceptible and infected individuals. This model assumes a vital dynamic with a mortality rate corresponding to the birth rate, implying that $S + I = 1$. Besides, the parameter denoted as β pertains to the infection rate, representing the rate at which new infections are contracted. Conversely, γ corresponds to the recovery rate, delineating the pace at which individuals recuperate from the disease. A deterministic form of (1) is given $\mathcal{R}_0 = \frac{\beta}{\mu+\gamma}$. When the basic reproduction number $\mathcal{R}_0 \leq 1$, the globally asymptotically stable condition is achieved at the disease-free equilibrium state $E_0(1,0)$. Conversely, when $\mathcal{R}_0 > 1$, the stability of E_0 is compromised, leading to the emergence of an endemic equilibrium state $E* \left(\frac{1}{\mathcal{R}_0}, \frac{\mathcal{R}_0 - 1}{\mathcal{R}_0}\right)$ which attains global asymptotic stability. In the past few years, a number of mathematical programs for the transmission dynamics of infectious diseases have been proposed, [2], [4], [5], [6], [7], [8], [13], [20], [21], [22], [23], as SIS (Susceptible-Infectious-Susceptible), SIRS (Susceptible-Infectious-Reduced-Susceptible). The SIS models provide proper categorizations of human population dynamics for specific bacterial diseases such as malaria, certain protozoan diseases such as meningitis and some sexually diseases such as tuberculosis ("gonorrhea") in which individuals generally build up immunity to the disease within 24 hours and do not develop resistance to the disease when infected. Based on the following initial conditions (S_0, I_0) in the set

$$\Delta = \left\{ x \in \mathbb{R}^2_+;\ x_1 + x_2 = 1 \right\}.$$

Here, B is a Brownian motion on the probability space $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t\geq 0}, \mathbb{P})$ and $\sigma > 0$ indicates the intensity of the white noise. Next, the authors investigated the dynamic behavior of $I(t)$ as a function of

the new threshold

$$\mathcal{R}_s = \frac{\beta}{\mu+\gamma+\frac{1}{2}\sigma^2}.$$

They proved that if either $\mathcal{R}_s < 1$ and $\beta \geq \sigma^2$ or $\sigma^2 > \beta \vee \frac{\beta^2}{2(\mu+\gamma)}$, the disease will disappear. However, if $\mathcal{R}_s > 1$, then the disease will persist. In [24], the author also suggested that if $\mathcal{R}_s < 1$ and $\beta < \sigma^2 \leq \frac{\beta^2}{2(\mu+\gamma)}$, then the disease disappears with the probability 1. In this paper, we will assume that $\beta \geq \frac{1}{2}\sigma^2$ is not exactly a limitation because it indicates that the estimation error σ^2 is smaller than the estimated value β. We investigated the case where $\mathcal{R}_s \leq 1$. More precisely, we prove if $\mathcal{R}_s = 1$, E_0 is exponentially stable. Furthermore, the disease is extinct in the mean. The manuscript follows a structured approach, beginning with Section 2, which presents exponentially stable for the system described in equation 3. In Section 3, we establish sufficient conditions for disease extinction. Subsequently, Section 4 provides an in-depth discussion of our theoretical discoveries and includes numerical simulations to illustrate them. Finally, a concise conclusion succinctly outlines the primary contributions made in this study.

2 Exponentially stable

In this section, our objective is to analyze the stability of the disease within the SDE system (3) and establish the stochastic threshold condition for disease control or eradication.

Theorem 2.1 *Let $(S_0, I_0) \in \Delta$. If $\mathcal{R}_s < 1$, then for any n such as*

$$0 < n < 2\beta\sigma^{-2}\left(\mathcal{R}_S^{-1} - 1\right), \tag{5}$$

the solution $I(t)$ meets

$$\mathbb{E}\left(I^n(t)\right) \leq I_0^n \exp\left(-ct\right),$$

where

$$c = -n\left[\beta\left(1-\mathcal{R}_S^{-1}\right) + \frac{n}{2}\sigma^2\right] > 0. \tag{6}$$

Thus, the disease-free equilibrium state E_0 is n-th moment exponentially stable.

Proof. Let $V_1(I) = I^n$, $n > 0$ be any real constant verifying the condition (5). By the Itô formula we get for $p > 1$

$$dV_1(I) = \mathcal{L}I^n dt + n\sigma(1-I)^p I^n dB, \tag{7}$$

and

$$\begin{aligned}\mathcal{L}V_1(I) &= nI^n\Big[-(\mu+\gamma)+\beta(1-I)^p \\ &\quad +\frac{1}{2}\sigma^2(n-1)(1-I)^{2p}\Big], \\ &\leq nI^n\Bigg[\sup_{0<x\leq 1}\Big(-(\mu+\gamma)+\beta x^p \\ &\quad -\frac{1}{2}\sigma^2 x^{2p}\Big)+\frac{n}{2}\sigma^2\Bigg].\end{aligned} \tag{8}$$

We can show clearly that if $\beta \geq \frac{1}{2}\sigma^2$ and $\mathcal{R}_s < 1$ then

$$\begin{aligned}&\sup_{0<x\leq 1}\left(-(\mu+\gamma)+\beta x^p - \frac{1}{2}\sigma^2 x^{2p}\right) \\ &= \beta\left(1-\frac{1}{\mathcal{R}_s}\right),\end{aligned} \tag{9}$$

Combining this with (8) we get

$$\mathcal{L}I^n(t) \leq -cI^n(t),$$

where c can be found in (6). By injecting it into (7), then integrating the result and taking the expectations on both sides yields

$$\mathbb{E}V_1(I(t)) \leq V_1(I(0)) - c\int_0^t \mathbb{E}V_1(I(u))du,$$

which implies with the Gronwall inequality that

$$\mathbb{E}\left(I^n(t)\right) \leq I_0^n \exp\left(-ct\right).$$

3 Extinction of the disease

The subsequent theorems address the scenario where the stochastic threshold $\mathcal{R}_s = 1$.

Theorem 3.1 *For a given initial value $(S_0, I_0) \in \Delta$, if $\mathcal{R}_s = 1$, then the solution of the equation (3) follows*

$$\lim_{t\to\infty}\frac{1}{t}\int_0^t I(s)ds = 0. \tag{10}$$

Proof. Let $V_2(I) = \log(I)$. With the equation (3), $I \leq 1$, $\mathcal{R}_s = 1$, and the formula of Itô we get

$$\begin{aligned}dV_2(I) &= \Bigg[-(\mu+\gamma)+\beta(1-I)^p \\ &\quad -\frac{\sigma^2}{2}(1-I)^{2p}\Bigg]dt + \sigma(1-I)^p dB, \\ &\leq -p\left(\beta-\sigma^2\right)Idt + \sigma(1-I)^p dB.\end{aligned} \tag{11}$$

By integrating (11) and using Newton's binomial formula we obtain

$$\begin{aligned}\log I(t) &\leq -p\left(\beta-\sigma^2\right)\int_0^t I(s)ds+\log I(0)\\ &\quad+\sigma\int_0^t(1-I(s))^p dB_s. \qquad (12)\end{aligned}$$

By the large number theorem for martingales it exists $\Omega_1\subset\Omega$ with $\mathbb{P}(\Omega_1)=1$, so that for all $\omega\in\Omega_1$ and $\epsilon>0$, there exist $T(w,\epsilon)$ such that for any $t\geq T$ we get

$$\log I(0)+\sigma\int_0^t(1-I(s))^p dB(s)\leq\epsilon t,$$

which implies with (12) that

$$\begin{aligned}&\frac{1}{p\left(\beta-\sigma^2\right)}\frac{d}{dt}\left[\exp\left(p\left(\beta-\sigma^2\right)\int_0^t I(s)ds\right)\right]\\ =\;& I(t)\exp\left(p\left(\beta-\sigma^2\right)\int_0^t I(s)ds\right),\\ \leq\;&\exp\left(\epsilon t\right). \qquad (13)\end{aligned}$$

By integrating (13) from T to t and multiplying the two sides by $\frac{1}{t}$ we have

$$\begin{aligned}&\frac{1}{t}\int_0^t I(s)ds\\ \leq\;&\frac{1}{p\left(\beta-\sigma^2\right)t}\log\left[\exp\left(p\left(\beta-\sigma^2\right)\int_0^T I(s)ds\right)\right.\\ &\left.+\frac{p}{\epsilon}\left(\beta-\sigma^2\right)\left(\exp\left(\epsilon t\right)-\exp\left(\epsilon T\right)\right)\right]. \qquad (14)\end{aligned}$$

Then, by applying twice the rule of the Hospital on (14) we get

$$\limsup_{t\to\infty}\frac{1}{t}\int_0^t I_s(\omega)ds\leq\frac{\epsilon}{p\left(\beta-\sigma^2\right)}.$$

By letting $\varepsilon\to 0$, the desired result (10) is obtained.

Theorem 3.2 *Let $(S_0,I_0)\in\Delta$. If $\mathcal{R}_s=1$, then for any $\eta>0$ and $\varepsilon>0$ we have*

$$\lim_{I_0\to 0}\mathbb{P}\left(\sup_{0\leq t\leq\eta}I(t)>\varepsilon\right)=0, \qquad (15)$$

that is the disease-free steady state E_0 is stable in probability.

Proof. If $\mathcal{R}_s=1$, then using the formula of Itô, (7), (8) and (9) we get

$$dV_1(I(t))\leq\frac{1}{2}n^2\sigma^2I^n+n\sigma(1-I)^pI^n dB,$$

Integrating between 0 and t, it is easy to have for $n\leq 1$

$$\begin{aligned}I^n(t)-I^n(0) &\leq \frac{1}{2}n^2\sigma^2t\\ &\quad+n\sigma\int_0^t(1-I(s))^pI^n(s)dB_s,\end{aligned}$$

then

$$\begin{aligned}\sup_{0\leq t\leq\eta}I^n(t) &\leq I^n(0)+\frac{1}{2}n^2\sigma^2\eta\\ &\quad+n\sigma\sup_{0\leq t\leq\eta}\int_0^t(1-I(s))^pI^n(s)dB(s).\end{aligned}$$

Using $I<1$ we get

$$\begin{aligned}\mathbb{P}\left(\sup_{0\leq t\leq\eta}I(t)>\varepsilon\right) &\leq \mathbb{I}_{I^n(0)\geq\frac{\varepsilon}{3}}+\mathbb{I}_{\frac{n^2}{2}\sigma^2\eta\geq\frac{\varepsilon}{3}}\\ &\quad+\mathbb{P}\left(n\sigma\sup_{0\leq t\leq\eta}M_t>\frac{\varepsilon}{3}\right),\end{aligned}$$

where $\mathbb{I}_A$ is the characteristic function of A and

$$M_t=\int_0^t(1-I(s))^pI^n(s)dB(s),$$

which implies that

$$\begin{aligned}&\lim_{I_0\to 0}\mathbb{P}\left(\sup_{0\leq t\leq\eta}I(t)>\varepsilon\right) \qquad (16)\\ \leq\;&\mathbb{I}_{\frac{n^2}{2}\sigma^2\eta\geq\frac{\varepsilon}{3}}+\lim_{I_0\to 0}\mathbb{P}\left(n\sigma\sup_{0\leq t\leq\eta}M_t>\frac{\varepsilon}{3}\right).\end{aligned}$$

Moreover, M_t is a real-valued continuous martingale, so by Doob inequality we get

$$\begin{aligned}&\mathbb{P}\left(n\sigma\sup_{0\leq t\leq\eta}M_t>\frac{\varepsilon}{3}\right)\\ \leq\;&\frac{9n^2\sigma^2}{\varepsilon^2}\mathbb{E}\left[\left(\int_0^\eta(1-I(s))^pI^n(s)dB_s\right)^2\right],\\ =\;&\frac{9n^2\sigma^2}{\varepsilon^2}\mathbb{E}\left[\int_0^\eta\left((1-I(s))^pI^n(s)\right)^2ds\right],\\ \leq\;&\frac{9n^2\sigma^2}{\varepsilon^2}\eta.\end{aligned}$$

Combining it with (16) we have

$$\lim_{I_0\to 0}\mathbb{P}\left(\sup_{0\leq t\leq\eta}I(t)>\varepsilon\right)\leq\mathbb{I}_{\frac{n^2}{2}\sigma^2\eta\geq\frac{\varepsilon}{3}}+\frac{9n^2\sigma^2}{\varepsilon^2}\eta.$$

By letting $n\to 0$ we obtain the requested formulation (15).

4 Simulation

To validate the robustness of our discoveries, we intend to carry out numerical simulations employing the Milstein scheme as our chosen computational approach, as outlined in [25]. More precisely, we plan to discretize equation (3) utilizing the subsequent scheme:

$$\begin{cases} S_{k+1} = S_k + \left[\mu_k - \mu_k S_k - \beta_k S_k^p I_k + \gamma_k I_k\right]\Delta t \\ \qquad -\sigma_k S_k^p I_k \sqrt{\Delta t}\tau_k - \frac{\sigma_k^2}{2} S_k^p I_k(\tau_k^2 - 1)\Delta t, \\ I_{k+1} = I_k + \left[-\left(\mu_k + \gamma_k\right) I_k + \beta_k S_k^p I_k\right]\Delta t \\ \qquad +\sigma_k S_k^p I_k \sqrt{\Delta t}\tau_k + \frac{\sigma_k^2}{2} S_k^p I_k(\tau_k^2 - 1)\Delta t. \end{cases}$$

In this context, τ_k, $(k = 1, 2, ...)$ symbolizes an uncorrelated stochastic variable adhering to standard normal distributions, specifically represented as $\mathcal{N}(0, 1)$.

Example 1 *We choose* $\mu = 0.5$, $\beta = 0.92$, $\gamma = 0.4$, $\sigma = 0.2$, $p = 1$, $I_0 = 0.4$, *then*

$$\mathcal{R}_0 > 1, \quad \mathcal{R}_s = 1.$$

Consequently, based on Theorems 3.2, 3.1, and Fig. 1, *the stochastic disease will be removed from the population while the deterministic disease occurs.*

Example 2 *Set* $\mu = 0.4$, $\beta = 0.9$, $\gamma = 0.45$, $\sigma = 0.5$, $p = 1$, $I_0 = 0.4$, *then*

$$\mathcal{R}_0 > 1, \quad \mathcal{R}_s < 1.$$

Thus, based on Theorem 2.1, and Fig. 2, *the disease-free equilibrium state* E_0 *is* n*-th moment exponentially stable.*

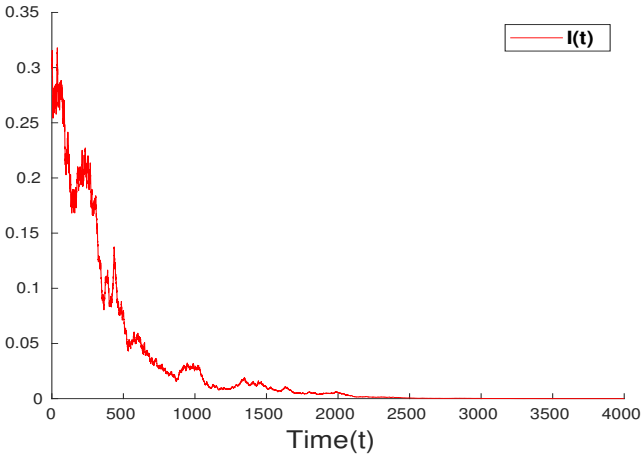

Fig. 1. Trajectories of $I(t)$ for SDE model 3 by using the parameters of Example 1.

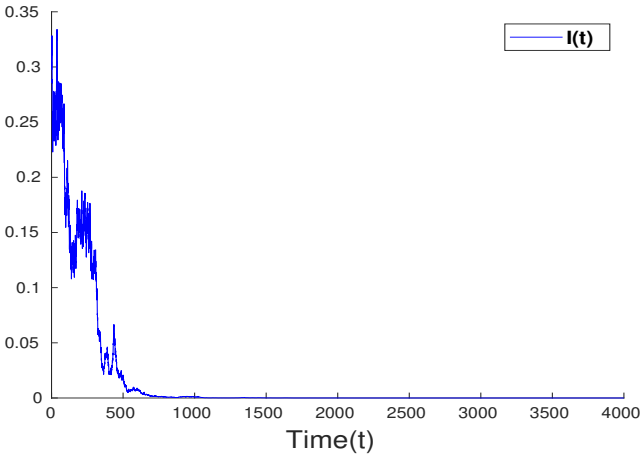

Fig. 2. Trajectories of $I(t)$ for SDE model 3 by using the parameters of Example 2.

5 Conclusion

This study rigorously examined a stochastic SIS epidemiological model within a constant-sized population under white noise control. Through our analysis, we have comprehensively explored the long-term behavior of the SIS stochastic epidemiological model and drawn significant insights. Our findings provide compelling evidence for the overall stability of the disease dynamics and the eventual extinction of the disease within the population. This indicates that the control measures implemented, represented by the white noise control in this context, have effectively mitigated disease-sustained transmission, leading to disease-free equilibrium over time. The implications of our research extend to public health and disease management strategies. By elucidating the model stability and extinction properties, we offer valuable guidance for designing effective control interventions and optimizing resource allocation to combat infectious diseases in similar settings. Nevertheless, we acknowledge that the stochastic nature of infectious disease dynamics poses inherent complexities. Future research could explore further refinements to the model, considering additional factors such as demographic heterogeneity, spatial interactions, and temporal variations in control measures to capture real-world epidemiological scenarios better.

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Contribution of individual authors to the creation of a scientific article (ghostwriting policy)

Bilal Harchaoui, Mourad El Idrissi: were responsible for the conceptualization, validation, formal analysis, writing - original draft, methodology, writing - review & editing. **Adil El Haitami, Abdeladim Nait Brahim:** have implemented the software, formal analysis, writing - original draft, writing - review & editing. **Adel Settati, Aadil Lahrouz, Mustapha El Jarroudi:** have implemented the software, formal analysis, writing - original draft, writing - review & editing. **Mustapha Er-Riani, Tarik Amtout:** Carried out the validation, investigation, conceptualization, writing - review & editing.

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To begin with, we have received an invitation from your esteemed mathematical journals to submit our work. Additionally, the authors declare that they have no identifiable financial conflicts of interest or personal relationships that could influence the findings and conclusions presented in this article.

Conflict of Interest

The authors have no conflicts of interest to declare that are relevant to the content of this article.

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